MINDFULNESS MEDITATION REDUCES STRESS-RELATED INHIBITORY GATING IMPAIRMENT

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ABSTRACT

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This project examined how the human brain responds to stress and how mindfulness meditation can reduce stress-related sensory processing deficits. An early brain function called inhibitory gating is impaired in various mental illnesses. Inhibitory gating promotes healthy cognitive function, as gating is theorized to play an important role in the pre-attentional stages of information filtering in the brain. Inhibitory gating is evaluated with electroencephalography (EEG), in which the electrical activity of neural networks is non-invasively assessed via electrodes placed on the scalp. Gating deficits can be induced in healthy people for a brief time with exposure to physical or mental stress, which allows for the gating impairments seen in mental illnesses to be modeled in healthy people. Mindfulness meditation training has been a benefit to patients in various therapeutic settings, but treatments for gating impairment remain unknown. It is essential to target this pervasive deficit for treatment. In the current study, mindfulness meditation was tested as a technique to reduce stress-induced gating impairment. Participants attended four meditation training sessions and underwent a cold-pressor stress task twice; once at the beginning and once at the end of the four appointment experiment. EEG recordings were taken before and after the stress task. The results of this experiment show that mindfulness meditation training can reduce stress-induced inhibitory gating impairment. Two control groups completed personality surveys or progressive muscle relaxation exercises and did not exhibit reduced impairment after four sessions. These findings are promising in that they contribute to the wider understanding of gating impairment and its relationship to stress, and expand on potential treatment options by introducing a safe, low-cost technique with potential to reduce inhibitory gating impairment.

This manuscript is dedicated to Peter. Your unconditional love and encouragement helped me accomplish so much. Thank you for always being there. I love you.

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CHAPTER 1

INTRODUCTION: EVENT-RELATED POTENTIALS AND THE ERP TECHNIQUE

1.1 What are ERPs?

General Introduction

Hans Berger discovered that summed post-synaptic potentials could be recorded by placing an electrode on the scalp, amplifying the electrical signals from the brain, and measuring the changes in voltage over a period of time – a technique called electroencephalography (EEG) (Berger, 1929; Luck, 2005). It took several years for EEG brain recordings to gain traction as true evidence of brain function, but EEG has since become a valuable tool in brain research and in clinical settings (Luck, 2005). EEG provides a wealth of information that can be difficult to parse apart, since EEG recordings are sums of electrical activity. The event-related potential (ERP) technique was introduced to extract neural responses that are specific to sensory, motor, and cognitive processes through averaging (Luck, 2005). These responses are called event-related potentials (originally evoked potentials) because they are evoked by a specific event or stimulus (Luck, 2005). Luck (2005) defines an ERP as: "Scalp-recorded neural activity that is generated in a given neuroanatomical module when a specific computational operation is performed (p.59)."

ERP waveform averages were automatically computed as early as 1962 (Galambos and Sheatz). Many pioneering ERP studies measured brain responses as volunteers completed cognitive tasks. Researchers noted that ERP responses were specific to the nature of attentional and anticipatory tasks (Sutten et al., 1965; Walter et al., 1964). The overall discovery was that event-related potentials provide a valuable measure of discrete brain processes that are unique to different events. For several years, endeavors in ERP research were cognitive in nature and dedicated to the discovery of new ERP components (Luck, 2005). It was not until the

components had been widely tested and verified in the 1980s that the real work began: What do these ERP components represent, and how do they contribute to brain function and dysfunction?

Source of Electric Signal

EEGs and ERPs are measures of post-synaptic electrical activity, which is neural activity that follows individual action potentials (Luck, 2005). Post-synaptic potentials are measured as local field potentials from neurons underneath the scalp electrodes. This is as precise as ERP techniques can be, but other invasive measures, such as single and multi-unit recordings, allow for action potentials to be assessed at the nearly speed of light – the speed of electricity (Luck, 2005). ERPs are not a good solution for measuring action potentials because these potentials do not occur at the same time and often cancel out (Luck, 2005). Though surprisingly little research has been conducted on the neural origin of ERPs, they are thought to result from the release of excitatory neurotransmitters that trigger a net negativity in extracellular space near dendrites. The electrical circuit is completed by the creation of a net positivity within the cell that flows from the cell body through the dendrites, creating a dipole (Luck, 2005). Single neuron dipoles are extremely small in amplitude, and EEG is not sensitive enough to pick them up. EEG can, however, measure the summed, simultaneously occurring dipoles of many similarly oriented neurons (Luck, 2005). If the neurons are not aligned properly, the currents will cancel each other out. The currents travel through the volume of the brain, skull, and ultimately the surface of the scalp, and there is some distortion as the currents span out at the skull. However, ERP is still a useful tool to illuminate sensory, motor, and cognitive processes even if it does not allow for the identification of signal generation points. Knowing where something occurs is not knowing how or why it occurs (Luck, 2005). ERP is, in general, reliant upon a specific set of biophysical

phenomena occurring precisely in tandem. The complexity this captures can provide a window of insight into a variety of brain processes.

1.2 Different Forms of ERPs

ERP components are divided into latency stages post-stimulus. There are early, mid, and late components in terms of the amount of time it takes for them to occur in response to stimulation. Early responses can be as quick as less than ten milliseconds post-stimulus, while later responses may take several hundred milliseconds (Luck, 2005). ERP components are further divided by positive or negative polarity (commonly abbreviated as P or N). It is important to distinguish auditory and visual responses that share names based on these latency and polarity guidelines, such as the auditory N1 and the visual N1 components. Typically, components are not functionally related across domains (Luck, 2005). ERP components cover a wide variety of physiological responses including somatosensory, olfactory, gustatory, motor, error detection, and even language processes. However, auditory and visual components are the most popular focus of cognitive ERP experiments (Luck, 2005).

Visual Components

The C1 component is thought to generate in the primary visual cortex (Luck, 2005). It is one of the earliest ERP responses to visual stimuli. C1 is somewhat unique in that it is not assigned a polarity because it can be positive or negative (Luck, 2005). The C1 wave typically begins 40-60ms after a stimulus and reaches its maximum peak at 80-100ms. In this regard it is one of the longer-lasting components. C1 varies depending upon visual stimulus factors like spatial frequency (Luck, 2005). The P1 component follows C1 and is, as the name implies, a positive component with an onset of approximately 60-90ms and a peak 100-130ms post-

stimulus. P1 has been observed at the lateral occipital areas and is influenced by stimulus parameters such as contrast as well as internal states like arousal and spatial attention (Hillard et al., 1998; Vogel and Luck, 2000). There is overlap in the timing of C1 and P1, which makes it difficult to differentiate P1 from C1. There have been attempts to localize the generation point of the P1 wave using fMRI (Di Russo et al., 2002), but this endeavor is complicated by the fact that various brain areas are involved in visual processing by the 100ms post-stimulus mark. Nonetheless, Di Russo and colleagues (2002) tentatively identified an early P1 generator in the dorsal extrastriate cortex and a later P1 generator in the fusiform gyrus, which is widely known as an essential area for face recognition processing.

The N1 wave follows P1 in visual sensory response analysis. The N1 family covers many subcomponents (Hopf et al., 2002). Some N1 components peak at 100-150 post-stimulus while others occur later at 150-200ms. Anterior sites peak earlier than the posterior sites that have been localized to the parietal cortex and the lateral occipital cortex. In contrast to C1 and P1, N1 subcomponents are less influenced by spatial parameters, although they are affected by spatial attention. Some N1 subcomponents are larger during a discrimination task than a detection task, which suggests more complex sensory processing at the N1 stage (Vogel and Luck, 2000). The P2 wave comes after P1 and is generally observed at anterior and central electrode sites (Luck, 2005). The P2 wave has larger peaks during target identification tasks and may be influenced by stimuli novelty (Luck and Hillyard, 1994). The P3 wave occurs as increases in response to progressively complex target parameters (Luck, 2005). The N170 wave was identified by Jeffreys (1989) as unique to the presentation of faces, although the N170 response has been observed for familiar non-face stimuli as well (Schendan et al., 1998). Thus, various visual processes can be studied from ERP responses in an array of areas.

Auditory Components

Brainstem evoked responses to auditory stimuli can be recorded at very early stages, such as 10ms post-stimulus. These are followed by mid-latency components occurring within 10-50ms post-stimulus. Variables like attention may influence mid-latency auditory components at this stage but this is not the consensus (Luck, 2005). Mid-latency responses are generated in part by the primary auditory cortex and the geniculate nucleus. They are followed by an auditory P1 wave observed at frontal and central sites (Luck, 2005). Like the visual N1 component, the auditory N1 has various subcomponents (Alcaini et al., 1994). These include but are not limited to subcomponents generated in the auditory cortex that peak at 75ms post-stimulus, a 100ms post-stimulus peak of uncertain origin, and a 150ms peak generated in the superior temporal gyrus (Luck, 2005). There is some evidence that the auditory N1 wave is influenced by attention, as mismatch-related negativity can be observed when repetitive auditory stimuli are interspersed with non-identical stimuli (Woldorff et al., 1993). Mismatches can cause a negative wave that peaks around 160-220ms after the conflicting stimulus is presented. This negative wave is usually found in the midline regions. Interestingly, there is a caveat. Mismatch negativity is only influenced by attention if a participant exerts effort to willfully concentration on the stimuli (Woldorff et al., 1991). Otherwise, mismatch negativity is unaffected by attentional manipulations and is therefore thought to reflect pre-attentional or automatic processes. Luck (2005) suggests that this process may actually involve sensory memory traces that facilitate the comparison of stimuli. The role of attention in mid-latency auditory components like P50 has been repeatedly tested and found to have no effect (Jerger et al., 1992; Olincy et al., 2000; White and Yee, 1997), but there are conflicting accounts (Hutchison et al., 2013; Lijffijit et al, 2009).

Cross-Domain Components

The N2 family of responses can be elicited by both visual and auditory stimuli. Repeated stimuli elicit the N2 wave while task-irrelevant and novel stimuli increase N2 peaks, which reflects mismatch negativity (Näätänen and Picton, 1986). If the deviant novel stimuli (auditory or visual) are relevant to a cognitive task, there is also a late-occurring increased peak that is considered to be separate from mismatch negativity. This late N2 wave is thought to generate from central sites when auditory stimuli are presented and posterior sites when visual stimuli are presented (Simson et al., 1977). The N2 component in general may be involved in sensory stimulus categorization, while tests of subcomponents have revealed potential roles in working memory, spatial attention to targets, and purposefully ignoring non-targets (Eimer, 1996; Vogel and Machizawa, 2004). Some N2 subcomponents are sensitive to probability, while others are not (Luck, 2005).

The P3 component is composed of two major subcomponents: 1) P3a, which is observed in frontal regions; and 2) P3b, which is observed in parietal regions (Squires et al., 1975). Both the P3a and P3b subcomponents are elicited by sudden shifts in tone pitch and intensity. Task-irrelevant stimuli can induce a frontal wave that resembles P3 (Soltani and Knight, 2000), although this may be another component entirely, since P3a waves respond to completely unexpected stimuli, while P3b waves respond to infrequent yet anticipated stimuli (Verleger et al., 1994). Although there is still a lack of agreement concerning its true function, the P3 wave is sensitive to certain factors. For example, the P3 wave is influenced by stimulus probability in that P3 peaks increase if the probability of target stimuli occurrences decrease (Duncan-Johnson and Donchin, 1977). Moreover, P3 amplitude increases as effort on a task increases, which may indicate a role in resource allocation (Isreal et al., 1980). P3 is also influenced by uncertainty.

P3 waves do not increase in amplitude if a participant has trouble distinguishing targets from non-targets (Luck, 2005). With these influences taken into account, experiments involving the P3 component may be a valid way to assess whether manipulations successfully affect stimulus categorization, response selection, and decisions related to these topics (Luck, 1998b). ERP components have shown a great deal of worth in cognitive neuroscience by allowing for such covert sensory processing and cognitive mechanisms to be measured objectively, although some inference is still necessary.

1.3 Typical Measurements of ERPs

Amplitude

Evaluation of peak amplitudes is one of the most common ways to interpret ERP data. Amplitudes are measured in reference to pre-stimulus baseline activity. Baseline activity is simply the mean voltage observed in the absence of experimental stimuli. Luck (2005) discusses two main ways to go about amplitude measurement: 1) Select a post-stimulus time window and identify the largest peak within every occurrence of this time window; or 2) Select a post-stimulus time window and calculate the average amplitude within that time window for each waveform. A third and uncommon option is to sum voltage information within a time window, but this area amplitude technique does not differ substantially from the mean amplitude approach and may introduce more noise (Luck, 2005). Amplitude ERP measures are complicated by components that overlap. Nonetheless, accuracy is increased with filtering and precise time window parameters.

Peak-to-Peak

ERP data can also be quantified as the difference between neighboring peaks and troughs. This comparison of waveforms provides peak-to-peak information that is less encumbered by confounds like the background noise found in pre-stimulus baseline information (Handy, 2004). If the peak-to-peak technique is used, it is essential to keep in mind the stability of waveform comparison reference points. These can change as experimental manipulations are introduced. If there is stability in the waveform landmarks, which is to say that they change in tandem across experimental conditions, then the peak-to-peak amplitudes can yield meaningful information. Otherwise, inferences drawn from peak-to-peak analysis could inadvertently reflect changes in a peak but not a reference point, or vice versa (Handy, 2004).

Oscillations

For decades it has been suspected that the generation of ERPs is tied to wider changes in brain oscillations. The basis of this theory is that changes in ERP responses often coincide with changes in oscillatory activity. For example, Pfurtscheller and Aranibar (1977) found that brief, area-specific desynchronization of alpha oscillations occurred during auditory and visual stimulation, as well as the follow-up ERP responses. According to Sauseng and colleagues (2007), there are two primary theories on the neural basis of ERP generation: 1) The evoked model which assumes that ERPs are unrelated to background EEG activity, i.e., oscillations; and 2) The phase reset model which assumes that changes in brain oscillations themselves are the source of ERPs. Yeung and colleagues (2004) point out that signal averaging ERP studies operate on the assumption that neural data obtained through ERP techniques are independent of background EEG activity, as in the evoked model. This assumption is called into question by a volume of research reviewed that suggests synchronization of brain oscillations during ERP

responses, as in the phase reset model. Yeung and colleagues (2004) reviewed a number of such studies and subsequently put the phase reset to the test using computer simulation of EEG data. The results of the simulated data were very similar to those of synchronized oscillation studies, yet the simulation allowed the researchers to determine there was no actual synchrony. This demonstration has serious implications for the techniques used to determine oscillation synchrony and its relevance to ERP data, although the phase reset model cannot be ruled out at this point in time. It may simply be more difficult to determine whether ERPs are related to oscillations and synchronization than to assume that they are independent of them.

1.4 Information Gleaned from ERP Studies

The results of ERP studies have various uses. Luck (2005) gave a personal example in which his children were tested for auditory pathology using ERP techniques. ERP studies have also contributed to the understanding of fundamental neural activity differences found in certain mental illnesses (Bramon et al., 2004), for which treatments have likewise been assessed (Boutros et al., 2004). ERP components can even be used as biomarkers in some cases, such as schizophrenia (Freedman et al., 2005). ERP components can also be used as predictors of future pathology, as in anxious and depressive symptoms during development (Hutchinson et al., 2013), or genetic predisposition to psychosis (Clementz et al., 1998). ERP has an excellent advantage as a noninvasive and surreptitious option to assess physiological responses to various stimuli, tasks, and processing stages.

1.5 Problems and Controversies in ERP Research

The reliability of ERP waveforms has been called into question from the beginning (Luck, 2005). The main issue is inherent to the nature of ERPs in that the signal must be separated from a rather substantial amount of electrical noise in the EEG recording. Another caveat of the ERP technique is that most researchers use a grand average to present ERP findings at the expense of individual variability (Luck, 2005). This can be a benefit in terms of representing the most probable typical response, but it can be a poor way to account for potentially substantial differences in responses or response types between subjects (Luck, 2005). Individual variability in ERP components is theorized to be the result of differences in cortical fold arrangement, medications, age, illness, and perhaps even personality (Luck, 2005). In other words, ERP signals can be very difficult to pinpoint.

ERPs are often used in conjunction with cognitive and behavioral research. For example, ERP techniques complement reaction time experiments by providing processing data in real time. ERPs are a measure of continuous processing rather than the end product as measured in behavioral output. Because of this quality, ERPs can measure overlapping processes and more behaviorally obfuscated processes, such as ignoring a stimulus (Luck, 2005). ERPs also have the advantages of excellent temporal resolution (i.e., responses are measured as they happen), relatively inexpensive setups in comparison to fMRI sister projects, and providing a safe, non-invasive option for studying brain processes in humans, for which the implanted microwires in animal models are rarely feasible.

ERPs may improve upon some reaction time experiments, but they are nonetheless impeded by the sheer amount of information provided by EEG recordings. It can be very challenging to accurately isolate an ERP signal and average multiple responses correctly (Luck,

2005). In this way, a behavioral response is a much clearer measure. A greater deal of inference is needed when interpreting real time processing signals, such as why is the delay at a certain latency, what processes and components come before and after, why isn't the component showing up in 100% of the trials, and so on. Another drawback of ERP research is that the target signals are small in comparison to what surrounds them, and they don't always appear reliably (Luck, 2005). This means that a large number of experimental trials are almost always necessary in order to obtain enough data to accurately draw conclusions from ERP data (Luck, 2005). This has several potential unintended consequences, such as participant (and experimenter) fatigue alongside the somewhat disputed policy of removing trials in which an ERP response did not cleanly appear. It is arguable that the "failed" trials represent normal processing as well.

Moreover, the significant amount of time required to collect adequate data limits the types of practical questions ERP techniques can answer, as well as how well they represent naturalistic function if a participant must endure three hour long EEGs while watching flashing lights or listening to tones! To address these issues, Luck (2005) suggests designing experiments with the limitations of ERP in mind, and avoiding research questions that ERPs cannot answer completely, such as neural generation points.

CHAPTER 2

INHIBITORY GATING

2.1 What is Inhibitory Gating?

Theorized Function

Inhibitory gating can be described as the brain's ability to modulate (or "gate") its responses to incoming sensory stimuli (Boutros et al., 1999), which ultimately results in the brain interpreting the outside world via physical sensations. Inhibitory gating is part of a multistage process that involves modulation of the brain's responses to incoming sensory information (Gjini et al., 2010). Theoretically, inhibitory gating allows for irrelevant information to be filtered out in the early stages of sensory processing, thereby promoting effective cognitive and emotional functioning (Gjini et al., 2010; Knight et al., 1989). Healthy inhibitory gating function supports effective adaptation to changing environments and attention to relevant information within them (Patterson et al., 2008).

Inhibitory gating differs from several other concepts that may seem similar on the surface, such as habituation, adaptation, and neural fatigue. Habitation is a learned behavior in which responses decrease to repeated stimuli. Inhibitory gating differs from habituation in that it is a neural response to incoming sensory stimuli. There is an increase in response to novelty and a decrease in response to repetitive stimuli, but the neural mechanism involved recovers extremely quickly and will identify even a repeated stimulus as "novel" provided there is enough time for neural recovery between stimulus presentations (Boutros et al., 1999). Thus, inhibitory gating only requires enough time for the response to recover and does not reflect adaptations in behavior in the same way as habituation.

How Gating is Measured

In humans, inhibitory gating function is measured using electroencephalography (EEG) and event-related potential (ERP) techniques, in which summed action potentials are monitored via electrodes placed on the scalp (Luck, 2005). Inhibitory gating is characterized by a reduction in neural responsiveness to redundant stimuli (Boutros et al., 1999). The P50 component is the essential measure in the evaluation of human inhibitory gating. P50 is a positive, evoked potential response that occurs approximately 50ms after the presentation of a stimulus (Boutros et al., 1999; Boutros et al., 2004; Jerger et al., 1992; Johnson and Adler, 1993; Yee and White, 2001). Auditory paired click paradigms, an ERP technique, are frequently used to elicit P50 responses. Identical first and second clicks are separated by an intertrial interval of 500ms while click pair trials are separated by 8 to 10 seconds (Adler et al., 1998; Boutros et al., 1999; Dolu et al., 2001; Mears et al., 2006; Zouridakis and Boutros, 1992). The 500ms interstimulus interval is essential, as anything earlier could instead reflect a startle response and anything later is unlikely to capture the characteristic pattern that defines healthy gating: a strong response to the conditioning stimulus and a reduced response to the test stimulus (Bak et al., 2011; Shore and Keith, 1993). There does not appear to be a record of any studies that have used an interstimulus interval other than 500ms with any success (Hong et al., 2007). Paired click paradigms have shown that P50 amplitudes decrease if auditory stimuli are repeated, and increase if auditory stimuli are novel or not identical (Boutros et al., 1999). This is strong evidence that the P50 component is sensitive to stimulus patterns that indicate the degree of gating or inhibition through attenuation (decreased responses) to repeated stimuli and augmentation (increased responses) to novel stimuli (Boutros et al., 1999).

In auditory paired click paradigms, the first click in a pair is commonly identified as the conditioning stimulus (C), while the second click is called the test stimulus (T). The test response is the true evaluation of the inhibitory circuit (Boutros et al., 2004; Dolu et al., 2001). The relationship between P50 responses to T and C stimuli can be assessed as a T/C ratio, which is calculated as the test response value divided by the conditioning response value, and then multiplied by 100 to yield a percentage (Davies et al., 2009). The values used in this equation are averaged amplitudes of maximum positive peak (P50) responses occurring within a certain range, such as 40 to 80ms, with a preferred latency near 50ms after stimulus onset (Chang et al., 2011).

Typical Gating Profile

The T/C ratio theoretically represents inhibitory gating function as a value (Boutros et al., 2004). A low ratio would indicate that the response to the conditioning click is high in amplitude, while the response to the test click is reduced in comparison to the conditioning click. A low ratio is thought to indicate better inhibitory gating because the novel stimulus is attended to while the redundant stimulus is filtered out (Patterson et al., 2008). A high T/C ratio is an indicator of impaired inhibitory gating.

The cut-off value for normal versus impaired inhibitory gating has been debated, since a high T/C ratio can be the result of different response patterns. When the test response is higher in amplitude than the conditioning response, which would result in a T/C ratio over 100, it is possible that a response to stimulus change is being measured rather than inhibitory gating impairment (Gjini et al., 2010). A recent meta-analysis (Chang et al., 2011) shows that average T/C ratios in healthy control populations varied from 16% to 94% across 35 studies, although ratios for schizophrenia patients were higher overall. Chang and colleagues (2011) examined

whether inhibitory gating impairments, as reflected by T/C ratios, were traceable to a reduced response to the conditioning stimulus or an exaggerated response to the test stimulus. The authors found that conditioning responses tended to remain stable in both healthy controls and persons with schizophrenia. This means that initial responses to novel stimuli did not change. However, there was great variation in the redundant stimuli or test response in persons with schizophrenia as compared to healthy controls. This indicates that persons with schizophrenia may have a reduced ability to inhibit or "gate" unnecessary information, which has the general result of increasing the test response and causing it to more closely resemble the conditioning response in amplitude.

Unlike the increased test response, a weak response to the conditioning stimulus may indicate an error in registration rather than a problem with inhibitory gating (Chang et al., 2011). An issue with registration differs from inhibitory gating impairment in that decreased response amplitudes to initial or novel sensory stimuli imply that there was a failure in stimulus detection rather than detected information being recognized as irrelevant and gated out (Braff et al., 1992). Inhibitory gating is specifically the attenuation of the response to the test stimulus in relation to the conditioning stimulus. Even a very low response to the conditioning stimulus should not be considered a gating problem as long as the T/C ratio is low. It is theoretically possible to have registration and gating problems at once, although they would be indistinguishable in a high T/C ratio. Chang and colleagues suggest a C minus T difference score to supplement interpretations of T/C ratios for this reason. It is possible to conclude from T/C ratios and difference scores that a normal inhibitory gating profile consists of a strong P50 response to a conditioning stimulus and a lessened response to a test stimulus.

Controversy Surrounding Gating

There is an ongoing debate on the reliability of the P50 measure of gating in humans. Patterson and colleagues (2008) conducted a review of current literature to aid in the standardization of P50 assessment with the goals of increasing accuracy and consistency of results. The authors found a wide range of individual differences in T/C ratios in healthy controls. As much as 40% of healthy controls had gating ratios that overlapped with those of schizophrenia patients. If the T/C ratio is to be used as an indicator of mental illness, it must be reliable and replicable. Patterson and colleagues (2008) suggested various ways to reduce artificial variation by precisely monitoring stimulus parameters and incorporating other components to help identify P50.

High T/C ratios are common in persons with schizophrenia, but there is not universal agreement that this represents inhibitory gating impairment. Rather, it may be a symptom of more complex changes in the processing of sensory information and could span into encoding problems or changes in alpha and gamma oscillatory activity, which could exert top-down modulation of the P50 response (Popov et al., 2011). Rentzsch and colleagues (2008) have also called into question the test-retest reliability of the P50, N100, and P200 components as elicited by paired click paradigms. The authors' arguments are that N100 and P200 gating have not been tested sufficiently in this regard, while P50 gating has a reputation for unreliability. Whether P50 is inherently unreliable or simply difficult to measure is another matter of debate, which the authors intended to address. Rentzsch and colleagues (2008) tested the reliability of the P50, N100, and P200 gating components in 41 healthy people. Paired click paradigms were used and EEG data were collected two times over two sessions that were four weeks apart. Ratios and S1-S2 differences were assessed. It was found that P50 gating had excellent reliability across the

board, including peak-to-peak, baseline-to-peak, ratio, and difference measurements. One caveat was that the baseline-to-peak measurement was much more reliable than the peak-to-peak measurement of amplitude. P200 gating was very stable over time. The authors' conclusion was that the most reliable gating measure over the long-term was P200 since P50 and N100 gating were more difficult to replicate when they were assessed as ratios. It is difficult to say whether P50 reliability experiments are getting at changes in measurement accuracy or changes in fickle components. It is easy to assume that ERP components are generated in a predictable fashion, but the reliability of the P50, P200, and N100 gating indices in Rentzsch and collegues' (2008) study is a reminder that individual differences in mental health, arousal, attention, and medications have to be considered before gating can boast the stability to be utilized as a biomarker.

Notably, Rentzsch and colleagues' (2008) investigative study was limited to healthy controls; others, however, have noted that some schizophrenic patients can temporarily exhibit normal gating ratios (Griffith et al., 2005). Jin and colleagues (1997) have examined the T/C ratio in schizophrenia patients and controls and found that patients have more variation in their conditioning responses as well as higher ratios overall, while the average amplitudes for conditioning and test responses were not different across the control and patient populations. The authors argue that it is extremely important to take the temporal variability of P50 into account because there is more variation in schizophrenia patients' inhibitory gating responses, and averaged responses can provide a clearer picture of gating function. Gating in healthy controls would presumably be less variable and more stable, although even healthy gating is not fully understood and long-term reliability remains to be determined. More rigorous testing is

required, and the stability of P50 over time should not be assumed without more converging evidence.

2.2 Functional Importance of P50 Gating

Despite valid criticisms of what remains unknown about gating, the P50 component measure has nonetheless proven to be a useful assessment tool. P50 gating has been thoroughly investigated in dozens of reputable studies, as recent meta-analyses summarize (Patterson et al., 2008; Chang et al., 2011). P50 is distinct from other gating components such as N100 and P200, in that P50 is pre-attentional and a potential reflection of very early sensory inhibition. P50 is important not only for its potential as a biomarker of psychosis and other depressive and anxious pathologies (Freedman et al., 2005; Hutchison et al., 2013), but also for its potential to elucidate how pre-attentional inhibitory gating can influence and potentially protect later, higher-order processes like response bias, behavioral inhibition, working memory, and attention (Lijffijt et al., 2009).

Sleep and Oscillations

An excellent way to further basic understanding of gating is to assess gating function during sleep. Kisley and colleagues (2001) attempted to measure inhibitory gating during REM sleep instead of the waking state in order to better understand how gating dysfunction could contribute to perceptual and cognitive impairments in mental illnesses. The study of gating function is made more challenging by the fact that it is impacted by states like acute stress. Studying gating function during REM sleep could remove behavioral confounds. Kisley and colleagues (2001) measured gating in healthy controls and expected gating to be improved during sleep due to the absence of behavioral and attentional interference. They found that

gating was present during REM sleep and gating ratios tended to improve during sleep in comparison to the waking state. Although this reduction was not statistically significant, the sleep study approach to gating is intuitive since gating is a pre-attentional process. Pre-attentional processes like gating differ from attentional processes in that they occur before willful control can be exerted and are therefore largely unconscious. This is supported by findings that suggest distractions and attentional manipulations have no effect on gating performance (Jerger et al., 1992; Olincy et al., 2000).

Given gating's pre-attentional nature, could chronic gating impairment be restored to normal during sleep? Griffith and colleagues' (1995) findings show that the answer to this question is both yes and no for persons with schizophrenia, as impaired gating persisted during REM sleep, and transient normalization occurred during non-REM sleep. These findings support the idea of P50 gating as a process beyond conscious control and differentiate it from other early processes like attention. The authors speculate that gating normalization in non-REM could be due to the re-sensitization of typically desensitized nicotinic receptors during this phase of sleep. Interestingly, persons with schizophrenia have reduced amounts of non-REM sleep on average, which may tie in with the chronic inhibitory gating impairments seen in this disorder. A later study by Kisley and colleagues (2003) confirmed that P50 inhibitory gating remained impaired in schizophrenia patients when awake and when in REM sleep, while N100 gating was not impaired during sleep. This may point to special potential in P50 gating as an assessment tool, in that impairment in P50 suppression can be assessed during REM sleep and allow for various waking state confounds to be ruled out.

P50 gating impairments in persons with schizophrenia have also been linked to changes in event-related brain oscillations. Popov and colleagues (2011) found that schizophrenia

patients had impaired oscillatory responses to conditioning clicks. This was reflected as smaller alpha and gamma responses in comparison to healthy controls. This link between oscillations and P50 gating carries the interesting implication that persons with schizophrenia may process incoming sensory information in a fundamentally different way, which could cause a wide variety of dysfunction. A theory that supports this is Buszaki's (2006) idea of brain function having a default activity profile or mode. If P50 gating is linked with oscillations, then it is also possible that disruptions at the early sensory processing stage can have bottom-up effects on the larger network. Taken together, previous research suggests that P50 inhibitory gating has larger roles that may not be obvious. P50 gating is involved in early sensory information processing and can influence changes in broader oscillatory patterns. In a sense, gating also lays groundwork for higher order functions to build upon. Multiple cognitive functions can suffer down the line if this foundation of sensory input is not accurate or stable. For example, Lijffijit and colleagues (2009) found better P50 gating to be correlated with fewer false alarm errors and longer reaction times leading to correct choices on a delayed memory task. Despite the limitations of gating assessment via the P50 component in humans, the functional importance of this measure should not be underestimated. This is especially true in the study of mental illnesses in which gating is impaired. A great deal can be learned about the purpose and function of P50 gating when it ceases to work properly.

2.3 Mental Illness and Observed Gating Impairments

Research has shown that inhibitory gating is altered in persons with schizophrenia, obsessive-compulsive disorder, post-traumatic stress disorder, and Alzheimer's disease (Boutros et al., 2004; Gjini et al., 2013; Jessen et al., 2001; Jin et al., 1996; Johnson and Adler, 1993;

Louchart-de la Chappelle et al., 2005; Myles-Worsley et al., 1996; Neylan et al., 1999; Patterson et al., 2008; Rossi et al., 2005). Of these disorders, inhibitory gating impairments in schizophrenia have been studied most comprehensively. Inhibitory gating impairments have been so reliably observed in schizophrenia that P50 has been suggested as a biomarker for the disorder (Freedman et al., 2005). These impairments offer various evidence of relationships between gating and emotion, as well as gating and cognition.

Schizophrenia

P50 suppression deficits have been extensively documented as endophenotypes of schizophrenia (Knott et al., 2009). P50 deficits have been recognized in patients with recent schizophrenia diagnoses (Yee et al., 1998) as well as in first degree family members of schizophrenia patients at a rate of 50% (Clementz et al., 1998). Suppression deficits have also been observed in schizotypal personality disorder, which shares symptomology with schizophrenia (Candenhead et al., 2000), and in prodromal adolescents who are at higher genetic risk of developing schizophrenia (Myles-Worsley et al., 2004). Bramon and colleagues (2004) suggest that P50 suppression deficits have a great deal of potential as biomarkers of schizophrenia spectrum disorders. Bramon and colleagues' (2004) conducted a meta-analysis that included 20 P50 gating studies with a total of 421 schizophrenia patients and 401 controls. The combined results show gating deficits in the patient population, which implies that the very electrophysiology of the brain is affected by schizophrenia. P50 gating impairments are present even in patients on antipsychotics. The gating impairments are described as having "effect sizes so large that, should the P50 ratio…be [an] observable phenotype, differences between patients and controls would be appreciated by the naked eye (Bramon et al., 2004, p.321)." This idea is substantiated by other findings that inhibitory gating is impaired in other mental illnesses that

can involve psychosis, such as bipolar disorder (Olincy et al., 2006), epilepsy (Boutros et al., 2006), traumatic head injury (Arciniegas and Topkoff, 2004), and Huntington's disease (Uc et al., 2003).

What is the underlying basis of this impairment? Bleuler (1911) hypothesized that perceptual dysfunctions in schizophrenia were related to an inability to ignore information from irrelevant sensory inputs. Venables (1964) later expanded on this idea and described inhibitory gating impairment as "sensory flooding," in which unnecessary information is not filtered effectively and ends up inundating sensory buffers. Sensory flooding is a fitting description of inhibitory gating impairment as problems in early sensory processing point to later disruptions of higher order cognitive function (Lijffijit et al., 2009).

Recent research (Boutros et al. 2004) suggests that there later processing deficits are at work in schizophrenia since other mid-latency information processing components, such as N100 and P200, are impaired in addition to P50 gating. This indicates that the underlying problem may be more than pre-attentional, and perhaps that deficits with P50 gating at the pre-attentional stage have trickle-down effects on later information processing as indicated by later N100 and P200 mid-latency gating components. Inhibitory gating deficits have also been correlated with specific symptoms of schizophrenia; Louchart-de la Chapelle and colleagues (2005) found that gating was significantly more impaired in a subgroup of schizophrenia patients with negative symptoms like flat affect. While responses to test stimuli were significantly higher in both the negative and non-negative symptoms schizophrenia groups in comparison to controls in Louchart-de la Chapelle and colleagues' (2005) study, this interesting evidence supports Knott and colleagues' (2009) notion that gating differs depending upon the presentation of the illness on an individual basis. These results are also very novel because one may expect impaired

gating to coincide with hallucinations and delusions, since gating impairment could be related to a problem with information filtering. The opposite is true, as patients with non-negative symptoms often exhibit normal gating profiles (Louchart-de la Chappelle et al., 2005). This evidence is extremely powerful and suggests that gating is tied in with stress and emotional experiences.

Post-Traumatic Stress Disorder

There is an increased chance that persons with post-traumatic stress disorder (PTSD) will experience perceptual disturbances similar to psychosis. Neylan and colleagues (1999) examined inhibitory gating function in Vietnam combat veterans who were diagnosed with PTSD and compared them to healthy controls. In this case, inhibitory or sensory gating was taken to be a measure of habituation. Habituation is impaired in persons with PTSD, especially in terms of startling stimuli. However, the P50 procedure used in this study was typical in that it did not involve intentional acoustic startle, as inhibitory gating is separate from the startle response. Analyses of P50 gating showed that PTSD patients had reduced responses to the repeated stimuli, and therefore had higher T/C ratios and impaired gating as compared to controls. These findings shed light on the nature of PTSD and reflect impairment in a process that typically deals with neutral stimuli, which means that brain abnormalities associated with PTSD are not limited to trauma-related cues and startling stimuli. A later study by Gjini and colleagues (2013) examined inhibitory gating in Iraqi refugees who had developed PTSD due to a history of torture exposure. This group was compared to Iraqi refugees with similar experiences who did not develop PTSD as well as controls with no history of trauma. The PTSD patients had significantly reduced P50 gating compared to the two other groups, and better gating

was correlated with higher quality of life scores. The findings of these studies further imply that inhibitory gating can be affected by emotion, stress, and anxiety.

Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) is an anxiety disorder that is separate from psychosis, yet gating impairments are still present. Dysfunction in the basal ganglia circuit has been theorized as a driving force in OCD symptoms, which are characterized by abnormal inhibitory responses and a ceiling effect of cortical excitability (Rossi et al., 2005). Rossi and colleagues (2005) purported that sensory gating deficits may be a result of the cortical hyper-excitability observed in OCD. Median nerve somatosensory evoked potentials were assessed in OCD patients and healthy controls who were instructed to relax or move their fingers in response to stimulation. The OCD patients exhibited reduced gating, which the authors interpret as a lack of control over inhibitory responses as well as a symptom of chronically high motor excitability in cortical areas, both of which are linked with basal ganglia dysfunction. Although this study did not use an auditory paired click paradigm, it still offers evidence that gating impairments may be related to chronic anxiety.

Alzheimer's Disease

A shared feature of schizophrenia and Alzheimer's disease is a cholinergic deficit and the specific dysfunction of alpha-7 cholinergic nicotinic subunits (Jessen et al., 2001). Post-mortem findings have shown alpha-7 receptor loss is associated with Alzheimer's disease, and as such, Jessen and colleagues (2001) suspected that inhibitory gating may also be disrupted in this neurodegenerative disorder. Using a paired click paradigm, they compared inhibitory gating in Alzheimer's patients to gating in healthy, age-matched controls. Gating ratios were significantly

higher in the Alzheimer's group, which supports the authors' theory that problems with P50 suppression are related to the compromised integrity of the cholinergic system observed in both Alzheimer's disease and schizophrenia. Interestingly, impaired gating was not correlated with any specific Alzheimer's symptoms such as cognitive status. This aligns with the findings of Olincy and colleagues (2000) revealing that inhibitory gating is not impaired in illnesses in which attention or cognition alone is affected, such as adult attention-deficit disorder. Jerger and colleagues (1992) also observed that P50 gating was not affected by attentional manipulations and suggested that the suppression process may be neuronal. Nonetheless, gating may be influenced by certain psychological factors like affect and stress.

Limitations

Although P50 components are a valuable tool in the study of various mental illnesses, reliability and interpretability still pose challenges. P50 suppression deficits have been studied extensively in schizophrenia research, but Knott and colleagues (2009) suggest that effect sizes reported by a meta-analyses of P50 studies by Heinrichs (2004) are mediocre ($M = 1.55$). Furthermore, differences between patients and healthy controls can be difficult to determine due to the varying ways mental illnesses are expressed on an individual level, as is the case with schizophrenia (Louchart-de la Chappelle, 2005) and Alzheimer's disease (Jessen et al., 2005). It is also possible that P50 suppression deficits occur in only a subset of the schizophrenic population (Heinreichs, 2004), and that alpha-7 receptor loss occurs in only a subset of the Alzheimer's disease population (Jessen et al., 2005) and puts these patients at higher risk of inhibitory gating impairment. These difficulties must be acknowledged and fully taken into account as research on gating impairment is translated into clinical use. More general issues with ERP in neuropsychology include difficulties with measurement accuracy, localization, and

replication (Luck, 2005). Nonetheless, ERP techniques are continually improving and undergoing refinements that allow conclusions from such data to be reached with confidence. Meta-analyses and procedure standardization are of primary importance in this regard.

2.4 Neuroanatomy of Gating

Localizing ERPs

Event-related potentials (ERPs) are measured by repeating sensory stimuli in a way that evokes stimuli-specific responses that can be differentiated from baseline activity (Neylan et al., 1999). Luck (2005) outlines how ERP components are typically defined by their latency (e.g., the time that elapses from stimulus presentation until response), polarity (positive or negative), and general distribution on the scalp (e.g., P50 gating at the Cz site in humans). Some experts hold that there is no infallible way to localize the source of an ERP signal and that localization efforts should be undertaken and interpreted only with great caution (Luck, 2005). However, Luck (2005) has several suggestions to increase validity for localization estimation: 1) Ensure data are clean; 2) Ensure there are only one to two dipoles; 3) Ensure that the electrical activity is concentrated; 4) Isolate individual components; 5) Use event-related magnetic field analyses (ERMF) in conjunction with EEG to improve resolution; and 6) Use magnetic resonance imaging (MRI) structural scans as well as fMRI functional scans to clearly define cortical regions.

Luck (2005) has many great points and concedes that probabilistic techniques may surpass the need for such expensive solutions. Spatial resolution is simply not the strong point of EEG or ERP techniques, and fMRI is too slow to visualize changes in real time. All the same, invasive techniques like single unit recordings are rarely an option with human participants, and practically speaking, ERP *does* provide useful spatial information if it is handled properly.

Investigators are continually improving upon sophisticated localization computations that account for the occurrence of a component at specific times and in response to specific stimuli in order to deduce the generation point of said component. Luck (2005) and other sceptics are correct that localization studies need to combat human error by rigorously testing and replicating evidence.

Attempts to Localize the P50 ERP in Humans

Several studies have attempted to localize the source of gating in humans. The larger goal of these efforts is to trace P50 suppression deficits to underlying neuroanatomical and neurochemical differences between healthy and mentally disordered persons (Knott et al., 2009). Bak and colleagues (2011) examined potential generation sources of gating in healthy controls using a combination of EEG and fMRI. Gating was evoked using mild electrical stimulation of the left median nerve. Though tones were not used, the procedures otherwise followed the paired click paradigm in terms of inter-stimulus intervals and paired presentations. Localization of neural sources was accomplished using a grand average model and automated computations. Analyses of EEG data revealed that gating could be localized to four larger source areas: the medial frontal gyrus, the insula, the hippocampus, and the primary somatosensory cortex. The fMRI data revealed additional localization regions: the cingulate gyrus, secondary somatosensory cortex, insula, thalamus, lenticular nucleus, and the caudate. Combined EEG and fMRI models revealed gating sources in the medial frontal gyrus, the insula, the claustrum, and the hippocampus. Successful gating correlated with higher activity levels in the hippocampus and claustrum, and lower activity in the medial frontal gyrus and insula. The authors interpreted this as lower activity areas being involved in generation and the higher activity areas as being involved in the inhibitory response. This ambitious study has shown that P50 suppression can be

measured by EEG and fMRI concurrently and accurately. The advantage of this combined technique is that clear spatial (fMRI) and temporal (EEG) information about gating can be deduced at once. Moreover, the successful of this work shows the pervasive nature of gating. It would be beneficial to extend this assessment combination technique to persons with schizophrenia in order to learn more about the neural origins of gating deficits.

To this end, Knott and colleagues (2009) explored localization of P50 gating using electromagnetic topography. Participants were divided into groups based on whether their conditioning amplitudes were higher (better gating = high suppressors) or lower (non-impaired but worse gating = low suppressors). The authors found gating activation in the limbic, temporal, and parietal regions in persons with high suppression, but interestingly, not in persons with low suppression and presumable gating impairment. Moreover, high suppressors with healthy gating also exhibited extended activation in the frontal lobes. These findings harken to Hutchinson and colleagues' (2013) developmental work that suggests P50 suppression deficits could be the result of differences seen early in development, such as the prevalence of anxious, depressive, and externalizing symptoms. These differences could subsequently help to explain differences in neural generation points of gating.

Korzyukov and colleagues (2007) deduced sensory gating neural generation points even further by implanting electrode grids on the frontal and temporal lobes of persons with epilepsy, and presenting them with an auditory paired click paradigm. The generators were described as areas in which brain activity alterations were associated with gating. This experiment found that the primary sensory gating generator could be localized to the temporal lobes, although the frontal lobes contributed neural activity that resulted in P50 suppression. This study had the advantage of a very precise electrode array, which helped to differentiate one generation point

from another. These findings strengthen those of scalp recording experiments and align with findings demonstrating impaired inhibitory gating in persons with dorsolateral prefrontal damage (Knight et al., 1999). Interestingly, the idea that P50 gating deficits can be defined as impairments in the test stimulus response is mirrored in the finding that frontal lobe generators are active only during test responses (Koryukov et al., 2007). A lack of modulatory inhibitory activity in the frontal lobes may therefore help to explain the reduction in test stimulus response and the subsequent impairment of P50 suppression in persons with schizophrenia. Overall, gating potentially spans multiple brain regions as the inhibitory response is generated and carried out. Moreover, gating is so ubiquitous that it can be observed in several areas at different points in time.

Brain Recordings, Gating, and Stress in Animal Models

Localization of the neural P50 gating source can be difficult to determine from human EEG scalp recordings, due to both a low signal to noise ratio and a stimulus response delay during which multiple ERP components occur (Korzyukov et al., 2007). Moreover, evoked potentials recorded from the scalp are the summation of all neural activity at that point in time and therefore all generator activity at that point in time. Animal models offer some advantages in this regard because recordings can be directly taken from individual cells, or single units, and assessed for gating individually with great precision. Single unit recordings are also not limited to cortical areas, as in human EEG.

Various efforts have been made to investigate brain areas in which gating can be observed in animal models. Cromwell and colleagues (2005) examined a rat model of inhibitory gating in the amygdala. The amygdala is a deep brain region within the limbic system that is involved in emotional processing and responses. Several amygdala gating response types were

found, including anticipatory responses to stimuli that the animals had become familiar with, inhibition occurring after stimulus presentation, and excitatory responses with short and long durations. The typical inhibitory gating pattern of a reduction in response to a second, identical stimulus was observed in all four response types. The animals in this experiment were also exposed to stress. Saline injections were used to induce acute stress, which led to impaired gating responses across all response types. The most pronounced results were in found in the short duration and anticipatory types, which may have implications for the human midlatency P50 component measure of gating.

Mears and colleagues (2009) conducted a follow up study that investigated whether a similar variety of responses could be found in the medial prefrontal cortex, which is thought to regulate signals to the amygdala (Liktik et al., 2005). Gating in both single unit responses and local field potentials were assessed in rats. Baseline inhibitory gating was evaluated as was gating after fear conditioning, in which one tone within click pairs became associated with an electric shock (Kim et al., 2013). Inhibitory gating was impaired for tones associated with the aversive stimulus. Inhibitory gating normalized, however, after extinction procedures in which the tone and shock were no longer paired together.

Gating has also been examined the striatum and midbrain in rat models. The striatum is an essential information processing center in the brain. Cromwell and colleagues (2007) proposed that inhibiting gating of sensory information is key to the successful integration of the various inputs in this region. Single unit recordings and local field potentials were measured, and interestingly, gating improved at the single unit and field potential levels in the striatum following acute injection stress. The midbrain is an ancient part of the nervous system and involved in motor control. Anstrom and colleagues (2007) assessed whether gating occurred in

single midbrain neurons in rats. If the animals were restrained as a form of stress, gating was impaired in midbrain neurons that were specifically GABAergic. This was reflected as a decrease in response to conditioning tones, which would be unusual for human gating impairment. Administration of haloperidol improved gating by increasing the conditioning response and therefore lowing the T/C ratio. This research suggests that inhibitory gating could be state dependent insofar as increases in dopamine neurotransmission can transiently impair gating by weakening the conditioning response and potentially gating as well.

Earlier work by Moxon and colleagues (1999) used single cell and auditory evoked potential recording techniques to assess gating in the CA3 hippocampal region, the medial septal nucleus, the brainstem reticular nucleus, and the auditory cortex of rats. Local auditory evoked potential gating was more prevalent in in the brainstem reticular nucleus and less prevalent in the auditory cortex. Gating activity occurred at various points in time and was correlated across regions with the exception of the auditory cortex. Single unit gating activity was highest in the brainstem reticular nucleus and the medial septal nucleus and lowest in the CA3 hippocampal region and auditory cortex. It is possible to infer from this work that auditory inhibitory gating is not generated in the cortex or thalamic pathway in rats. Rather, gating for auditory responses may begin the brainstem and continue through various structures like the hippocampus.

Although the animal models of gating are accurate to the level of individual neurons, the data from these models does not always match finding from human studies. This is problematic as the goal of many animal models is to ultimately better understand and research treatments for human dysfunctions. For example, Freedman and colleagues (1996) found that gating-related activity in the rat hippocampus did not translate to findings from human hippocampal recordings in which no gating-related activity was found (Grunwald et al., 2003). Even so, both human and

animal inhibitory gating experiments help clarify the roles of cognition and emotion in early processing by parsing out specific factors that can influence or disrupt the gating process. As such, it is beneficial to explore treatments for this gating impairments through both avenues.

CHAPTER 3

STRESS, AFFECT, MINDFULNESS MEDITATION, AND CURRENT AIMS

3.1 Stress, Affect, and Inhibitory Gating

Although almost all living things have to experience stress, it is not an easy thing to quantify. Generally speaking, stress is a physiological reaction to threats or challenges that require physical, psychological, or emotional adjustments. The stress response is influenced by many factors, including the controllability and predictability of stressful events (Lambert and Kinsley, 2004). Responses to stress vary and can be physical, psychological, or behavioral in nature. In any case, stress causes changes in the functioning of the body and the brain.

Cannon (1935) was the first to propose that the sympathetic nervous system restores the body to its normal state, homeostasis, after exposure to stress. Selye (1946) argued that the stress response was what he termed "general adaptation syndrome," as various types of physical stress elicited the same three-stage response: an organism attempts to defend itself against the stressor, the organism adapts to the stressor, and the organism is eventually exhausted by these efforts. Generally speaking, the autonomic system automatically controls bodily functions with the parasympathetic and sympathetic sub-systems, which respectively control the body's resting state and fight-or-flight (stressed) state.

However, Sapolsky (1996) argued that general adaptation syndrome was not a sufficient explanation of the stress response and proposed that a stressor's duration is central. An animal is more likely to habituate to a chronic stressor, whereas acute stressors have only transient physiological effects. Sapolsky (1996) further argued that stress intensity and the subsequent changes in stress hormone levels influence the stress response. Primate studies have shown that sustained high levels of glucocorticoids (an adrenal steroid hormone) cause a reduction in the resiliency of hippocampal cells (Lambert and Kinsley, 2004; Sapolsky, 1996). If intense stress

persists over many days, the damage to these cells can become permanent, and the cells are more likely to die (Lambert and Kinsley, 2004; Sapolsky, 1996).

In addition to initiating the release of stress hormones, stress can also cause increases in blood pressure, heart rate, breathing rate, and metabolic rate (Lambert and Kinsley, 2004; Sapolsky, 1996). Sapolsky proposed that these physiological changes evolved alongside the stress response in order to help animals escape from predators (Lambert and Kinsley, 2004). Stress can also have psychological effects that manifest as symptoms of anxiety, aggression, depression, and cognitive impairment (Lambert and Kinsley, 2004). Various stressors have been tested to see how they might affect inhibitory gating function.

Physical Stressors

There is strong evidence that inhibitory gating is a state dependent process in that stressors can alter its function. The cold-pressor task is used is used to elicit physical stress through the brief induction of pain. Johnson and Adler (1993) argued that physical stressors should have stronger effects on inhibitory gating than mental stressors, since physical stressors directly influenced autonomic nervous system arousal and increased levels of norephinephrine.

The cold-pressor task involves voluntarily keeping one hand in ice water (32-34°F) for several minutes, and it is considered safe enough for use in pediatric studies (von Baeyer et al., 2011). Johnson and Adler (1993) demonstrated that the cold-pressor task can impair inhibitory gating in healthy controls for up to 30 minutes. In this study, the P50 response to the conditioning stimulus was not altered after stress induction, while the response to the test stimulus was heightened in amplitude and caused an increase in T/C ratios. Amplitude and latency remained relatively stable. Half of the participants in the Johnson and Adler study exhibited significantly increased T/C ratios after the cold-pressor task. Thus, there appears to be

a great deal of individual variation in distress responses to the cold-pressor task and subsequent effects on inhibitory gating. A later study by Atchley and Cromwell (2013) replicated Johnson and Adler's (1993) finding that stress caused by the cold-pressor task can transiently impair inhibitory gating in healthy controls. In some ways, the results of these experiments contradict general adaptation syndrome. The acute stress did not cause a change in physiology. It is possible that other physical stressors could be more effective, but few are used in human studies other than controlled exposure to cold or hot temperatures. In animal studies, however, other physical stressors like injection have been used to assess gating (Cromwell et al., 2005). The magnitude of the physical stressor would likely impact gating performance, but this is a difficult ethical area to traverse, especially in humans.

Mental Stressors

Mental stressors can also impair inhibitory gating function. Mental stress differs from physical stress in this case due to the absence of aversive external stimuli. Frustrating cognitive tasks can be used instead of physical stressors to induce gating impairment. White and Yee (1997) had participants answer arithmetic questions aloud while inhibitory gating responses to auditory clicks were simultaneously recorded. Participants also completed a reaction time task, although this attentional manipulation had no effect on inhibitory gating. The oral arithmetic task was an effective psychological stressor on par with the cold-pressor task, although Johnson and Adler's (1993) physical stress task did induce a greater T/C ratio increase form baseline. Still, the oral arithmetic task was more effective in inducing stress in that gating was presumable impaired in more than half of the participants, unlike the cold-pressor. The oral arithmetic task induced inhibitory gating impairment to the degree that the White and Yee (1997) suggested it could be used to model schizophrenia-like inhibitory gating impairment in healthy persons.

Responses to the conditioning and test clicks were reduced for the oral arithmetic task as well as a silent arithmetic task, although results were more pronounced for the oral version. The decrease in the conditioning response from the passive baseline state is noteworthy, as inhibitory gating impairment is usually identified as a diminished test response. However, since the T/C ratios measure the test response relative to the conditioning response, it is reasonable to conclude that inhibitory gating is impairmed. A follow-up study by Yee and White (2001) demonstrated that psychological stress induced by a social stressor (prepare to give a speech) also impaired inhibitory gating. The White and Yee (1997) finding of stress-related suppression of the P50 response in the silent and oral arithmetic tasks were also replicated. Inhibitory gating was only disrupted by the social and silent arithmetic stressors when the participants rated them as anxiety-inducing. Like Johnson and Adler (1993), there was variation in how stress was experienced and subsequently how inhibitory gating responded to the stress, as White and Yee's (1997) average post-stress P50 ratio was .68, with a standard deviation of .39. Thus, some participants' post-stress ratios may still be in the normal range while others were greatly impaired.

Yee and White (2001) considered impaired gating to be the absence of a reduction in response to test stimuli relative to conditioning stimuli. The lack of a reduction is reflected as a higher post-stress T/C ratio in comparison to baseline inhibitory gating. Although the conditioning and test responses were individually sensitive to background noise and sound intensity, the T/C ratios remained constant. Participants listened to a speaking voice in addition to the auditory clicks or performed a silent counting task. The voice task provided auditory competition and successfully disrupted P50 gating. The counting task was a cognitive activity intended to distract attention, but it did not disrupt inhibitory gating unless a participant rated the

task as stressful. These findings suggest that competing auditory activity and stress can impair inhibitory gating, while cognitive and attentional manipulations may not. Moreover, the same task may be stressful and cause inhibitory gating impairment in one individual while being innocuous for another individual. This is further evidence of individual variation in the experience of stress and its potential to disrupt inhibitory gating. Another key difference here is that competing auditory stimuli and stress are related to the salience of the sensory stimuli more so than willful cognitive or attentional efforts. In other words, an attentional and cognitive distractions may not impair gating at the P50 level because they occur much later in processing. However, competing auditory stimuli and a stressed state alter how incoming sensory stimuli are processed early on and may impair gating in a way similar to Venables (1964) description of sensory flooding.

Affect and Inhibitory Gating

Additional findings indicate that inhibitory gating is also influenced by mood and emotion. It is important to note that while stress and negative emotion are sometimes related, they are not the same thing. Stress can be positive or negative in how it relates to arousal. An example of good stress would be excitement while watching a sporting event. Another potential example of good stress is the anxious feeling related to an exam that drives one to study. Bad stress can be related to negative mood in disorders like depression, or also manic episodes in bipolar disorder. In essence, stress goes beyond negative emotion in the variance of its possible expressions, both physiologically and psychological.

So, how might mood specifically affect gating? Negative mood states have been the primary focus of study. Higher anxiety scores in schizophrenia patients correlate with increased inhibitory gating impairments (Yee et al., 1998). Furthermore, Hutchison and colleagues (2013)

measured anxious and depressive symptoms in infants alongside other variables such as attention and externalizing behaviors that can be linked with potential behavioral problems in the future. Parents filled out inventories on these measures when the infants were 70 days old and later at 40 months old. Diminished P50 gating at 70 days of age predicted higher anxious and depressive symptoms at 40 months of age, although it should be noted that anxious and depressive symptoms in these children were deduced by their parents via non-diagnostic behavioral reports. Attentional and externalizing behavioral problems were also predicted by early life P50 gating disruptions. This is strong evidence that gating-related brain abnormalities may be detectable early in life and that early gating impairment could predict later psychopathology.

Animal models have also shown how emotion directly influences inhibitory gating function. Mears and colleagues (2009) conducted single cell recordings in the prelimbic cortices of rats and found that fear conditioning can briefly impair inhibitory gating in this region. Inhibitory gating was strengthened during extinction sessions for the learning behaviors related to this fear conditioning. Moreover, rats exposed to physical stress displayed weakened inhibitory gating in the amygdala, a region that is heavily involved in emotional processing (Cromwell et al., 2005). This is further evidence of the potential role of emotional state on inhibitory gating.

Additional studies on emotion in humans provide reasons to suspect that stress-induced inhibitory gating deficits can be reduced, as it has been shown that emotions can modulate the stress response and how stress itself is perceived by the person experiencing it. Leventhal and colleagues (1979) investigated this phenomenon in detail and tested whether physical stress was influenced by emotional and cognitive states. Their participants were given information on the cold-pressor task before they engaged in it. Neutral (low magnitude) information reduced

distress while emotionally arousing (high magnitude) information increased distress. Leventhal and colleagues observed that instructing participants to focus on and modulate sources of distress also resulted in reduced distress. This approach may facilitate habituation to stressors and possibly enable quicker recovery from stress-induced inhibitory gating impairment.

A later study by Ahles, Blanchard, and Leventhal (1983) replicated these effects for the cold-pressor task and provided more detail. Participants who attended to sensory aspects of a stressful task experienced reduced distress, while participants who shared emotions about the stressful task experienced increased distress. Distractions had no effect. Interestingly, the participants in this study had incorrectly predicted that attention to sensory information and emotion sharing would increase distress, whereas the distraction task would decrease distress.

Rhudy and Meagher (2001) further explored the role of anticipation and emotion on stress, and found that positive emotions reduce pain in low arousal situations whereas negative emotions reduce pain in high arousal situations. Negative emotions in low or moderate arousal situations actually increased pain reports. Rhudy and Meagher concluded that emotional states and arousal levels play central roles in how pain is perceived. This is strong additional evidence that emotions modulate how basic sensory information is processed.

Waldo and Freedman's (1986) work expands on this finding in terms of inhibitory gating. Participants who reported high levels of anxiousness and/or hostility at baseline displayed impaired inhibitory gating profiles without any sort of stress induction. Waldo and Freedman verified that a subtraction task distractor and active/passive motor activity levels did not affect the P50 response measure, and concluded that mood alone accounted for this difference in P50 suppression. This result builds upon previous work on stress and inhibitory gating because anxiety and hostility are often the product of stress (Johnson and Adler, 1993; Yee and White,

2001). Even in neurologically healthy persons, inhibitory gating is still a vulnerable process in that its function is susceptible to stress and emotional state. Given the pervasiveness of gating deficits in psychopathology and negative affect, it would be beneficial to develop therapies that target this deficit.

3.2 Mindfulness Meditation

Meditation and the Brain

Treatments for inhibitory gating impairment are relatively unknown. Nicotine is known to normalize gating, but the common method of smoking nicotine can also result in serious health problems (Ghisolfi et al., 2006). It is well-known that persons with schizophrenia have very high rates of smoking in comparison to the larger population. This may reflect self-medication for gating impairment. Another strategy has been to use nicotine to reduce schizophrenia symptoms and see if gating is affected as well (Olincy et al., 2006). Treatments for inhibitory gating impairment do not exist yet, as inhibitory gating has never been a target for treatment. Evidence suggests that healthy inhibitory gating function is state dependent, and it follows that treatment options encouraging the regulation of stress could ultimately reduce gating impairment. A top candidate for non-pharmaceutical gating treatment is mindfulness meditation, which has been extensively studied as a clinical tool (Davidson et al., 2003; Hözel et al., 20011; Jerath et al., 2012; Kabat-Zinn et al., 1992; Zeidan et al., 2011).

According to Zeidan and colleagues (2011), the main principles of mindfulness meditation are: 1) Focus on the present experience and breathing (Shamatha or calm abiding); and 2) Acknowledgement of thoughts and sensations without evaluation (Vipassana or insight into the nature of reality. Mindfulness meditation has been shown to have beneficial emotional

and physical effects in various ways. The neurophysiological effects of meditation have been assessed with functional magnetic resonance imaging (fMRI). This technique allows increases and decreases in blood flow and oxygenation to different brain areas to be measured. Research using fMRI has shown that focused meditation can increase blood flow to the frontal and parietal lobes (Newberg et al., 2010). These changes in blood flow can also correlate with changes in affect and behavior, as Goldin and Gross (2010) found that mindfulness-based stress reduction exercises reduce the number of negative emotional experiences in social anxiety patients. These changes also coincided with decreased activity in the amygdala, a brain region that is heavily involved in emotional processing, as well as increased activity in the inferior and superior parietal lobule, cuneus, precuneus, and middle occipital gyrus, which are regions associated with visual attention.

Jerath and colleagues (2012) have also noted selective inhibition in the thalamus, which plays a large role in relaying information to higher-order areas of the brain, and increased cortical connectivity associated with meditation. The neurophysiological changes in the brain observed after meditation training have been described as the product of a mind-body response in which the autonomic nervous system switches to a parasympathetic dominant state (Jerath et al., 2012). Mindfulness meditation in particular may prompt neural plasticity in attentional networks (Berkovich-Ohana et al., 2012).

MRI studies have shown that people who practiced daily meditation for an average of eight years had increased grey matter concentrations in the right anterior insula, left inferior temporal gyrus, and right hippocampus in comparison to matched controls (Hözel et al., 2008). The insula region is thought to be involved in introspection and awareness, which are emphasized in mindfulness meditation. In a follow-up to this study, non-meditators were trained

for eight weeks in stress-reduction mindfulness meditation techniques and displayed increased grey matter concentrations in the posterior cingulate cortex, temporo-parietal junction, and cerebellum (Hözel et al., 2011). Unlike the regions with increased gray matter concentrations in experienced meditators, the regions affected in novice meditators are involved in learning, memory, and emotion regulation. Thus, the effects of meditation may change over time and are still present after relatively brief interventions. Overall, mindfulness meditation is an excellent candidate for the treatment of inhibitory gating impairment given the varied and robust findings on mindfulness meditation's neurophysiological benefits.

3.3 Current Experiment

Rationale

Previous research has also shown that inhibitory gating has potential as a biomarker and diagnostic tool for schizophrenia (Freedman et al., 2005) and as a developmental predictor of attentional, anxious, and depressive symptoms (Hutchison et al., 2013). Affective factors could likewise influence inhibitory gating function, as inhibitory gating is impacted by physical stressors, psychological stressors, and anxiety levels (Johnson and Adler, 1993; White and Yee, 1997; Yee and White, 2001; Yee et al., 1998). The wide array of potential influences on inhibitory gating strongly suggests that the process is state dependent. It is essential to translate the neurophysiological techniques and findings in inhibitory gating research to clinical practice. Clinical neuroscience is playing an increasingly larger role in diverse areas of mental health work. The DSM-5 strongly encourages investigation into the biological underpinnings of mental illness in order to expand and better specify diagnosis and treatment options. The recent push to adopt reliable and valid clinical neurophysiological tools for prevention, diagnosis, and treatment

make the study of cost effective, rapid neurophysiology measures highly significant. Researchers demonstrated that inhibitory gating impairments are found in diverse mental illnesses (Boutros et al., 2004; Jin et al., 1996; Johnson and Adler, 1993; Louchart-de la Chappelle et al., 2005; Myles-Worsley et al., 1996; Neylan et al., 1999; Patterson et al., 2008; Rossi et al., 2005). Treatments that target inhibitory gating impairment are non-existent. One way to begin this work is to model inhibitory gating impairments in healthy controls by exposing them to stress and thereby inducing temporary inhibitory gating impairment. From there, the efficacy of potential treatment interventions can be explored.

Primary Aims

The current study is an example of this translational approach. The overall goal is to explore whether mindfulness meditation can reduce stress-related inhibitory gating impairment in healthy adults. Inhibitory gating impairment was modeled in healthy people using a physical stressor, the cold-pressor task, and participants were assigned to a control group (personality questionnaires), muscle relaxation group, or mindfulness meditation group. All participants attended four sessions. Inhibitory gating was assessed as P50 responses that were evoked with an auditory paired click paradigm and recorded using electroencephalography (EEG). EEG recordings were taken pre-/post-stress task and pre-/post training. Given previous evidence, the following outcomes were predicted: 1) Mindfulness meditation training will reduce stress-related inhibitory gating impairment; 2) Mindfulness meditation will reduce cold-pressor pain ratings; 3) Mindfulness meditation will reduce cold-pressor stress ratings; 4) Mindfulness meditation will reduce negative affect; and 5) Mindfulness meditation will reduce state anxiety.

CHAPTER 4

METHODS

4.1 Participants

Participants were recruited through the Bowling Green State University (BGSU) research participation website (https://bgsu.sona-systems.com/), and fliers posted throughout the city of Bowling Green, Ohio. The sample included undergraduate students, graduate students, and members of the community who were not affiliated with Bowling Green State University. All participants were required to be 18 or older, without current mental illness or history of neurological disease, without circulatory problems, and with normal hearing. Participants were also asked to refrain from non-prescription drugs and alcohol for 24 hours as well as nicotine for a minimum of 30 minutes prior to appointments (Johnson and Adler, 1993; Yee and White, 2001). Rescheduling was possible if the drug, alcohol, or nicotine time-tables were not met. Eligibility criteria were verified through participant self-report.

This study summarizes the data of 30 participants. Participants were randomly assigned to one of three groups (N = 10 per group): mindfulness meditation training, progressive muscle relaxation training, or Jungian personality survey controls. The majority of the participants were female (67%) and European-American (70%) with an age range of 18 to 36 years (M = 23). African-American, Asian-American, Hispanic, and Middle-Eastern participants composed 30% of the sample. Men and women were included in the study, as there is general agreement that inhibitory gating does not differ by sex (Boutros et al., 1999; Davies et al., 2009; Freedman et al., 1987; Louchart-de la Chappelle et al., 2005; Myles-Worsley et al., 1996). Four additional participants were recruited but ultimately excluded. Three were excluded due to technical malfunctions and one did not complete all four appointments. Partial data were not included.

4.2 Equipment

Neurophysiological data were collected using a Biopac MP150 module, three Biopac ERS100C amplifiers, and a Biopac CAP100C electroencephalography cap with 16 Ag/AgCl electrodes. Data were recorded from the Fz, Cz, and Pz sites, which are along the center of the scalp. Fz is a frontal measure, Cz is a central measure, and Pz is a posterior measure of electrical network activity in the cortex directly underneath these respective electrodes. The cap was held in place with two side straps that snapped onto a body band. Fz, Cz, and Pz connections were plugged into VIN+ ports on the amplifiers. Ear reference electrodes joined in a Y cable that was plugged into the center VIN- slot and securely connected to the other two amplifiers' VIN- slots via jumper cables. All electrode sites were abraded and filled with electrode gel. Impedances were below 30 kilohms. Although data were recorded from the Cz electrode site, these data were found to be anomalous. Subsequent testing revealed amplifier failure unique to the Cz site; therefore, Cz data are not included in subsequent analyses.

The cold-pressor task consisted of a large cooler filled with approximately five pounds of ice and enough water to create a basin. A clear plastic insert was used to separate the ice from the basin water in which participants would place their hands. A small aquarium pump was used to circulate the water and maintain a uniform temperature between 32-34°F.

4.3 Auditory Stimuli

Auditory clicks were generated with a Biopac STM100C stimulator at 40ms duration and 70dB (Dolu et al., 2001). The decibel levels were verified by a sound level meter. Clicks were presented binaurally through noise-cancelling Sennheiser headphones. Individual clicks within a pair were separated by 500ms, intertrial intervals were separated by 10 seconds, and each block

contained 41 trials of click pairs. The blocks were repeated before and after stress induction on both the first and last appointments. A single block lasted approximately seven minutes. Participants were offered breaks after each block.

4.4 P50 Component Analysis

EEG data were recorded and filtered with the AcqKnowledge 4.2 software program and band-pass filtered at 1.0 to 100 Hz during acquisition with a sampling rate of 1,000 Hz. Amplifier gain was set at 20,000 (Boutros et al., 2004; Johnson et al., 1993; Olincy et al., 2000; Yee et al., 2001). An analog filter of 1 to 100 Hz and a low pass filter of 3.0 kHz were applied as data were acquired with a sampling rate of 1,000 Hz (Gjini et al., 2013; Jerger et al.,). Data were digitally filtered at 10-50 Hz, since P50 most often occurs at a frequency of approximately 40 Hz (Arnfred and Chen, 2004; Boutros et al., 2004; Hutchison et al., 2013; Jessen et al., 2001; Yee and White, 2001; Jerger et al., 1992). P50 measurements were taken relative to the prestimulus baseline and identified individually as the maximum positive peak occurring within 30-90ms after stimulus onset (Zhang et al., 2012). P50 values that fell beyond two standard deviations above or below an individual participant's mean P50 score were identified as artifacts and removed from further analyses. Artifacts accounted for less than 2% of the data at the Fz and Pz electrode sites. Experimenters were blind to group assignment during P50 peak extraction.

4.5 Psychological Measurements

Participants filled out a demographic form on their first appointment. The cold-pressor task took place on the first and last appointments, and participants rated their experience of pain

during the task every 20 seconds. After the cold-pressor task was over, participants filled out a questionnaire assessing the stressfulness of the task. Mood (Positive and Negative Affect Scale or PANAS) and anxiety levels (State Anxiety Inventory or SAI) were assessed at the beginning and end of all four appointments. The mindfulness meditation group completed MAAS (Mindful Attention Awareness Scale) and FMI (Freiburg Mindfulness Inventory) inventories to assess their internalization of mindfulness techniques after each training session. All questionnaires were presented using professional online survey software (http://www.surveygizmo.com/). Please see the Appendices for the full instructions and text of each questionnaire.

4.6 Group Training

There were four sessions total for all groups and each session lasted approximately 15 minutes. All sessions were held individually with one experimenter and one participant. Meditation participants were guided through a mindfulness meditation exercise adapted from Segal and colleagues (2002) that focused on acceptance, attending to the present moment, and focusing on breath. The relaxation group was guided through an exercise that involved tensing and releasing tension from various muscle groups in the body (adapted from the Inner Health Studio, 2012). Experimenters in charge of guided sessions were trained by Bowling Green State University clinical psychology colleagues in proper techniques. The Jungian personality survey (adapted from HumanMetrics, 2012) was administered to control group members, but the answers were not scored as the purpose of this was to allow the control group an equivalent amount of interaction with an experimenter. Please see the Appendices for the full text of the training exercises and personality survey.

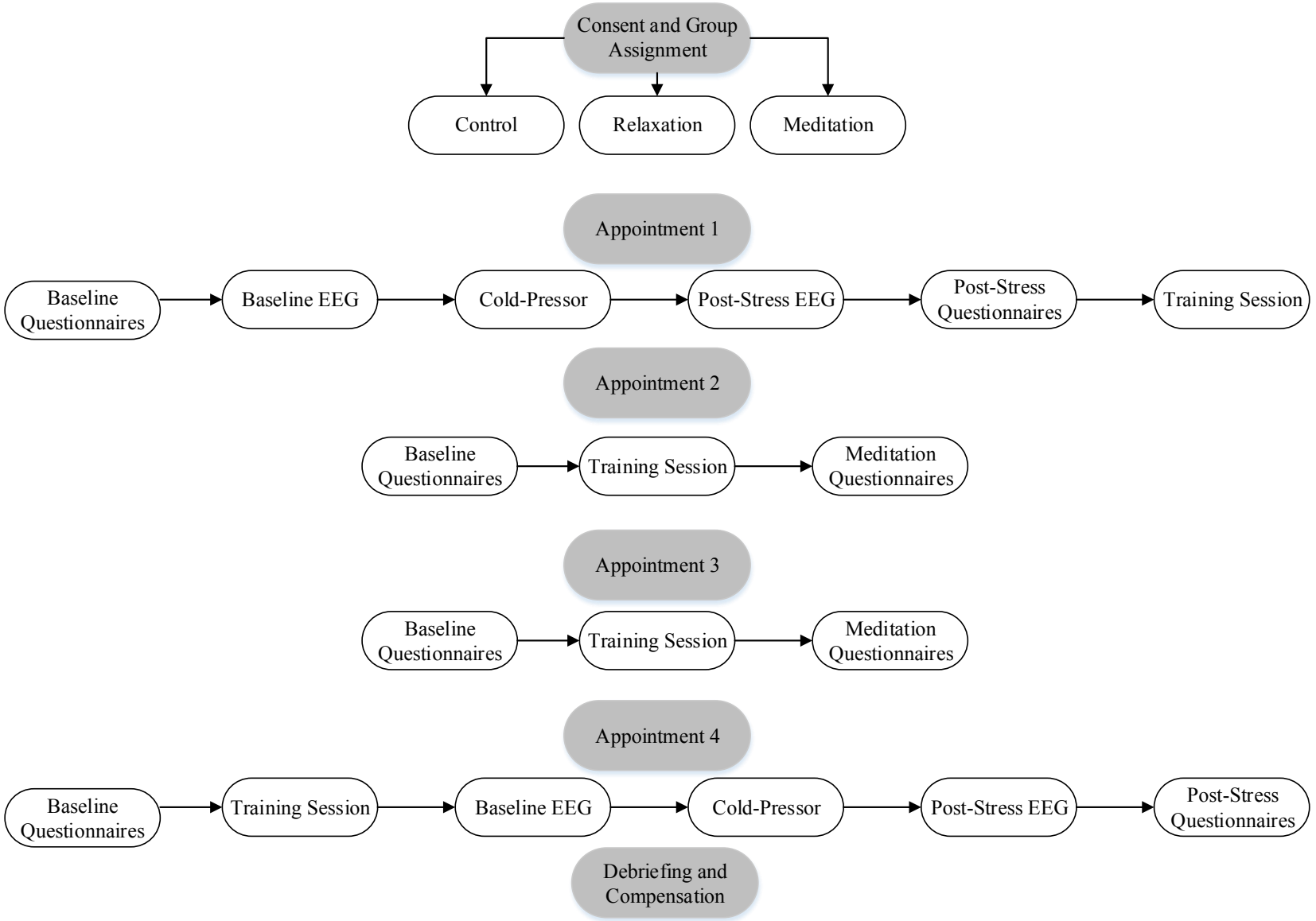
4.7 Procedures

The experiment was spread out over four appointments, with the first and final appointments lasting approximately 1.5 hours with EEG and the second and third appointments lasting approximately 20 minutes (see Appendix L for a visual of the EEG setup on a participant). For the first appointment, participants completed screening and consent procedures (see Appendix M) then filled out the demographic questionnaire, the PANAS, and the SAI. The participant combed and parted their hair and put on the EEG cap. Side straps on the cap were attached to a body band, which held the cap in place. The Fz, Cz, Pz, and reference electrode sites on the cap were abraded and filled with gel. Impedances were assessed using a UFI 1089NP Checktrode and kept as low as possible, with a range of <1 to 30 kilohms. Participants sat in a comfortable chair and were instructed to sit still, close their eyes, relax, and listen to clicks for seven minutes through headphones. Participants were offered a short break afterwards. Next, participants read instructions for the cold-pressor task and were reminded that they could terminate the task at any time. Each participant placed one hand in the cold water basin for up to two minutes, at which point the experimenter ended the task. During the cold-pressor task, participants looked at a pain rating scale on a computer screen and provided the experimenter with a rating every 20 seconds. The experimenter recorded these responses for the participants. Afterwards, the participant completed another inhibitory gating block lasting seven minutes. The EEG cap and body band were then removed. Participants completed the PANAS and SAI again, and only participants in the meditation group completed a MAAS and FMI. Training or personality surveys followed, and the next appointment was scheduled within 48 hours.

The second and third appointments were identical to one another. Participants filled out PANAS and SAI forms and completed a training session or personality survey, while meditation

participants also filled out the MAAS and FMI. The fourth and final appointment was very similar to the first with the exception that training took place before the cold-pressor task and participants in the meditation group filled out MAAS and FMI inventories before the cold-pressor as well. All participants were debriefed when they finished the experiment and compensated with cash or course credit. See Figure 1 for a visual summary of the experimental procedures.

Figure 1. Summary of Experimental Procedures



4.8 Statistical Analyses

The first stimulus within an auditory paired click pair is identified as the conditioning stimulus (C), which is intended to familiarize the participant with the tone, while the second stimulus is identified as the test stimulus (T), which is an evaluation of the inhibitory circuit's integrity. A 2x2x2x3 omnibus ANOVA was conducted to assess overall effects and interactions for the four main variables. Testing stimulus/conditioning stimulus (T/C) ratios were calculated for P50 responses to pairs of auditory clicks and assessed using 2x2x3 and 2x3 mixed ANOVAs. The ratio calculation was the mean T amplitude divided by the mean C amplitude, which was then multiplied by 100 to yield a percentage (Davies et al., 2009; Dolu et al., 2001). Ratios were compared at pre-training baseline and post-stress, as well as post-training baseline and post-stress, to evaluate inhibitory gating function. If ANOVA analyses were significant, potential group differences were evaluated with independent sample t-tests. Non-parametric analyses were used for questionnaire data, as these data did not meet the assumption of normal distribution. Friedman tests were used for between-group comparisons and Mann-Whitney tests were used for pairwise comparisons. Pearson correlational analyses were conducted on all data. Analyses were conducted in IBM SPSS Statistics version 20.

CHAPTER 5

RESULTS

5.1 Omnibus Results

A mixed 2 (pre- or post-training) by 2 (pre- or post-stress) by 2 (Fz or Pz electrode site) by 3 (control, relaxation, or meditation group) omnibus ANOVA evaluated all main variables' effects and interactions. Training, stress, and electrode site are within-group factors while group is the only between group factor. There was main effect of electrode site, $F(1, 27) = 21.60$, $p < .001$, meaning that gating ratios at the Fz and Pz electrode sites differed under certain conditions. There was also a main effect of training, $F(1, 27) = 4.46$, $p = .046$. Overall, gating ratios differed before and after four sessions of training. There was a main effect of stress as well, $F(1, 27) = 480.98$, $p < .001$. Gating ratios increased after stress induction as expected. A training by stress by group interaction was also present, $F(1, 27) = 5.72$, $p = .009$. The following analyses will test and specify main effects for all variable levels and clarify what the interactions entail.

5.2 S1 and S2

Conditioning clicks (S1), as the name suggests, are meant to familiarize participants with the auditory stimulus. In healthy persons, the P50 response to S1 is usually high in amplitude, while the P50 response test clicks (S2), which serve as an evaluation of the inhibitory circuit, are typically lower in amplitude (Chang et al., 2011). When gating ceases to function normally, P50 responses to S1 and S2 no longer fit this profile. For example, P50 responses to S1 can be lower in amplitude than responses to S2, or S1 and S2 responses may become more similar to each other (Chang et al., 2011). These changes can be due to a decrease in the response to S1, an increase in response to S2, or a combination of both.

S1 and S2 response types were not included in the omnibus ANOVA because they were not independent variables. However, S1 and S2 P50 responses were compared separately to elucidate which type(s) of impairment profile(s) were elicited through stress exposure. Only one prototypical gating impairment profile was expected, specifically an increase in S2 amplitude relative to S1 (Chang et al., 2011). This was the overall finding for both electrode sties, as detailed below.

Two (pre- or post-stress) by 3 (control, relaxation, or meditation group) mixed ANOVA analyses were conducted to compare S1 to S1 and S2 to S2 at the pre- and post-training stages. There were differences between the Fz and Pz electrode sites in the results of these analyses. At the Fz site, only pre-training P50 responses to S2 differed before and after stress, $F(1, 27) = 26.28$, $p < .001$. Responses were lower at baseline ($M = .0025$) and higher after stress induction ($M = .0038$). There were no other significant effects for S1 or S2 P50 responses at the Fz electrode site (Tables 1 and 2).

At the Pz electrode site, P50 responses to S2 prior to training also differed before and after stress, $F(1, 27) = 140.73$, $p < .001$. Like Fz, baseline responses ($M = .0029$) were lower in amplitude than post-stress responses ($M = .0042$), although the difference was larger for Pz. However, there were also post-training stress effects for S2 at the Pz site, $F(1, 27) = 36.28$, $p < .001$. Again, baseline P50 responses ($M = .0025$) were lower in amplitude than post-stress responses ($M = .0033$). Pre-training P50 responses displayed the only stress effect for S1 at the Pz site as well, $F(1, 27) = 4.31$, $p = .048$. This impairment profile is different in that baseline P50 responses ($M = .0036$) were similar to post-stress responses ($M = .0037$). There were no significant group effects for Fz or Pz P50 responses to S1 or S2 (Tables 3 and 4).

Table 1. Pre-Training Summary of S1, S2, and T/C Ratios, Fz Site

		Control		Relaxation		Meditation		Grand Average	
Trial Parameters		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Baseline									
Amplitude (μV)									
	S1 (Conditioning)	3.4	2.4	3.3	0.8	4.0	2.7	3.6	2.1
	S2 (Test)	2.4	1.7	2.2	0.6	2.9	1.9	2.5	1.5
T/C ratio (%)		68.4	17.3	70.0	18.1	73.0	15.2	70.5	16.5
Post-Stress Induction									
Amplitude (μV)									
	S1 (Conditioning)	2.8	0.9	3.9	1.3	4.2	2.9	3.6	2.0
	S2 (Test)	3.0	1.1	3.9	0.9	4.5	2.8	3.8	1.8
T/C ratio (%)		107.5*	19.7	111.7*	15.5	108.1*	11.2	109.1*	15.5

* Significant change from baseline, $p < .001$.

Table 2. Post-Training Summary of S1, S2, and T/C Ratios, Fz Site

		Control		Relaxation		Meditation		Grand Average	
Trial Parameters		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Baseline									
Amplitude (μV)									
	S1 (Conditioning)	6.0	9.5	2.9	0.9	6.3	7.1	5.1	6.8
	S2 (Test)	4.1	7.4	2.0	0.7	4.2	5.4	3.5	5.2
T/C ratio (%)		60.6	14.9	70.2	11.5	65.5	6.7	65.4	11.8
Post-Stress Induction									
Amplitude (μV)									
	S1 (Conditioning)	4.4	4.3	2.8	0.9	3.7	1.8	3.6	2.7
	S2 (Test)	4.8	4.4	3.1	1.1	3.5	1.8	3.8	2.8
T/C ratio (%)		111.3*	7.1	107.8*	13.7	96.6*	10.1	105.2*	12.1

* Significant change from baseline, $p < .001$.

Table 3. Pre-Training Summary of S1, S2, and T/C Ratios, Pz Site

		Control		Relaxation		Meditation		Grand Average	
Trial Parameters		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Baseline									
Amplitude (μV)									
	S1 (Conditioning)	3.4	1.3	3.8	1.8	3.6	1.0	3.6	1.4
	S2 (Test)	2.9	0.8	3.0	1.3	2.7	0.7	2.9	1.0
T/C ratio (%)		88.7	12.9	81.1	9.9	74.5	10.0	81.4	12.2
Post-Stress Induction									
Amplitude (μV)									
	S1 (Conditioning)	3.4	1.2	3.8	1.8	4.0	1.3	3.7	1.4
	S2 (Test)	3.8	0.9	4.5	1.6	4.2	1.2	4.2	1.2
T/C ratio (%)		112.1*	12.2	119.9*	14.7	108.4*	10.7	113.4*	13.1

* Significant change from baseline, $p < .001$.

Table 4. Post-Training Summary of S1, S2, and T/C Ratios, Pz Site

		Control		Relaxation		Meditation		Grand Average	
Trial Parameters		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Baseline									
Amplitude (μV)									
	S1 (Conditioning)	3.5	0.6	3.2	0.6	3.4	0.5	3.4	0.6
	S2 (Test)	2.7	0.5	2.4	0.5	2.7	0.4	2.6	0.5
T/C ratio (%)		76.2	12.5	74.4	8.1	80.1	12.9	76.9	11.2
Post-Stress Induction									
Amplitude (μV)									
	S1 (Conditioning)	3.6	0.4	3.3	1.0	4.0	0.8	3.6	0.8
	S2 (Test)	3.3	0.5	2.9	1.0	3.8	0.8	3.3	0.9
T/C ratio (%)		118.9*	17.0	116.6*	10.8	105.7*	11.3	113.7*	14.1

* Significant change from baseline, $p < .001$.

5.3 Fz Site Analyses

An omnibus 2 (pre- or post-stress) by 2 (pre-or post-training) by 3 (meditation, relaxation, or control group) mixed ANOVA was conducted for the Fz scalp site. A main effect of training was observed, $F(1, 27) = 318.98$, $p < .001$. No main effects were found for stress, $F(1, 27) = 3.45$, $p = .074$, $p = .074$ or group, $F(2, 27) = .52$, $p = .60$. There were no significant interactions for stress by group $F(2, 27) = 1.11$, $p = .34$, stress by training $F(2, 27) = .09$, $p = .77$, training by group, $F(2, 27) = 2.42$, $p = .11$, or stress by group by training $F(2, 27) = 1.70$, $p = .20$.

These findings were further explored as two (pre- or post-stress) by 3 (meditation, relaxation, or control group) mixed ANOVA analyses, which revealed an effect of stress on gating prior to training at the Fz electrode site, $F(1, 27) = 136.15$, $p < .001$. T/C ratios were higher after the cold-pressor task ($M = 109.1$) than at baseline ($M = 70.5$). As expected, there was no effect of group at baseline or post-stress before training. There were also no significant differences in baseline ratios between the first and last appointments, which indicates baseline ratio stability over time (Figure 2).

Participants experienced four sessions of mindfulness mediation training, progressive muscle relaxation training, or Jungian personality questionnaires based on group assignment. The effect of stress on gating was replicated at the end of training, $F(1, 27) = 237.46$, $p < .001$. Baseline T/C ratios ($M = 65.4$) increased after the cold-pressor task ($M = 105.2$). There was also a post-training main effect of group, $F(2, 27) = 4.98$, $p < .014$. Independent sample t-tests were calculated to clarify group differences after four sessions of training. Post-stress T/C ratios did not differ between control ($M = 111$) and relaxation ($M = 108$) groups. However, the meditation group ($M = 97$) had significantly lower post-stress ratios than the relaxation group after four

sessions of training, $t(18) = 2.09$, $p = .05$. The meditation group also had significantly lower post-stress ratios than the control group after training, $t(18) = 3.76$, $p < .001$. These findings offer strong evidence that meditation training helped reduce inhibitory gating impairment after exposure to stress, while muscle relaxation training and a control condition of personality questionnaires did not (Figure 3).

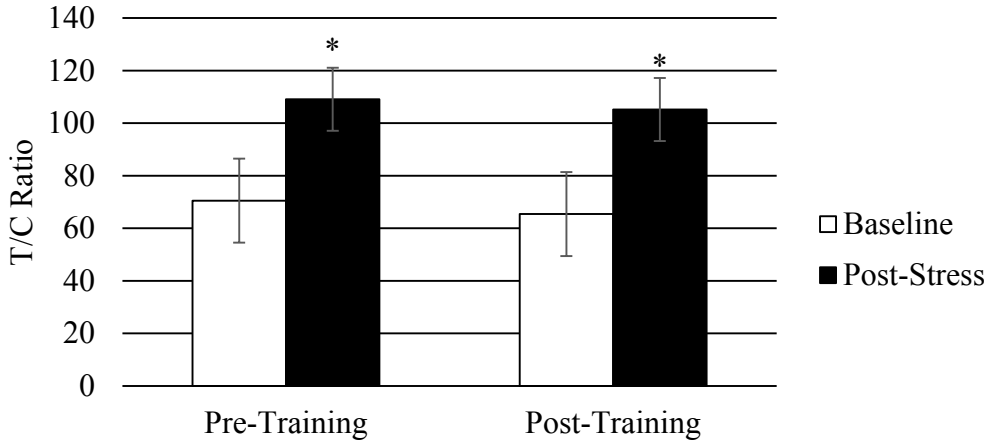
5.4 Pz Site Analyses

All Pz analyses were identical to those in the Fz section description. The omnibus ANOVA revealed another main effect of training, $F(1, 27) = 239.59$, $p < .001$. There was also a significant interaction amongst stress, training, and group, $F(2, 27) = 6.10$, $p = .007$. As with the Fz site, no main effects were found for stress, $F(1, 27) = 1.38$, $p = .074$, $p = .25$ or group, $F(2, 27) = 2.06$, $p = .15$. There were also no significant interactions for stress by group $F(2, 27) = 1.12$, $p = .34$, stress by training $F(2, 27) = 2.20$, $p = .15$, or training by group, $F(2, 27) = 2.05$, $p = .15$. Further analyses revealed that prior to training, baseline T/C ratios ($M = 81.4$) increased after the cold-pressor task ($M = 113.4$), $F(1, 27) = 237.46$, $p < .001$. There was no group effect, nor were there baseline ratio differences between first and last appointments, all of which is in line with findings at the Fz site (Figure 4).

After four sessions of training, baseline T/C ratios ($M = 76.9$) again increased after stress ($M = 113.7$), $F(1, 27) = 166.27$, $p < .001$. There was also a main effect of group, $F(2, 27) = 3.89$, $p = .033$. Independent sample t-tests revealed group differences that were similar to Fz site group effects. Pz post-stress T/C ratios did not differ for relaxation ($M = 117$) and control ($M = 119$) groups after training. Relaxation and meditation ($M = 106$) group differences bordered on significance, $t(18) = 2.04$, $p = .056$, with the meditation group trending toward lower post-stress

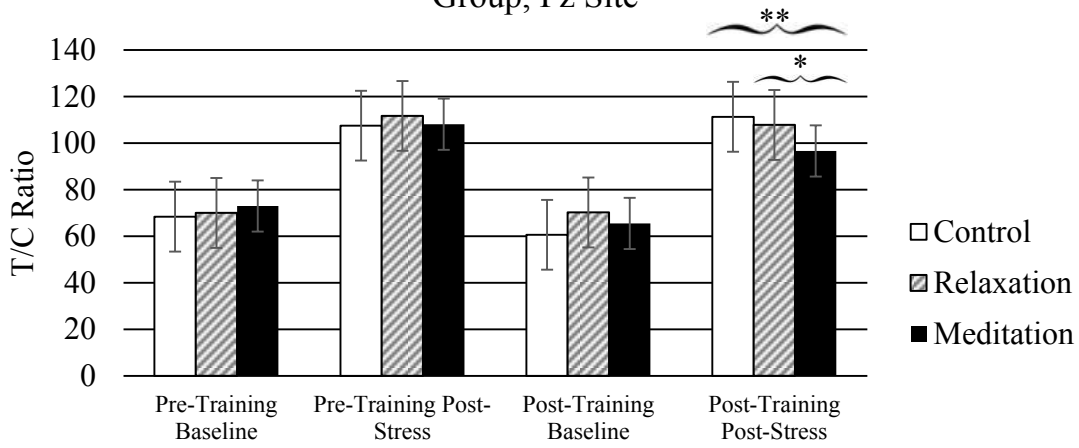
ratios after training. The significant difference between the control and mediation groups was replicated, $t(18) = 2.21$, $p = .04$, with the meditation group again exhibiting lower post-stress ratios after training (Figure 5). These findings suggest that meditation training effects on gating are more pronounced at the Fz site.

Figure 2. Training Effects on Gating Ratios across Groups, Fz site



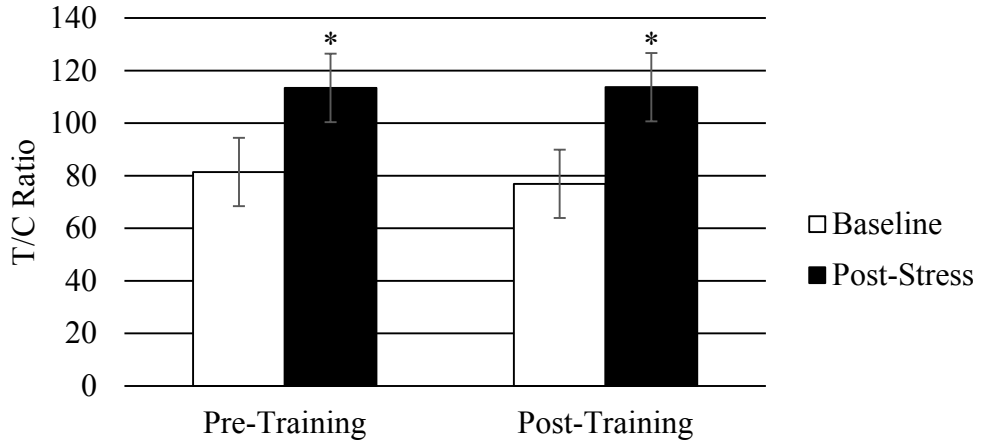
* Indicates significant increase from baseline, $p < .001$.

Figure 3. Training Effects on Baseline Gating Ratios by Group, Fz Site



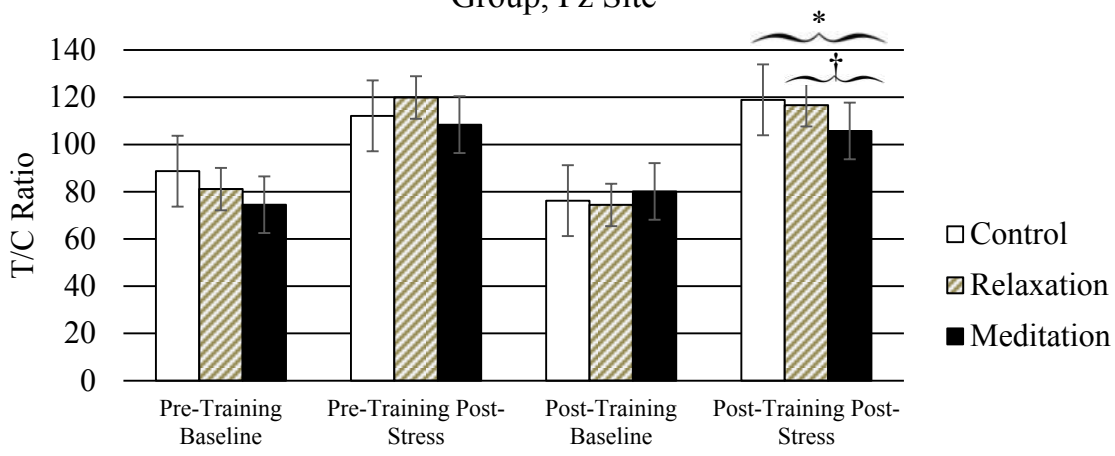
* Indicates significant group difference within condition, $p = .05$.
**Indicates significant group difference within condition, $p < .001$.

Figure 4. Training Effects on Gating Ratios across Groups, Pz site



* Indicates significant increase from baseline, $p < .001$

Figure 5. Training Effects on Baseline Gating Ratios by Group, Pz Site



*Indicates significant group difference within condition, $p = .04$.
† Indicates trend group difference within condition, $p = .056$.

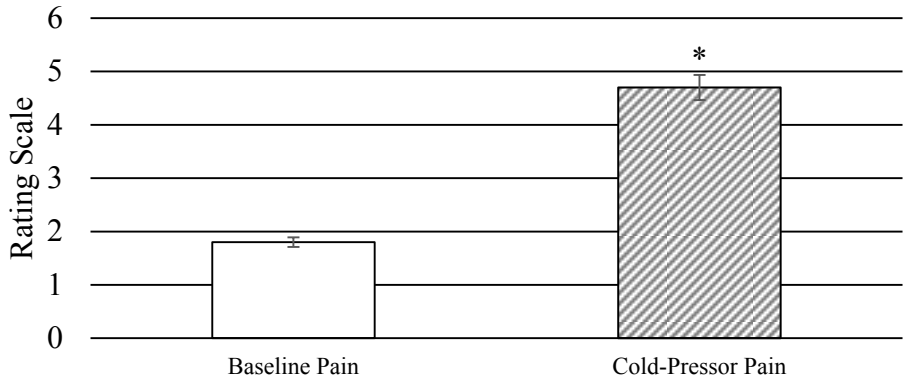
5.5 Stress Measures

Participants were asked to rate their levels of physical pain and psychological stress at baseline and after the stress induction task. Prior to training, ratings significantly increased from baseline for physical pain, $\chi^2(1) = 20.17$, $p < .001$, and psychological stress, $\chi^2(1) = 23.0$, $p < .001$, after the cold-pressor task. After training, ratings again increased with stress induction for physical pain, $\chi^2(1) = 23.15$, $p < .001$, and psychological stress, $\chi^2(1) = 21.16$, $p < .001$ (Figures 6 through 9). This indicates the effectiveness of the task in inducing pain and stress, but it is important to note that participants were informed that they were free to stop the task at any time, and that the cold-pressor is safe with no known long-lasting effects (Johnson and Adler, 1993; Leventhal et al., 1979; von Baeyer et al., 2011). Most participants (73%) endured the task for the full two minutes on the first appointment, and even more participants (83%) kept one hand in for two minutes on the last appointment. The stressful effects of the cold-pressor remained stable over time, as there was no significant difference between first and last appointment ratings of the painfulness of the cold-pressor task.

Mann-Whitney tests were conducted to explore potential group differences in stress and pain ratings for the cold-pressor task. There were no significant differences between groups before training for stress or pain ratings (Figures 10 and 12). After training, the meditation group rated the cold-pressor as less painful, $U(1) = 20.5$, $Z = -2.12$, $p = .034$ as well as less stressful, $U(1) = 20.0$, $Z = -2.10$, $p = .036$ than the control group (Figures 11 and 13). The relaxation group had no significant differences in pain or stress ratings in comparison to other groups. The McGill pain scale administered during the cold-pressor task yielded no changes over time between or within groups (Figures 14 and 15). Note that the stress measure data is non-

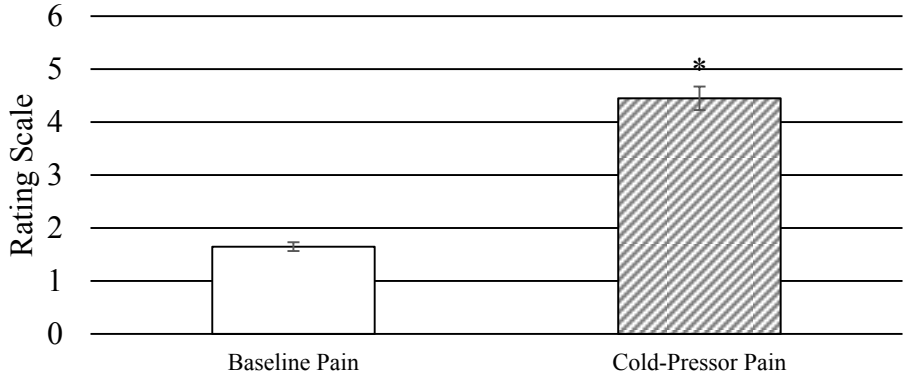
parametric and not normally distributed; therefore the error bars in Figures 6 through 15 represent 95% confidence intervals.

Figure 6. Pre-training Pain Ratings, across Groups



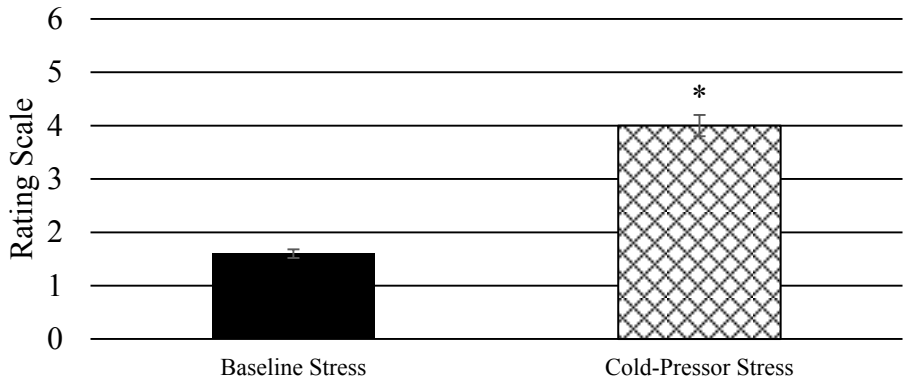
*Indicates significant increase from baseline, $p < .001$.

Figure 7. Post-Training Pain Ratings, across Groups



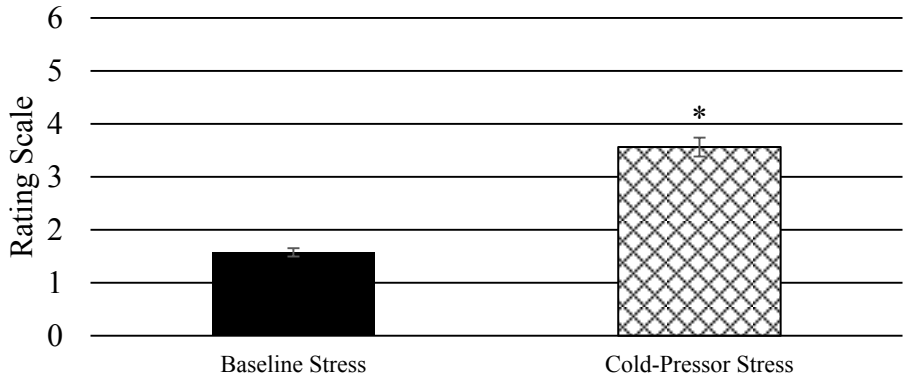
*Indicates significant increase from baseline, $p < .001$.

Figure 8. Pre-Training Stress Ratings, across Groups



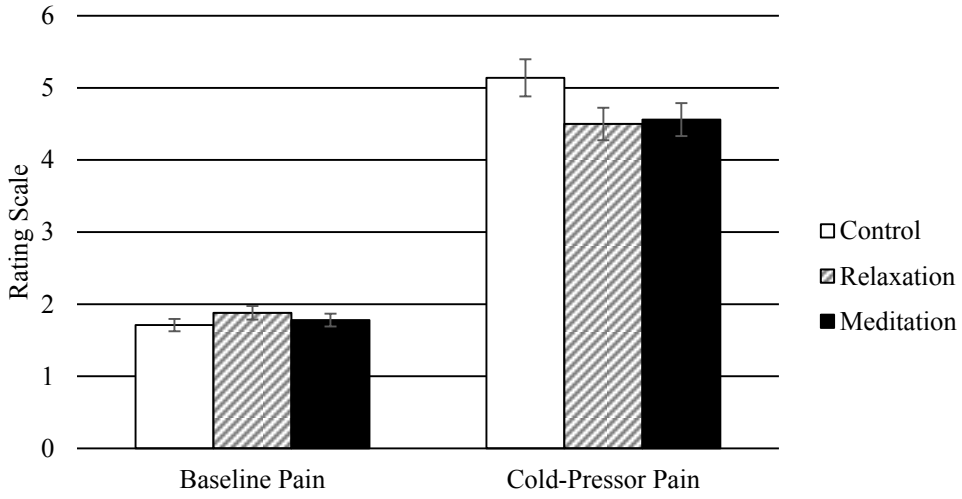
*Indicates significant increase from baseline, $p < .001$.

Figure 9. Post-Training Stress Ratings, across Groups



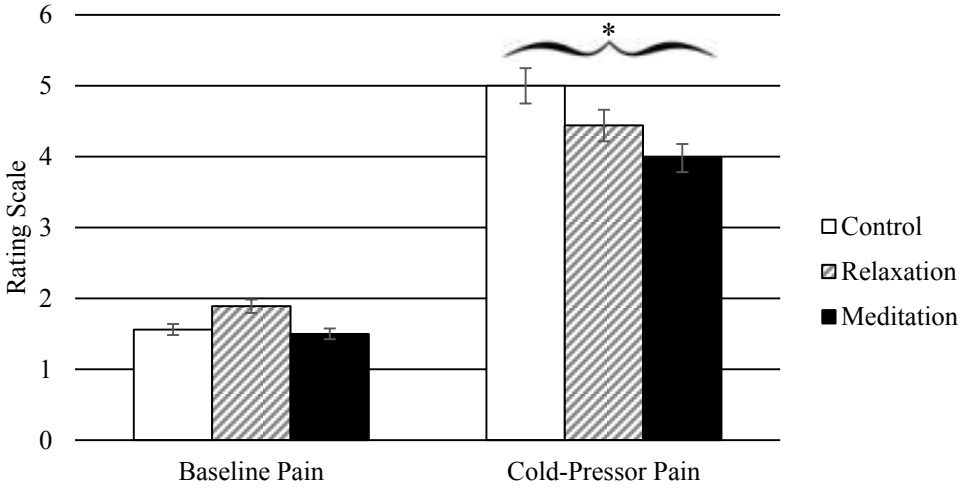
*Indicates significant increase from baseline, $p < .001$.

Figure 10. Pre-training Pain Ratings, by Group



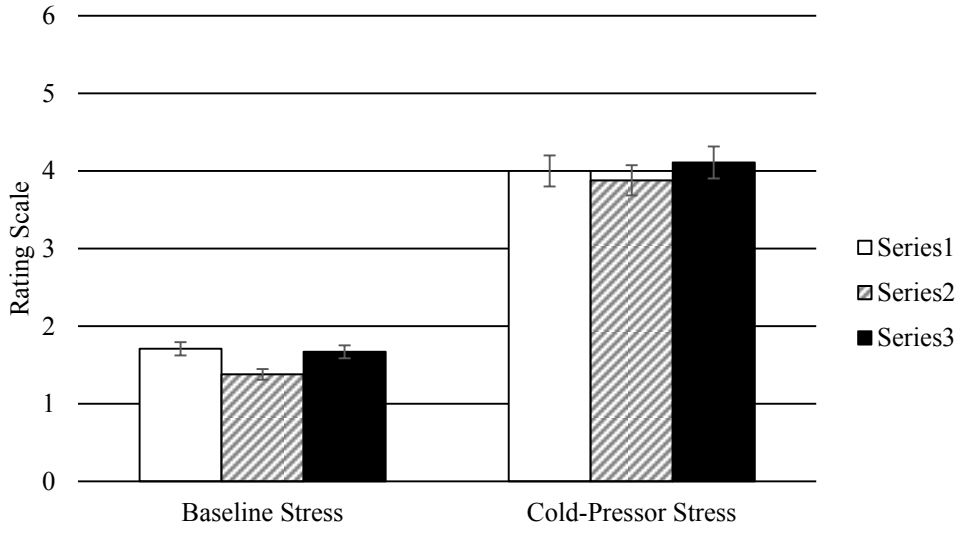
*No significant differences.

Figure 11. Post-Training Pain Ratings, by Group



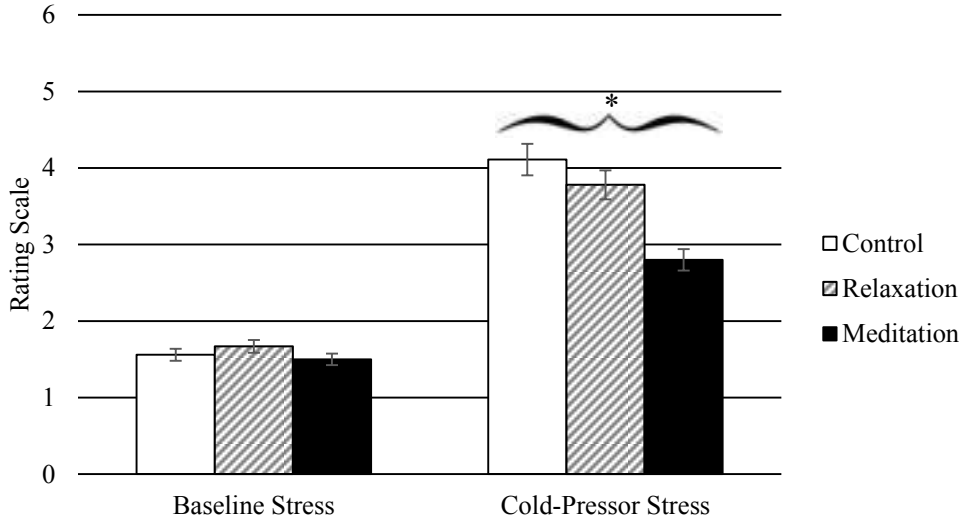
*Indicates significant group difference, p = .034.

Figure 12. Pre-Training Stress Ratings, by Group



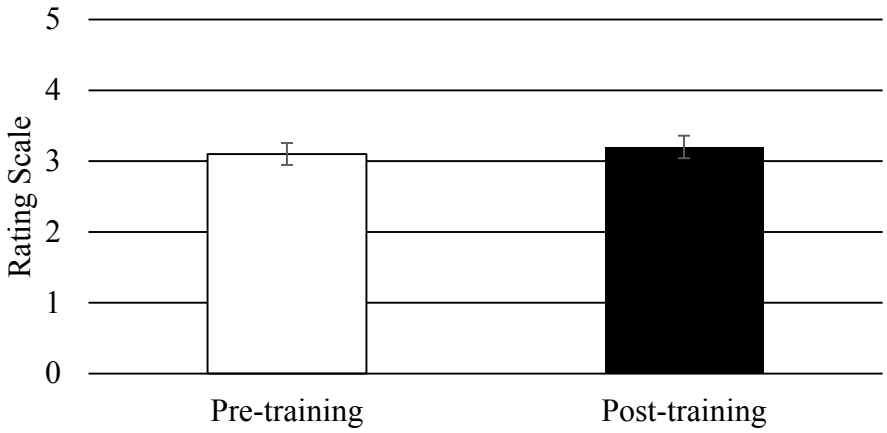
*No significant differences.

Figure 13. Post-Training Stress Ratings, by Group



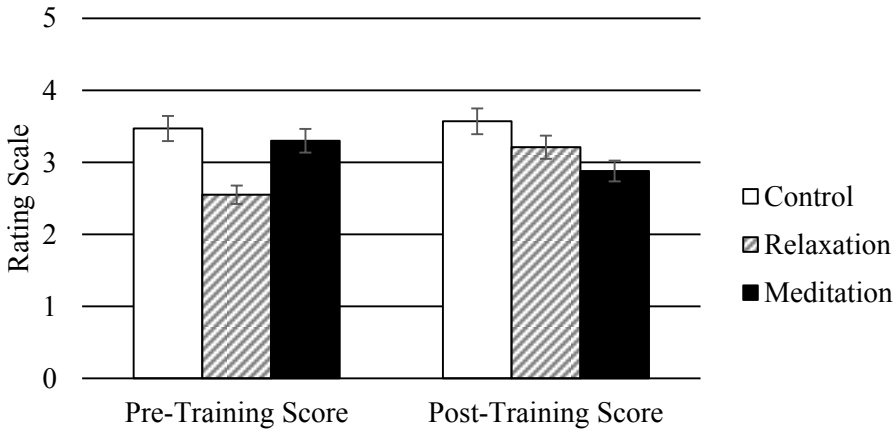
*Indicates significant group difference, p = .036.

Figure 14. McGill Pain Scores across Groups



*No significant differences.

Figure 15. McGill Pain Scores by Group

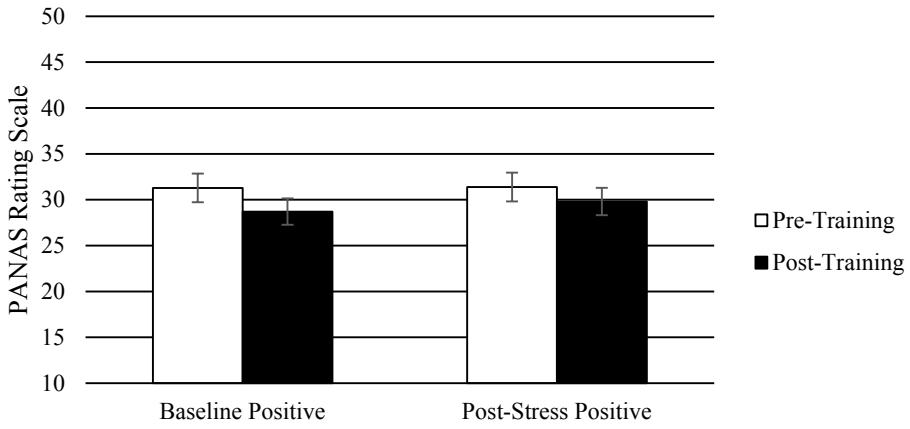


*No significant differences.

5.6 Psychological Measures

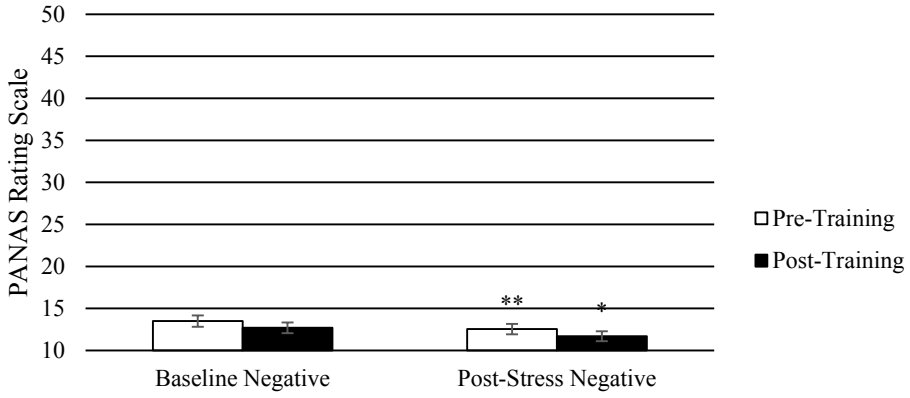
The PANAS questionnaire assessed positive and negative mood while the SAI questionnaire assessed feelings of anxiety versus feelings of calm. Participants used scales to rate how much they identified with various statements on these questionnaires. For the SAI, ratings on state anxiety level questions were summed. The positive and negative ratings were separately summed for the PANAS on each individual administration. Both the PANAS and SAI were administered before and after stress induction on the first and last appointments. Friedman tests were used to evaluate between-group differences on these questionnaires. Post-stress positive mood did not significantly differ from baseline either before or after training (Figures 16). However, negative affect scores were significantly lower after stress induction for the first appointment, $\chi^2(1) = 8.91$, $p = .003$ as well as the last appointment, $\chi^2(1) = 5.76$, $p = .016$ (Figure 17). To elucidate potential group differences, Mann-Whitney tests were applied to the significant findings for negative mood. These revealed no group differences for positive affect (Figures 18 and 19). There were no group differences in negative affect scores before training (Figure 20), yet after training, the relaxation group had significantly lower negative affect scores than the control group at baseline, $U(1) = 16.5$, $Z = -2.57$, $p = .01$ and post-stress, $U(1) = 18.0$, $Z = -2.52$, $p = .01$ (Figure 21). The meditation group also had lower negative affect scores than the control group at baseline, $U(1) = 16.5$, $Z = -2.57$, $p = .01$ and post-stress, $U(1) = 22.5$, $Z = -2.15$, $p = .03$ after training had taken place. There were no significant differences between the meditation and relaxation groups. There were no significant differences in SAI scores due to training or stress induction (Figures 22 through 24).

Figure 16. Positive Affect Scores, across Groups



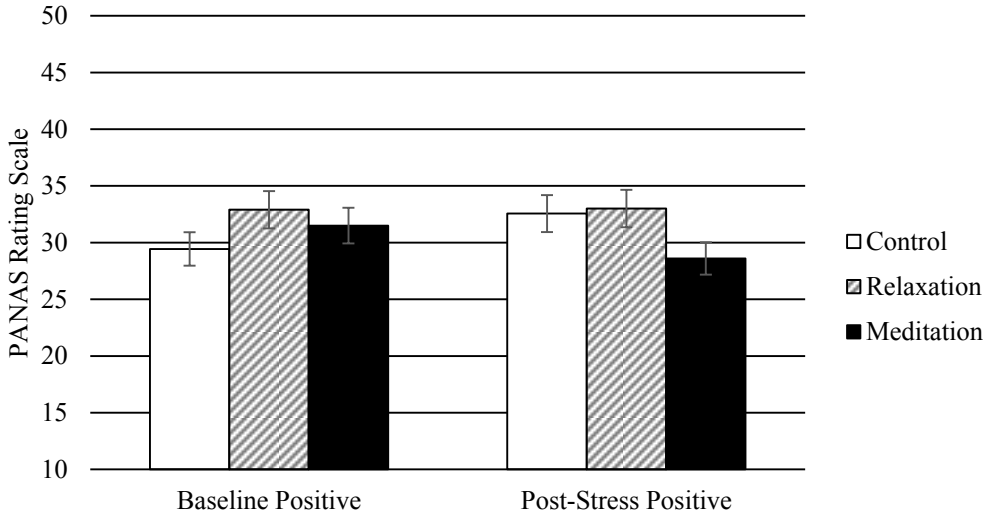
*No significant differences.

Figure 17. Negative Affect Scores, across Groups



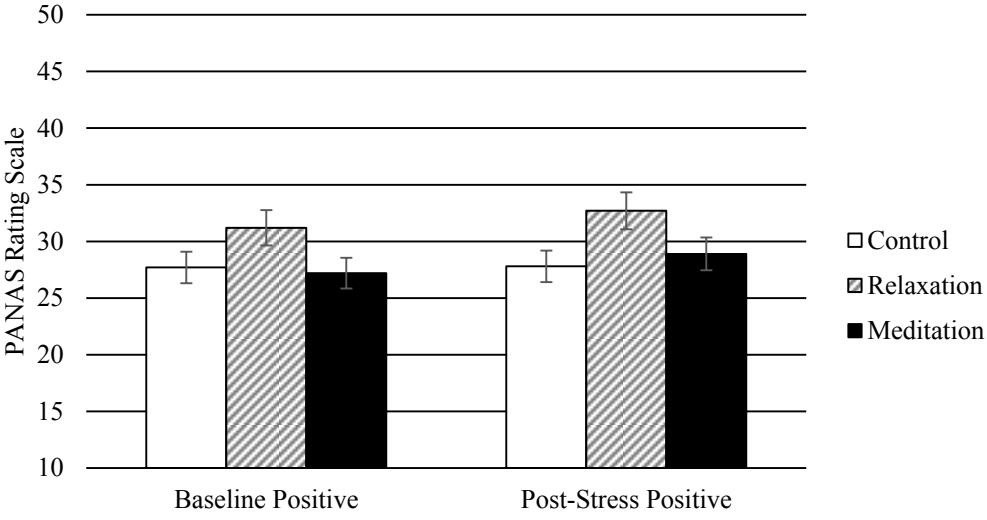
*Indicates significant decrease from baseline, $p = .016$.
**Indicates significant decrease from baseline, $p = .003$.

Figure 18. Pre-Training Positive Affect Scores, by Group



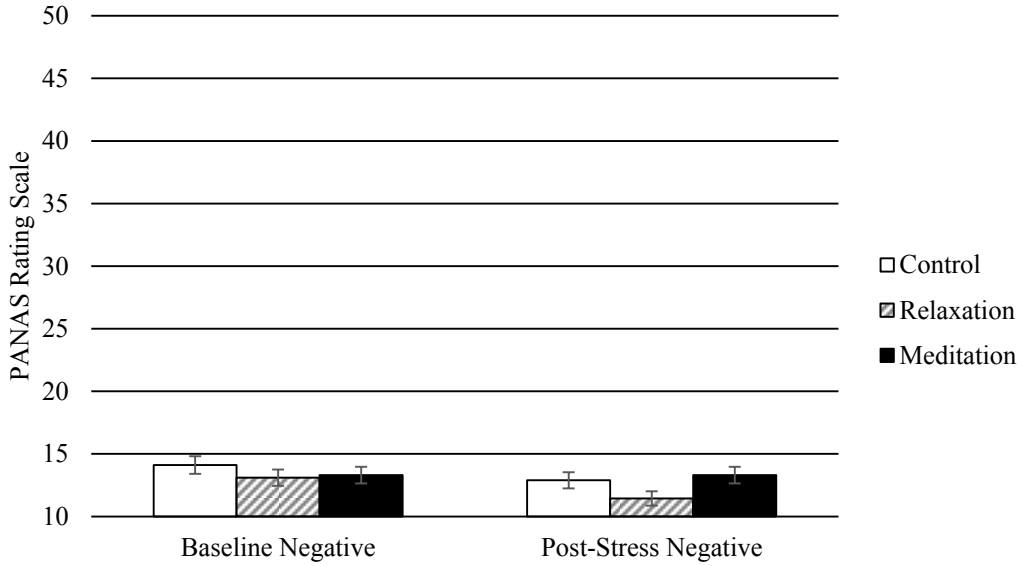
*No significant differences.

Figure 19. Post-Training Positive Affect Scores, by Group



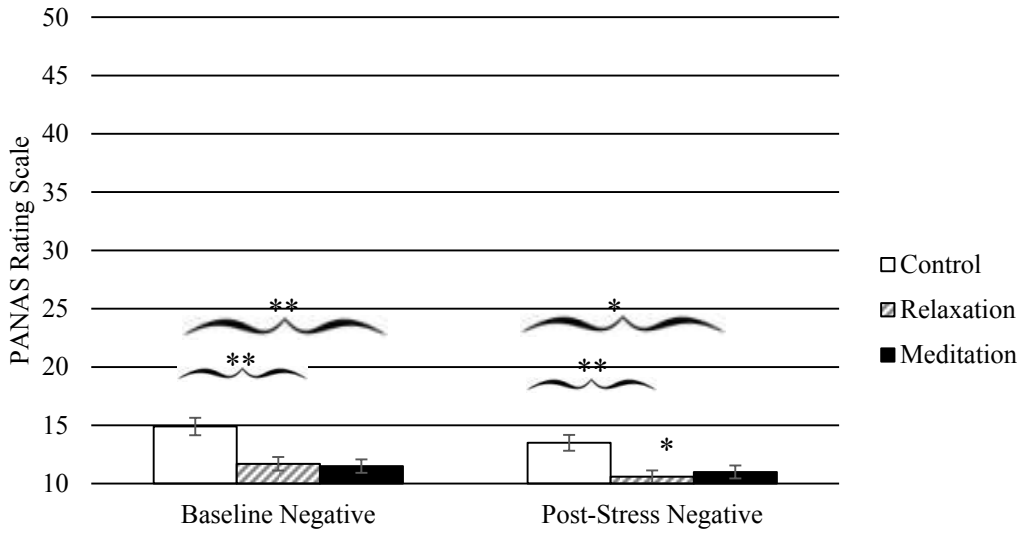
*No significant differences.

Figure 20. Pre-Training Negative Affect Scores, by Group



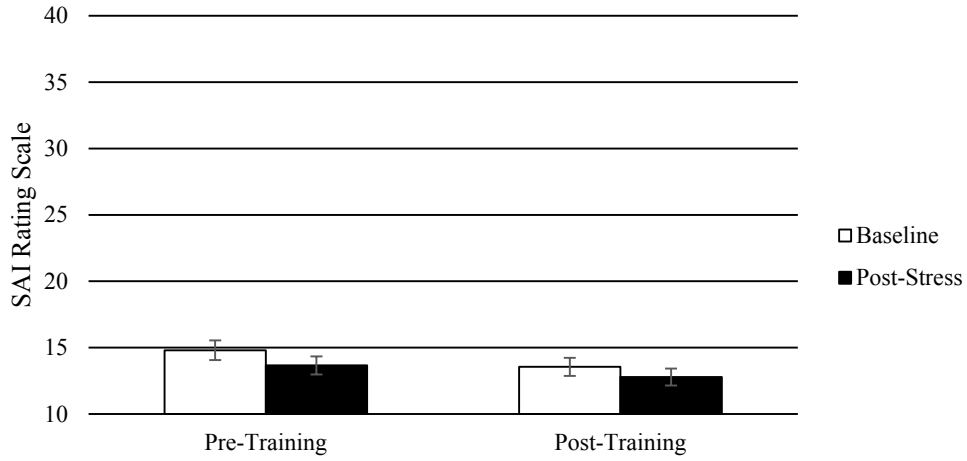
*No significant differences.

Figure 21. Post-Training Negative Affect Scores, by Group



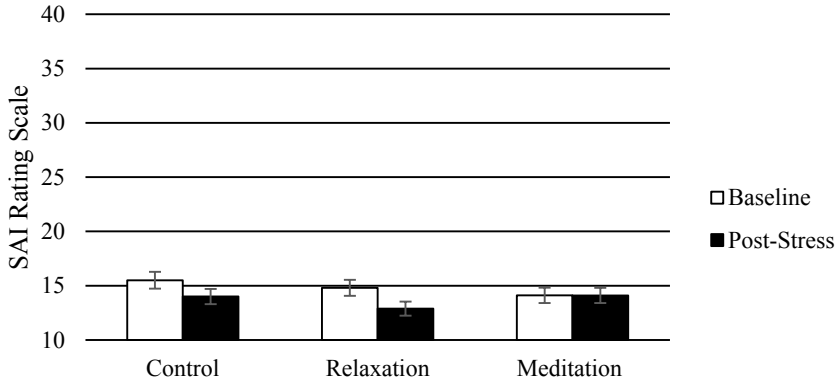
*Indicated signitifanct group differences, p < .03.
**Indicates significant group difference, p < .01.

Figure 22. State Anxiety Scores across Groups

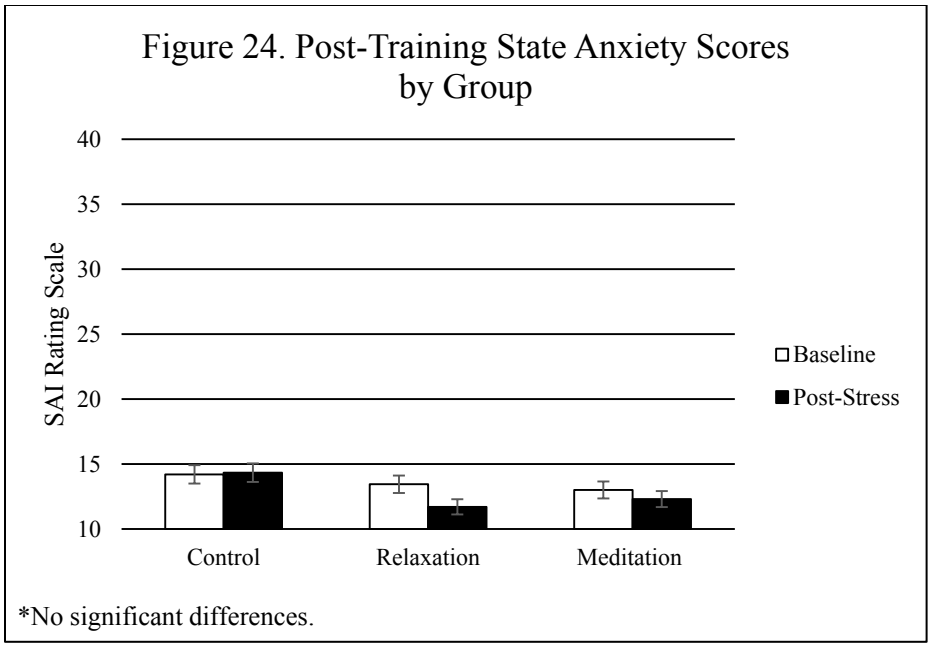


*No significant differences.

Figure 23. Pre-Training State Anxiety Scores by Group



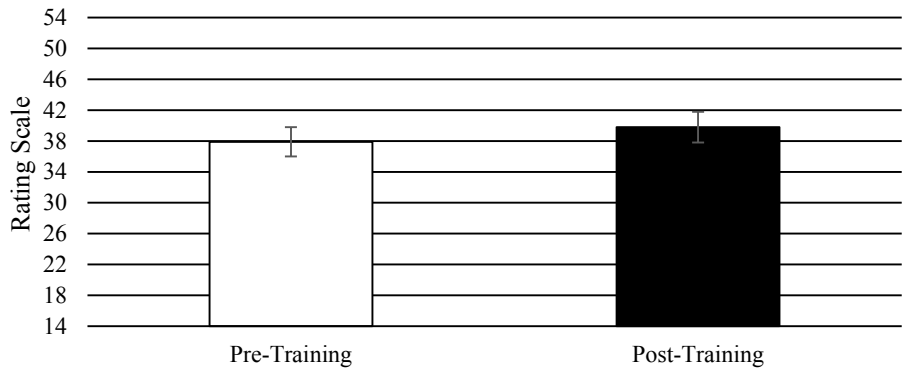
*No significant differences.



5.7 Meditation Measures

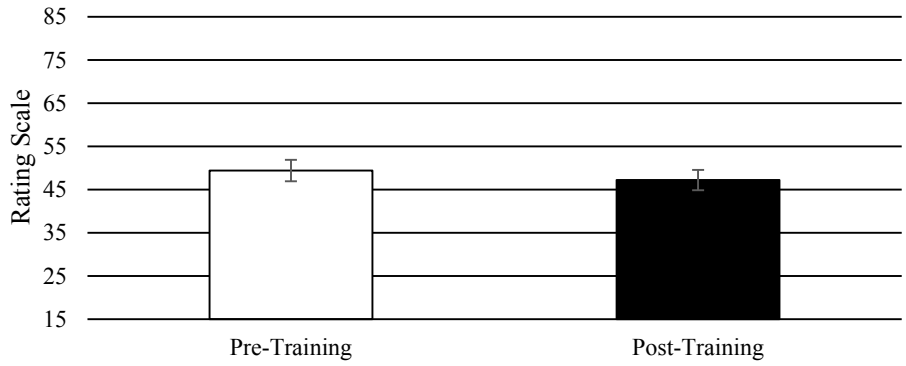
The FMI and MAAS questionnaires assess the presence of mindfulness thinking. These questionnaires were administered to the meditation group after each training session as potential assessments of training efficacy. Although mindfulness meditation training did lower stress-related gating deficits, self-reports of mindfulness did not significantly change over time. Friedman tests revealed no differences between FMI or MAAS scores on the first and last appointments (Figures 25 and 26).

Figure 25. Meditation Group FMI Scores Before and After Training



*No significant differences.

Figure 26. Meditation Group MAAS Scores Before and After Training



*No significant differences.

5.8 Correlational Results

Several correlational findings help elucidate the relationship between the T/C ratios and the psychological measures. For the Fz site, pre-training baseline ratios were correlated with pre-training baseline SAI scores, $r(29) = -.441$, $p = .017$, as well as post-training post-stress SAI scores, $r(29) = -.382$, $p = .041$ (please note that there is some variation from N = 30, as occasionally participants missed a questionnaire). Higher pre-training baseline ratios are associated with lower anxiety ratings both at the beginning of the experiment and at the very end. Pre-training post-stress ratio scores were correlated with pre-training stress scores, $r(24) = -.462$, $p = .023$. Post-stress ratios were higher despite lower stress ratings for the cold-pressor task. Post-training baseline ratios were correlated with post-training post-stress positive mood, $r(30) = .373$, $p = .042$. Higher baseline ratios were associated with lower positive mood ratings. Post-training post-stress ratios correlated with pre-training stress scores, $r(24) = -.427$, $p = .037$, pre-training baseline negative mood, $r(29) = -.368$, $p = .05$, and pre-training post-stress SAI scores, $r(27) = -.405$, $p = .036$. Higher post-stress ratios after training were associated with lower stress scores before training. Greater inhibitory gating impairment at the end of the experiment was associated with the positive traits of lower negative mood and anxiety at the end of the experiment.

For the Pz site, there were no noteworthy relationships between psychological variables and pre-training baseline ratios. Pre-training post-stress ratios correlated with post-training cold-pressor stress scores several days later, $r(28) = .403$, $p = .033$, as well as post-training post-stress SAI anxiety scores several days later, $r(29) = -.439$, $p = .017$. Higher pre-training post-stress ratios were associated with higher cold-pressor stress scores even after training, while higher pre-training post-stress ratios were associated with lower anxiety scores at the very end of the

experiment. Post-training baseline ratios correlated with pre-training FMI scores, $r(10) = .646$, $p = .044$, and post-training FMI scores, $r(10) = .767$, $p = .01$. Please note that the N for the FMI is lower since it only applied to the meditation group. These relationships suggest that higher baseline ratios were associated with lower FMI scores in general. Finally, post-training post-stress ratios were correlated with pre-training post-stress SAI scores, $r(27) = -.411$, $p = .033$. Higher post-stress ratios after training were surprisingly associated with lower post-stress anxiety scores before training had taken place. Section 6.1 contains a subsection with interpretations of correlational findings. Please see Appendix K to view a statistical summary of all correlational relationships.

CHAPTER 6

DISCUSSION

6.1 General Interpretations

The most interesting and strongest results of this study suggest that mindfulness meditation training can alter sensory brain signals, buffer experiences of stress and pain, and decrease negative affect both at baseline and after a stressful event. The following sections will summarize these results in detail, propose explanations for their effects, explore unanticipated findings, and offer directions for future work.

Revisiting Predictions

The overarching goal of this study was to assess if mindfulness meditation could reduce stress-related inhibitory gating impairment in healthy adults. Inhibitory gating impairment was induced using a physical stressor, the cold-pressor task. Participants were randomly assigned to a questionnaire control, progressive muscle relaxation, or mindfulness meditation group and gating was assessed at baseline and post-stress. Participants then attended four training sessions and had baseline and post-stress gating measured once more. The hypotheses were that mindfulness meditation training would reduce the following: 1) Stress-related inhibitory gating impairment; 2) Cold-pressor pain ratings; 3) Cold-pressor stress ratings; 4) Negative affect; and 5) State anxiety.

Summary of Experimental Outcomes

The results support the primary hypothesis that four days of mindfulness meditation will reduce inhibitory gating impairment in healthy people exposed to stress. The mindfulness meditation group exhibited lower post-stress gating ratios than the questionnaire control and progressive muscle relaxation groups. These effects are supported by the fact that baseline gating ratios remained stable over time and were equivalent across groups. Furthermore, stress

induction significantly increased gating ratios both pre-and post-training, but group differences only manifest after training. Thus, this evidence suggests that mindfulness meditation can protect against stress-induced inhibitory gating impairment in healthy adults.

The second and third hypotheses were also supported by experimental findings, as the meditation group rated the cold-pressor task as less physically painful and less mentally stressful than the control group after training. However, the group difference was limited to the control group; mindfulness meditation and progressive muscle relaxation participants did not significantly differ in this regard. This implies that mindfulness meditation training effects extended to how participants experienced the stressor task, and while progressive muscle relaxation may not have been as successful, there was nonetheless a small, non-significant reduction in pain and stress ratings after training.

The fourth and fifth predictions of mindfulness meditation reducing negative affect and anxiety yielded mixed results. Negative affect was significantly lower in the meditation group than the control group after four days of training, but negative affect was also lower in the progressive muscle relaxation group than the control group after training. The meditation and relaxation groups did not differ post-training. Thus, progressive muscle relaxation may have contributed a benefit in reducing negative mood, as mindfulness meditation did. An unanticipated finding was a lack of change in anxiety scores between or within groups due to training or stress. However, correlational results do point to relationships between baseline gating ratios and state anxiety before training, as well as post-stress gating ratios and state anxiety before and after training.

In conclusion, only mindfulness meditation training reduced post-stress inhibitory gating impairment and ratings of pain and stress, but both the meditation group and the progressive

muscle relaxation group exhibited between group reductions in negative affect after training. For a visual summary of experimental outcomes, see Figure 27.

Interpretations of Correlational Findings

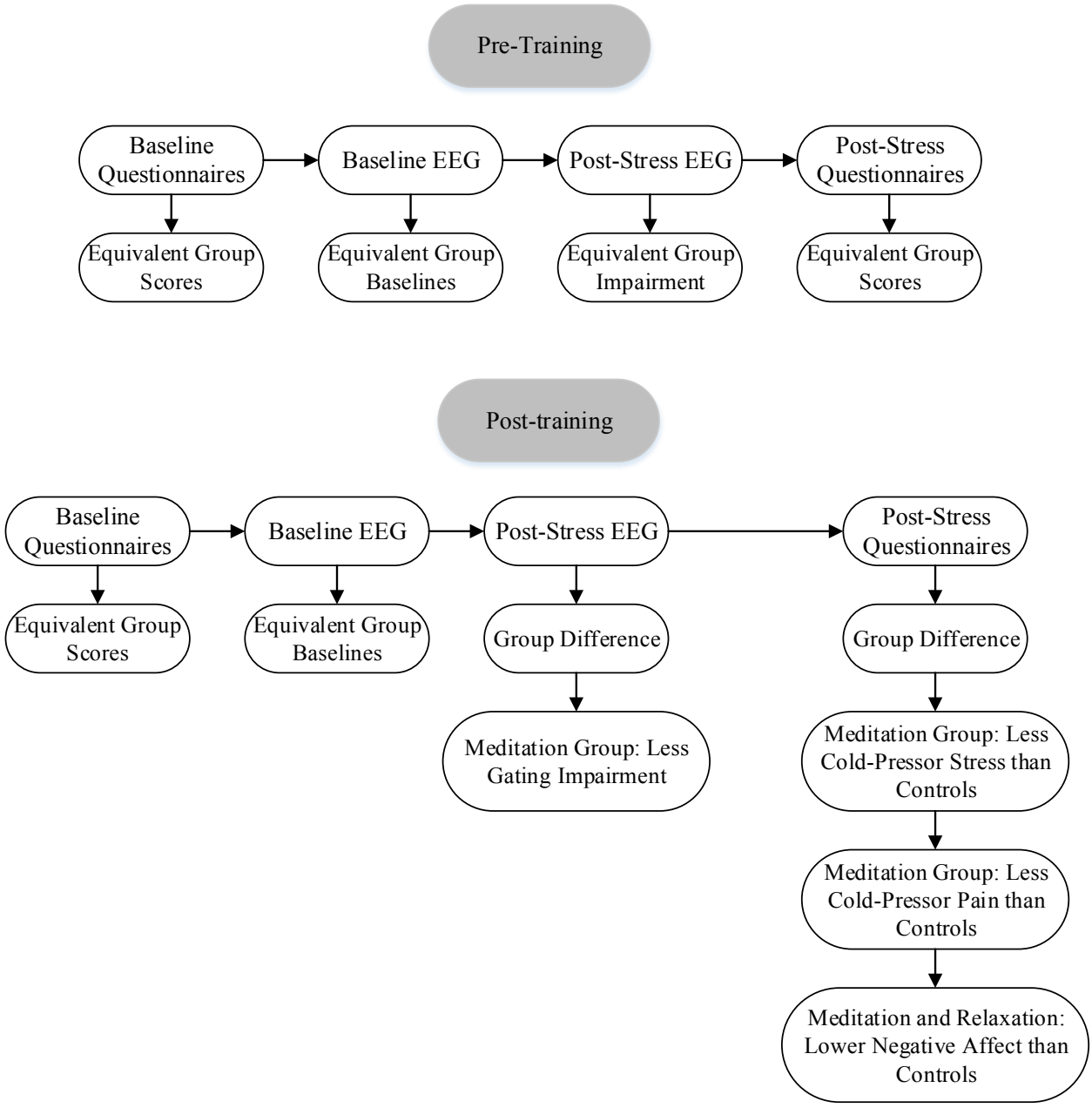
For the Fz site, two unexpected correlational findings were that higher pre-training baseline ratios were associated with lower anxiety ratings before and after training, and post-stress ratios were higher despite lower stress ratings for the cold-pressor task. This may reflect a difference in sensitivity for the physiological and psychological variables. An intuitive correlational finding was that higher baseline ratios were associated with lower positive mood ratings. If inhibitory gating impairment reflects stress, then it follows that lower positive mood would be associated with greater impairment. Furthermore, higher post-stress ratios after training were associated with lower stress scores before training. This relationship is difficult to interpret and suggests that lower ratings of cold-pressor stress at the beginning of the experiment are associated with higher ratios at the end of the experiment. It is likewise difficult to say why greater inhibitory gating impairment at the end of the experiment would be associated with the positive traits of lower negative mood and anxiety at the end of the experiment. These findings may be the result of several days' time between the first and last appointments.

For the Pz site, higher pre-training post-stress ratios were associated with higher cold-pressor stress scores even after training, while higher pre-training post-stress ratios are associated with lower anxiety scores at the very end of the experiment. Higher baseline ratios were also associated with lower FMI scores in general, which offers tentative evidence that mindful thinking and behavior can protect against higher baseline ratios. Higher post-stress ratios after training were surprisingly associated with lower post-stress anxiety scores before training had taken place. Overall, the correlational results point to relationships between baseline T/C ratios

and anxiety before training, as well as positive mood and mindfulness after training. Post-stress T/C ratios were associated with anxiety levels both before and after training. These findings suggest that T/C ratios are not unaffected by mindfulness and mood, although these relationships are not immediately apparent. Counterintuitive findings on higher ratios and lower anxiety should be investigated in future studies. It is possible that heightened anxiety also means heighted alertness, which is beneficial for T/C ratios when the anxiety levels are not indicative of pathology.

The mindfulness meditation group also filled out questionnaires to assess the progress of their training and their internalization of the mindfulness techniques. Outside of correlational results, there were no changes in the mindfulness questionnaire scores over time in the meditation group. However, the FMI and MAAS suffer from limitations as measures of mindfulness. For one, they measure only trait mindfulness (Bergomi et al, 2013). The FMI and MAAS also contain questions that participants regularly misinterpret (Belzer et al., 2011). A longer intervention or a state mindfulness inventory may have yielded clearer results with novice meditators. While statistical analyses did not reveal significant differences for inhibitory gating ratios in regard to the anxiety inventories, correlational results do point to relationships between baseline gating ratios and anxiety before training. The correlational findings suggest that gating ratios may be affected by mindfulness and mood, but these relationships need to be further explored.

Figure 27. Summary of Results



6.2 Synthesis and Critical Analysis

Connections with Previous Work and General Critiques

The results of this study expand and confirm various previous findings on inhibitory gating. Like Johnson and Adler (1993) and Atchley and Cromwell (2013), inhibitory gating in healthy controls was impaired by the cold-pressor task. The impairment pattern reflects others' definition of true gating impairment in that post-stress test responses increased overall while conditioning responses were more stable (Chang et al., 2011). However, unlike persons with schizophrenia (Yee et al., 1998), higher anxiety levels in healthy controls correlated with better gating. This is surprising and should be investigated further, but it is possible that heightened anxiety also means heighted alertness, which is beneficial for gating when the high anxiety levels are not associated with pathology, as in schizophrenia. This study also expanded the known positive effects of mindfulness meditation to inhibitory gating, especially at the Fz site on the frontal lobes, which play a large role in attention and working memory. Moreover, poor functioning in the frontal lobes may increase a person's risk of schizophrenia (Winterer et al., 2001). This ties in to the potential of inhibitory gating function as a predictive tool and biomarker (Freedman et al., 2005; Hutchison et al., 2013). This experiment also offers counter evidence to Rentzch and colleagues' (2008) finding that P50 is unstable over time, as in this case, P50 remained stable in healthy people for four to eight days.

Nonetheless, several aspects and outcomes of this study require scrutiny. For one, the anxiety scales showed no change over time outside of some correlation findings suggesting counterintuitive associations, such as a relationship between higher state anxiety and lower gating. Participants were predicted to experience spikes in state anxiety levels in anticipation of and after the cold-pressor task, especially the second time they completed it after training, but

this did not happen. One reason these predictions may have been off is that it is difficult to accurately assess anxiety without a supplemental physiological measurement. A student project in the BGSU ERP laboratory has since used a heart rate measure in conjunction with inhibitory gating in a stereotype threat experiment. This student (Benson et al., 2014) observed that heart rate increased after an oral arithmetic stress task. This indicates participants' increased anxiety, but ideally this type of measure would be combined with an anxiety inventory for better accuracy. However, it is also possible that the measure is accurate and that anxiety levels within a healthy range do not affect gating.

One anticipated concern was whether the physical stress induced by the cold-pressor would be relatively equal across participants. As a manipulation check for the cold-pressor stress task, participants were told to keep one hand in ice water for as long as they could stand it or two minutes maximum. The goal was to equally stress participants and control for the individual variability in pain threshold. This approach appears to have been successful as the amount of time participants endured the cold-pressor task did not significantly affect other measures. Despite individual variability in the times, participants reached equivalently stressed based upon individual tolerance of cold-temperature pain.

Another potential issue would be that the experimenters who provided training sessions were not experts and had not guided sessions beforehand. All experimenters practiced and timed administration scripts until the desired pacing was mastered. Scripts were taken from professional sources and verified with clinician researchers on mindfulness meditation prior to use. Attention was also paid to the training environment in order to promote a relaxing setting including soft light, cushions, comfortable chairs, and a quiet atmosphere.

Critiques of ERP Findings

The ERP data of this experiment have some limitations. For one, the Cz results were regrettably lost due to amplifier failure. Although the data recorded from Cz could not be used, it is worth noting that Weisser and colleagues (2001) found strong evidence for gating activity in the frontal lobes and the Fz site. Furthermore, the activity at the frontal sites was delayed by approximately 10ms, which aligns with preliminary latency findings of an average P50 peak occurring at approximately 60ms post stimulus. Central site gating (Cz) is thought to show larger P50 peaks than Fz site gating, and the larger Cz peaks facilitate the measurement of gating differences between controls and schizophrenia patients (Bak at el., 2011; Knott et al., 2013). It's possible that frontal gating represents a later gating process given the later latency observed in controls; Weisser and colleagues (2011) also suggest that frontal gating could be indicative of a higher level of auditory processing. Thus, the findings of this study may tap into important higher level auditory processing and the role the frontal lobe contributes to the gating mechanism. Ideally, it would have been extremely interesting to compare and contrast gating findings at all midline electrode sites. But although the Cz site may have larger P50 peaks on average, gating is not at all exclusive to this site. Gating at the Fz and Pz sites has potential to provide a wealth of useful information that could enhance Cz site findings on gating.

A separate issue with this study and others like it is that the parameters of healthy gating are vaguely defined in comparison to impairment parameters. As a product of this, there is not universal agreement on what constitutes normal gating in terms of a T/C ratio cut-off. Chang and colleagues' (2011) recent meta-analysis of 35 gating studies found a very wide range in healthy controls (.16 to .94). Patients with schizophrenia had higher gating scores on average, but individual variability is still high. It's very difficult to say whether a single value should be the standard for impairment. In an absolute sense, gating is impaired at a ratio of 1.0 and above,

which would mean that the test and conditioning responses are equal or the second response is greater. The degree of impairment in ratios under 100 is less clear. In general, healthier gating is a lower ratio and it may be that different people have different baselines. The gating baselines in this study may be considered as too high or within the normal range depending upon the comparison study. Standardization should be attempted, perhaps allowing more flexibility in acceptance of individual variation. This is part of the reason it's worthwhile to study gating in the same population over time. Moreover, there is still uncertainty as to baseline stability and how gating changes based on other internal states like anxiety or affect. It is astonishing how much more there is to learn about the basic mechanism of gating and its fluidity even at healthy levels of function. The brief gating impairment caused by stress may also be, in its own way, adaptive and not truly reflective of the chronic gating problems seen in mental illnesses.

Furthermore, despite long efforts with various programs, software limitations currently prevent the computation of grand average waveforms for this study. Such waveforms are standard in many gating publications and prove a valuable visual of the average P50 waveform. However, the technique through which the data were gathered allowed for only manual inspection and identification of the P50 waveforms relative to stimulus onset. The unique issue with current data collection is that the program cannot automatically identify and average P50 with the same accuracy as manual collection due to the range in which P50 occurs. Although the laboratory currently lacks the necessary tools to create accurate averaged waveforms, detailed information on averaged data has been provided in lieu of this visual (see Tables 1 through 4 in Chapter 5). Many published studies on P50 gating have taken this route (Candenhead et al., 2000; Clementz et al., 1998; Zhang et al., 2012; Zouridakis and Boutros, 1992).

In this study, the baseline correction method was used to determine P50 although another component like N40 or P70 could have supplemented this identification process. Other studies have successfully used baseline correction technique and selection of the largest peak within a range (Boutros et al., 2004; Ghisolfi et al., 2006; Gumenyuk et al., 2013; Inui et al., 2013; Louchart de la Chapelle et al., 2005; Yee & White, 2001). It may be an issue of several ways to arrive to the same result without one innately being superior to the other. Still, in the future, this technique could be tested with these data. Hopefully data collection can be automated as well in the future to greatly increase speed of P50 extraction. A higher number of gating trials (>40) could also have been used in this experiment, but this is not without its own ill effects, namely fatigue. Participants in this study had to undergo gating measurement four times and even just doubling the length of trials would result in two hours of listening to clicks total. This would comprise the data in a different way as participants grew bored, frustrated, or restless with the task. A similar number of trials has been used successfully by a number of studies (Johnson and Adler [32 trials], 1993; Arnfred and Chen [40 trials], 2004; Dawson et al. [48 trials], 2000).

It's also necessary to question why it is desirable only to include "successful" gating trials. Indeed, some studies (for example, Boutros et al., 2004) automatically reject trials without perfect gating and continue with data collection until the desired number of trials are reached. Aside from muscle artifacts and the like, is should be asked if it is accurate to compose an ideal picture of gating by using successful trials and ignoring variously defined unsuccessful trials. Why would gating only work erratically? This study did not assume that controls' gating was not working properly if it was not perfect and all trials were used if possible. This may be one reason participants' gating ratios were higher on average, but it also may represent a clearer picture of real gating function.

Interpretations of Unexpected Results: Anxiety versus Arousal

The findings on anxiety did not fit the predictions of the mindfulness framework. The correlational anxiety results are interesting and may help shed light on this matter. All participants were healthy controls who were screened for trauma and mental illness. Presumably, the anxiety levels reported on the State Anxiety Inventory are not indicative of pathology or chronic health issues. Because these anxiety levels were within a "normal" fluctuating range, this may help explain why higher levels of state anxiety after stress induction were correlated with lower inhibitory gating impairment ratios. There were no significant changes in anxiety scores pre- or post-training, pre- or post-stress, or between groups. Still, high anxiety was correlated with gating improvement. There are several ways to interpret this relationship. The first question to address is whether this correlational finding is indicative or arousal, anxiety, or both.

Arousal and anxiety are connected to stress, and participants were undoubtedly stressed during the cold-pressor task as scores on stress/pain questionnaires and gating impairment verified. It is possible that temporarily heightened physiological arousal in response to the cold-pressor task is the driving force behind heightened post-stress anxiety scores. The non-pathological levels of anxiety in this experiment could also manifest as a negative emotional state or as physical symptoms like sweating or increased heart rate. There was a decrease in negative mood post-stress in the meditation and relaxation groups. The control group did not share this effect, but overall these findings are not in strong agreement with the theory of anxiety as a negative emotional state correlating with better gating. In the absence of heart rate or galvanic skin response data, it is perilous to draw conclusions about arousal as a physical manifestation of stress in this study. The nature of the measure, state anxiety, leads one to

assume that the changes in scores capture transient tension and autonomic nervous system arousal. This is to say that the anxiety scores in this study were elicited in response to stress and do not represent a chronic response.

The relationship between lower gating impairment and higher temporary anxiety should be further researched, as it is unclear from this dataset whether physiological arousal or the negative emotional impact of anxiety (or both) are at the root of this effect. It is difficult to interpret arousal and anxiety findings since arousal can be interpreted positively or negatively and is a precursor to manifestations of anxiety. However, Cromwell and colleagues (2007) have found gating improvement in the rat striatum following acute stress, so this correlational finding is not without precedent. Anxiety, mindfulness, and gating likely have a complex relationship that is difficult to fully capture.

Discrepancy between Mindfulness Inventories and Physiological Data

The current study has shown that stress-induced gating impairment can be reduced by mindfulness meditation. Despite this, participants in the meditation group did not exhibit increased scores on mindfulness meditation inventories (FMI, MAAS) after training. This incongruence could be the result of participants wishing to remain consistent on questionnaires over the course of several days. It is possible that a more lengthy meditation intervention would have shown changes in mindfulness questionnaire scores. Another possibility is that the participants in the meditation group were becoming more mindful but did not know it yet – or they did not know how to quantify it. The current study used Zeidan and colleagues' (2011) experiment as a basis insofar as administering the Freiburg Mindfulness Inventory to assess participants' skills in mindfulness techniques and utilizing four 20-minute sessions of mindfulness meditation training. The authors found a 14% increase in FMI scores after training,

but unexpectedly, the current experiment did not replicate this finding. One discrepancy between the two studies is that Zeidan and colleagues (2011) had a meditation trainer with 20 years of experience. The primary investigator of this study consulted with mindfulness meditation researchers and practitioners in order to validate techniques, but did not have the benefit of administering mindfulness interventions beforehand.

The discrepancy in FMI score changes across these studies could also be due to a difference in sensitivity for the physiological and psychological measures. This ties in to the theory that participants may have had increased mindfulness skills that were reflected in their inhibitory gating ratios, even though they were not able to quantify these changes in a meaningful way on questionnaires. This is an excellent illustration of the benefits of using both psychological inventory and physiological measures together. Although these techniques would ideally produce complementary results, it is always possible that one technique will pick up on something the other may have missed.

It would have been ideal to have FMI and MAAS scores correlate with changes in inhibitory gating, but there is evidence that participants successfully integrated mindfulness meditation techniques nonetheless. For example, negative affect decreased after training in the mindfulness group, which mirrors the findings of Davidson and colleagues' (2003) eight-week mindfulness intervention study. However, the relaxation group also had decreased negative affect. Thus, the reduction in negative affect may reflect calm state rather than specific mindfulness strategies. The best evidence that mindfulness meditation was effective as a manipulation in this study is the fact that only the mediation group exhibited reduced post-stress gating impairment after training. If mindfulness meditation training did not work, then why wouldn't the gating results be the same as relaxation group? This unique result of mindfulness

meditation and reduced gating impairment signifies a deeper relationship. Although this study is able to hint at the positive effects of a brief mindfulness meditation intervention on stress-induced inhibitory gating impairment, the potential therapeutic interpretations of this work require extensive additional testing.

6.3 Proposed Explanation for Mindfulness Effects on Gating

Cortical Gating

Mindfulness meditation training reduced stress-induced gating impairment in healthy controls in this study. Previous research has demonstrated that mindfulness meditation training can reduce anxiety, depression and chronic pain (Kabat-Zinn et al., 1992; Miller et al., 1995; Beauchamp-Turner et al., 1992; Teasdale et al., 1995 and 2000; Zeidan et al., 2009). Neuroanatomical studies have also found a wide variety of apparent meditation-related effects, including increased grey matter concentrations (Hözel et al., 2008 and 2011) and decreased activity in the amygdala paired with increased activity in the inferior and superior parietal lobule, cuneus, precuneus, and middle occipital gyrus (Goldin and Gross, 2010). Although the thalamus is heavily involved in prefrontal cortex inhibitory system (Knight et al., 1999), EEG scalp recordings only allow for assessment of cortical gating at the Fz (central frontal) and Pz (central posterior) electrode sites in this experiment. Speculation will be limited to these areas.

Cortical gating has been observed in various studies. Alexander and colleagues (1976) argued that the prefrontal cortex relays and inhibits neural signals to the primary sensory cortex. Indeed, Knight and colleagues (1989) have shown that impairment in the prefrontal cortex coincides with inhibitory and modulatory dysfunction of sensory inputs to the primary sensory cortex. In a later study, Knight and colleagues (1999) found that humans with dorsolateral

prefrontal damage have impaired inhibitory gating. Persons with schizophrenia may suffer from "non-lesion" brain damage in the dorsolateral prefrontal cortex, which could be a root cause of inhibitory gating impairment (Knight et al., 1999). Persons with prefrontal cortex damage also have trouble maintaining attention and ignoring irrelevant information (Knight et al., 1999). This is exactly the sort of problems one would predict given the theorized function of gating – when gating is disrupted, the ability to attend to relevant information and filter out irrelevant information is impaired. Furthermore, Mears and colleagues (2009) used a rat model to determine that gating in the prefrontal cortex was impaired when tones were paired with stressful stimuli. This may complement the idea of "non-lesion" prefrontal damage in that impaired gating (and perhaps even temporarily impaired gating) can be traced to changes in activity in the prefrontal cortex. This is idea is also supported by Korzyukov and colleagues' (2007) work using electrode grid implants in epilepsy patients to isolate sensory gating generation to the temporal lobes. Neural activity in the frontal lobes contributed to P50 suppression and frontal lobe generators were only active during the test stimulus or inhibitory response (Korzyukov et al., 2007). This has interesting implication for the frontal Fz site as a purer measure of inhibition than Pz, which may be tapping more into the temporal or early auditory gating. The frontal areas are also well-known to contribute to working memory and attention.

GABA Theory of Meditation

What is it about mindfulness meditation that influences inhibitory gating? The effects of meditation on the brain are particularly widespread. Newberg and Iverson (2003) describe meditation as a complicated mental effort that can alter cognition, sensory perception, affect, hormones, and autonomic nervous system activity. Lutz and colleagues (2008) have shown that meditation can increase attention and improve emotional regulation. It could be assumed that the

effects of meditation are better known than the biological mechanism(s) behind them; however, neuroscience research has explored this subject in detail.

Meditation can cause an increase in prefrontal cortex activity that is associated with feelings of calm and improved concentration (Walsh et al., 2006). Building from this, Guglietti and colleagues (2013) investigated the role of a gamma-aminobutyric acid (GABA), a crucial inhibitory neurotransmitter, as a proposed basis for these meditation effects. Guglietti and colleagues (2013) speculated that the increase in prefrontal cortex activity associated with meditation also leads to increased activity in the thalamic reticular nucleus, which subsequently leads to increased GABA distribution. Heightened prefrontal cortex activity specifically increases the production of glutamate, and the ensuing stimulation of the thalamic reticular nucleus increases GABA secretion to the lateral posterior and geniculate nuclei (Armony et al., 2000; Cornwall et al., 2008). It's theorized that GABA selectively inhibits the visual cortex and posterior superior parietal lobule in meditation practitioners (Andrews et al., 1997; Bucci et al., 1999). This could contribute to in an increased ability to concentrate on target stimuli and ignore irrelevant stimuli (Newburg and Iverson, 2003). Essentially, GABAergic inhibitory interneurons are crucial to the modulation of cortical excitability and cortical inhibition, and Guglietti and colleagues (2013) used transcranial magnetic stimulation to assess differences in the cortical inhibition on meditators and non-meditators. The increase in GABA neurotransmitter levels is inferred from the cortical silent period paradigm that indirectly measures $GABA_B$ receptor-mediated cortical activity. Guglietti and colleagues (2013) found that meditators had longer cortical silent periods than controls after a meditation session. This work is excellent evidence of changes in GABA function in meditating individuals. Likewise, the effects of meditation

training on cortical inhibitory gating in this study could be linked to an increase in GABAergic activity.

6.4 Clinical Implications

Impact of Mindfulness Meditation on Gating

This section will cover several specific aspects of mindfulness meditation as well as related research that supports this technique's potential for positive psychological and physiological effects. The results of this experiment are pioneering in that they offer the first evidence of mindfulness meditation as an effective treatment option for inhibitory gating impairment. Studies have shown that nicotine can also prevent gating impairment, but this is complicated by side effects and the fact that nicotine's effect on gating is temporary, lasting approximately 30 minutes. Conversely, mindfulness meditation techniques involve life style changes with potential long-term effects and no risk to health. Mindfulness meditation can actually benefit health, as it has for persons with anxiety, depression, and chronic pain (Kabat-Zinn et al., 1992; Miller et al., 1995; Beauchamp-Turner et al., 1992; Teasdale et al., 1995 and 2000; Zeidan et al., 2009). Even a brief, four day mindfulness intervention can have positive effects on impaired gating. These effects would likely be even more pronounced in long-term meditators or participants in an extended training program.

Uniqueness of Mindfulness Meditation

Persons who practice meditation in general have been shown to have increased activity in the prefrontal cortex as well as increased emotional regulation, cognitive performance, attention, and well-being (Brefczynski-Lewis et al., 2007; Creswell et al., 2007; Davidson et al., 1990 and 1992; Jha et al. 2007; Kabat-Zinn, 2003; Lutz et al., 2008; Tang et al., 2007). However, there are

many different techniques underneath the umbrella of "meditation". How might the effects of these techniques differ? Nothing was known about the effects of meditation on gating until this study, but meditation is otherwise prevalent as a clinical tool. Transcendental meditation, Buddhist meditation, and mindfulness meditation are among the most popular in empirical studies (Dakwar and Levin, 2009). This section will cover the differences in these techniques and their application in clinical settings.

Transcendental meditation was developed by Maharishi Mahesh Yogi in the 1950s and has a religious focus. Trained instructors require payment to teach the technique and come up with specific mantras for their students (Dakwar and Levin, 2009). This practice has been met with criticism, as transcendental meditation training is limited to those willing and able to pay for it. The training is necessary since the focus of transcendental meditation is a personal mantra, which is repeated throughout the meditation session. This separates transcendental meditation from more complex forms of meditation. There is no focus on breath, thoughts, or sensations – only the mantra and repeating it. This may seem like a concentration exercise, but practitioners define the aim of transcendental meditation as purely spiritual and separate from focused attention, (Dakwar and Levin, 2009). Older research has observed a hypometabolic nervous system state in transcendental meditators signified by lower respiratory rates (Wallace et al., 1971). Previous EEG studies on transcendental meditation ascribed increases in theta and alpha waves to the practice, but these findings have since been reclassified as normal baseline activity (Dillbeck and Bronson 1981; Hebert and Tan, 2004). Long-term practitioners of transcendental meditation have decreased cortisol levels but also an exaggerated cortisol response to stress induction (Maclean et al., 1997). It's been theorized that transcendental meditation can increase

GABA levels (Elias et al., 2000), but this has never been empirically shown. The research on the psychiatric benefits of this method are largely speculative and inconclusive (Dakar et al., 2009).

Buddhist meditation has a longer history. To summarize, Siddhartha Guatama founded Buddhism on the principles that life is suffering and that suffering is caused by incorrect thinking, behavior, and understanding. Spiritual respite from suffering could be found within oneself by acknowledging the Four Noble Truths and Eightfold Path, and well as practicing meditation and following an ethical way of life and thought (Dakwar and Levin, 2009). Masters of this philosophy and practice could attain enlightenment. Basic Buddhist meditation is similar to a concentration exercise, e.g., think only of a blue flower. Other techniques incorporate exercises that cultivate attentiveness to detail and/or a passive attitude to all stimuli (Dakwar and Levin, 2009). Buddhist meditation differs from mindfulness in its philosophical emphasis on neutrality as well as metaphysical elements of truth and enlightenment. Studies have shown that Buddhist meditation produces a unique EEG profile with alpha and theta dominance (Echenhofer et al., 1992). In comparison to controls, long-term Buddhist meditators exhibited increased thickness in the prefrontal cortex and right anterior insula, which are associated with attention and sensory perception (Lazar et al., 2005). However, there have also been studies that show no difference between Buddhist meditators and non-meditators in terms of interoceptive awareness, such as assessing one's own heart rate (Khalsa et al., 2008). Buddhist meditation that stresses a focus on compassion has been shown to lessen stress-induced neuroendocrinological responses and cortisol secretion (Pace et al., 2009). Unfortunately Buddhist meditation has had limited success in clinical settings given that persons with mental illnesses may find the philosophical demands of the technique too difficult (Krisanaprakornkit et al., 2006). However, non-random

assignment studies have found an association between Buddhist meditation training and a reduction in substance abuse disorder symptomology (Bowen et al., 2006).

Mindfulness meditation is derived from Buddhist tradition but not affiliated with a religion. It is unique in its non-judgmental focus on the moment. Mindfulness meditation stresses attention to breath and passive acceptance of internal and external sensations (Zeidan et al., 2011). This differentiates it from the progressive muscle relaxation exercise used in this experiment as well, since there is no goal or manipulation, i.e., relaxation. Mindfulness meditation has a more universal appeal in its accessibility, simplicity, and nonreligious nature. Mindfulness meditation is extremely popular in therapeutic settings and has been incorporated into acceptance and commitment therapy, relapse prevention therapy, dialectical-behavioral therapy, mindfulness-based stress reduction, and mindfulness based cognitive therapy (Hayes, 1994; Kabat-Zinn, 1994; Linehand, 1993; Teasdale et al., 2000). Not all of these practices involve meditation in the strictest sense, but rather the adoption of mindfulness perspectives like neutrally acknowledging thoughts, detaching from them, and living moment-to-moment (Dakwar and Levin, 2009). The philosophy behind mindfulness meditation is very powerful in this sense, and perhaps better encapsulated as "mindfulness skills" rather than mindfulness meditation. Mindfulness training has many empirically supported psychological and behavioral benefits. Learning to apply a mindful perspective to aversive thoughts and stimuli is thought to cause desensitization and a reduction in distress (Kabat-Zinn, 1982). Teasdale and colleagues (1995) promote mindfulness training to decrease maladaptive behaviors in depression. Mindfulness-based training also improves coping and tolerance of distress (Kristeller and Hallet, 1999), as well as more adaptive and healthy behavior (Linehan, 1993). Mindfulness meditation is

promising as a treatment candidate for inhibitory gating impairment given the strength of its clinical and empirical effects in complementary medicine.

Impact of Mindfulness Meditation on Stress and Pain

There is a plethora of research on mindfulness meditation's effects on pain-related brain activation. The overarching theme is that mindfulness meditation can affect sensory experience, which is reflected by changes in brain activation. Mindfulness meditation can reduce activation in the primary somatosensory cortex, which lessens responses to pain (Zeidan et al., 2010). After only four days of mindfulness meditation training (Zeidan et al., 2011), participants report relief in terms of lower pain intensity (40% less) and pain unpleasantness (57% less) after a task in which one hand was exposed to heat. Lower pain unpleasantness ratings correlated with deactivation in the thalamus. The authors speculate that this may indicate changes in limbic gating and therefore affect how afferent sensory information is processed. There are many potential brain mechanisms involved in the modulation of pain that may also be affected by mindfulness meditation practice.

Anxiety levels can also be affected by mindfulness meditation training. Zeidan and colleagues (2009) used electrical stimulation to induce pain before and after three 20 minute sessions of mindfulness meditation training. The pain response was measured subjectively as ratings of pain intensity and objectively as pain sensitivity or pain threshold. Both pain intensity ratings and pain sensitivity decreased after meditation training. However, the authors noted a decrease in anxiety ratings (with the State Anxiety Inventory or SAI) as well, which may have added to this analgesic effect. Although participants only had three sessions of training, they still reported more mindful thinking (with the Freiberg Mindfulness Inventory or FMI) at the end of the experiment.

Liu and colleagues (2012) found that participants who had undergone a single session of mindfulness meditation training also had higher pain tolerance and reported decreased distress after completing the cold-pressor task. A distraction task also increased tolerance, but not ratings of distress. This suggests that mindfulness meditation may affect both the physical and mental experience of stress and pain even when intervention training is relatively brief. Indeed, participants score higher on mindfulness traits after only a few days of training and become more sensitive to internally generated stimuli, while responses to external stimuli are attenuated (Teper et al., 2013).

Long-term meditation interventions have also been explored as therapeutic options using different neuroimaging techniques. Davidson and colleagues (2003) held an eight week long training program. They measured brain activity before and after training using electroencephalography (EEG) and found that after training, left-sided anterior activation increased. Higher activity in this region has been associated with positive affect (Davidson, 1992; Davidson et al., 1990). Negative affect was self-reported before and after training and was lower in the meditation group. The eight-week intervention also had positive effects on the immune system; antibodies to a vaccine were at higher levels in the meditation group in comparison to a control group.

Overall, these scientific findings support the idea of meditation interventions having beneficial emotional and psychological effects. These positive changes are reflected both by self-report scales and anatomically in gray matter concentrations and patterns of brain activation. Meditation has been found to increase positive affect, reduce anxiety, and have enduring effects in decreasing chronic pain (Kabat-Zinn et al., 1992; Miller et al., 1995; Beauchamp-Turner et al., 1992; Zeidan et al., 2009). Mindfulness meditation-based cognitive therapy can even help

prevent relapses in patients with depression (Teasdale et al., 1995 and 2000). Inhibitory gating is pre-attentional, but practiced changes in cognitive and behavioral responses may modulate pain- and stress-related disruptions. For these reasons, the effect of mindfulness meditation training on inhibitory gating impairment should be investigated for its potential to reduce dysfunction and promote well-being.

Future Directions and Relevance

Perhaps the most important area to explore further is what these findings mean for persons with mental illnesses in which inhibitory gating is impaired. This study opens doors for further research on the potential effects of mindfulness meditation training and interventions in persons with mental illnesses. Mindfulness meditation cannot replace the efficacy of drugs in most instances, but it can supplement treatment and potentially help people suffering from conditions like chronic stress or pain (Zeidan et al., 2009). This is only a stepping stone, but it is a promising one in that it also helps elucidate the potential of other cognitive and behavioral interventions for inhibitory gating as a state-dependent, pre-attentional brain mechanism that is susceptible to impairment even in healthy adults.

The design of this study opens doors for additional experiments that with the goal of modeling gating impairment in healthy adults. It supports various research that has found stress to impair gating temporarily in healthy controls (Johnson and Adler, 1993; White and Yee, 1997). While previous research focused on what stressors could impair gating, none so far have explored how to help restore gating back to normal after it is impaired. The body of literature on this topic suggests that physical, mental, and social stress can impair gating function (Johnson and Adler, 1993; White and Yee, 1997; Yee and White, 2001), but none have compared these

different stressors in the same population. Would a physical stressor (e.g., the cold-pressor) cause greater gating impairment than a cognitive stressor (e.g., mental arithmetic), as Johnson and Adler (1993) hypothesized? Or would the cognitive stressor be on par with the physical stressor in terms of gating impairment induction, as argued by White and Yee (1997)? The statistical reports of these two studies do not make it possible to determine which stressor had a greater impact in terms of effect size or ratio averages. Future work should take this comparative approach so that different stressors and their potential for unique effects can be better understood. Saliva cortisol levels could be taken concurrently as a manipulation check of individual differences in response to a particular stressor.

This study supports previous work that has found beneficial effects of meditation in short interventions and mindfulness meditation in particular (Beauchamp-Turner et al., 1992; Kabat-Zinn et al., 1992; Miller et al., 1995; Teasdale et al., 1995 and 2000; Zeidan et al., 2009). Intervention length comparison in relation to gating effects would be a logical next step in this research. How long does it take for mindfulness meditation to reach its maximum effect? Moreover, is this intervention period different for healthy people versus people with mental illnesses in which gating is chronically impaired? Future clinical trials should aim to determine the feasibility and effectiveness of mindfulness meditation as a technique to reduce gating impairment in different mental illnesses. The findings of this study imply that four short sessions are enough to see results, but it remains to be seen if gating improvement will be seen in patient populations. This would help inform the fundamental understanding of gating function. Another question is whether longer-term mindfulness meditation practice can improve healthy baseline gating. If so, this results would further strengthen the general argument of Kabat-Zinn (1994; 2000) that mindfulness meditation can have wide benefits on well-being that extend to

psychological and physical health. In the future, with more studies building off of this foundation, treatment for inhibitory gating impairment may be added to the list of mindfulness meditation benefits.

6.5 General Conclusions

This study lays the groundwork for future experiments on mindfulness meditation and its effects on early information processes and inhibitory gating in particular. It is essential to combine neurophysiological methods with clinical application to improve treatment options for gating impairment. Only four days of mindfulness meditation training were needed to reduce stress-induced impairment in this mechanism, and there is great potential to reduce impairment further by utilizing mindfulness techniques in clinical settings once more testing has been done. Additional research should extend this work and apply mindfulness meditation as an intervention and/or prevention tool, and rigorously test its use to protect the inhibitory gating mechanism against impairment due to stress or dysfunction.

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APPENDIX A. GENERAL QUESTIONNAIRE

1. What is your age? ___
2. What is your gender? _____
3. What is your ethnicity? _____

APPENDIX B. POSITIVE AND NEGATIVE AFFECT SCALE (PANAS)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent *you feel this way right now, at the present moment*. Use the following scale to record your answers.

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely

___interested
___distressed
___excited
___upset
___strong
___guilty
___scared
___hostile
___enthusiastic
___proud

___irritable
___alert
___ashamed
___inspired
___nervous
___determined
___attentive
___jittery
___active
___afraid

APPENDIX C. STATE ANXIETY INVENTORY (SAI)

A number of statements which people have used to describe themselves are given below. Read each statement and then select the appropriate one to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

---

1. I feel calm
   - ○ Not at all
   - ○ Somewhat
   - ○ Moderately So
   - ○ Very Much So

---

2. I feel secure
   - ○ Not at all
   - ○ Somewhat
   - ○ Moderately So
   - ○ Very Much So

---

3. I am tense
   - ○ Not at all
   - ○ Somewhat
   - ○ Moderately So
   - ○ Very Much So

---

4. I am regretful
   - ○ Not at all
   - ○ Somewhat
   - ○ Moderately So
   - ○ Very Much So

---

5. I feel at ease
   - ○ Not at all
   - ○ Somewhat
   - ○ Moderately So

- Very Much So

---

6. I feel upset

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

7. I am presently worrying over possible misfortunes

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

8. I feel rested

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

9. I feel anxious

Not at all

- Somewhat
- Moderately So
- Very Much So

---

10. I feel comfortable

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

11. I feel self-confident

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

12. I feel nervous

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

13. I am jittery

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

14. I feel "high strung"

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

15. I am relaxed

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

16. I feel content

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

17. I am worried

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

18. I feel overexcited and rattled

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

19. I feel joyful

- Not at all
- Somewhat
- Moderately So
- Very Much So

---

20. I feel pleasant

- Not at all
- Somewhat
- Moderately So
- Very Much So

APPENDIX D. MCGILL PAIN SCALE

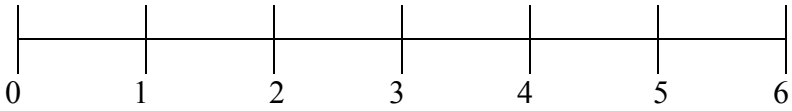
READ THESE INSTRUCTIONS TO PARTICIPANT: *For this task, you will insert your left hand up to the wrist in ice water. Your instructions are to keep your hand in the water for 2 minutes, at which point I'll ask you to take your hand out. However, you can remove your hand at any time if the task gets too uncomfortable. Every 20 seconds, I will ask you to rate the amount of pain you feel from 0 to 5, with 0 being no pain and 5 being excruciating pain. Please take a moment to become familiar with the scale in front of you. When I ask you to rate your pain, please answer out loud using a number. Are you ready to begin?*

1. 20 Seconds – *Please rate your pain from 0-5.* Verbal Rating: ____
2. 40 Seconds – *Please rate your pain from 0-5.* Verbal Rating: ____
3. 60 Seconds – *Please rate your pain from 0-5.* Verbal Rating: ____
4. 80 Seconds – *Please rate your pain from 0-5.* Verbal Rating: ____
5. 100 Seconds – *Please rate your pain from 0-5.* Verbal Rating: ____
6. 120 Seconds – *Please remove your hand from the water and rate your pain from 0-5.* Verbal Rating: ____

APPENDIX E. STRESS ASSESSMENT FORM

INSTRUCTIONS: Please circle a number on the scale to indicate your answer.

1. How physically uncomfortable was the cold-pressor task?



Not at All Extremely

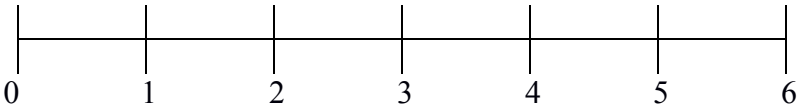
2. How mentally stressful was the cold-pressor task?

Not at All Extremely

3. How mentally stressed were you feeling before you came to the experiment today?

Not at All Extremely

4. How physically uncomfortable were you feeling before you came to the experiment today?



Not at All Extremely

APPENDIX F. MINDFUL ATTENTION AWARENESS SCALE (MAAS)

Below is a collection of statements about your everyday experience. Using the 1-6 scale below, please indicate how frequently or infrequently you currently have each experience. Please answer according to what *really reflects* your experience rather than what you think your experience should be. Please treat each item separately from every other item.

1	2	3	4	5	6
Almost Always	Very Frequently	Somewhat Frequently	Somewhat Infrequently	Very Infrequently	Almost Never

I could be experiencing some emotion and not be conscious of it until some time later.	1	2	3	4	5	6
I break or spill things because of carelessness, not paying attention, or thinking of something else.	1	2	3	4	5	6
I find it difficult to stay focused on what's happening in the present.	1	2	3	4	5	6
I tend to walk quickly to get where I'm going without paying attention to what I experience along the way.	1	2	3	4	5	6
I tend not to notice feelings of physical tension or discomfort until they really grab my attention.	1	2	3	4	5	6
I forget a person's name almost as soon as I've been told it for the first time.	1	2	3	4	5	6

It seems I am “running on automatic,” without much awareness of what I’m doing.	1	2	3	4	5	6
I rush through activities without being really attentive to them.	1	2	3	4	5	6
I get so focused on the goal I want to achieve that I lose touch with what I’m doing right now to get there.	1	2	3	4	5	6
I do jobs or tasks automatically, without being aware of what I'm doing.	1	2	3	4	5	6
I find myself listening to someone with one ear, doing something else at the same time.	1	2	3	4	5	6
I drive places on ‘automatic pilot’ and then wonder why I went there.	1	2	3	4	5	6
I find myself preoccupied with the future or the past.	1	2	3	4	5	6
I find myself doing things without paying attention.	1	2	3	4	5	6
I snack without being aware that I’m eating.	1	2	3	4	5	6

APPENDIX G. FREIBURG MINDFULNESS INVENOTRY (FMI)

The purpose of this inventory is to characterize your experience of mindfulness. Please use the last ___ days as the time-frame to consider each item. Provide an answer for every statement as best you can. Please answer as honestly and spontaneously as possible. There are neither 'right' nor 'wrong' answers, nor 'good' or 'bad' responses. What is important to us is your own personal experience.

---

1. I am open to the experience of the present moment.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

---

2. I sense my body, whether eating, cooking, cleaning or talking.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

---

3. When I notice an absence of mind, I gently return to the experience of the here and now.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

---

4. I am able to appreciate myself.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

---

5. I pay attention to what's behind my actions.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often

- Almost Always

---

6. I see my mistakes and difficulties without judging them.
   - Rarely
   - Occasionally
   - Fairly Often
   - Almost Always

---

7. I feel connected to my experience in the here-and-now.
   - Rarely
   - Occasionally
   - Fairly Often
   - Almost Always

---

8. I accept unpleasant experiences.
   - Rarely
   - Occasionally
   - Fairly Often
   - Almost Always

---

9. I am friendly to myself when things go wrong.
   - Rarely
   - Occasionally
   - Fairly Often
   - Almost Always

---

10. I watch my feelings without getting lost in them.
    - Rarely
    - Occasionally
    - Fairly Often
    - Almost Always

---

11. In difficult situations, I can pause without immediately reacting.
    - Rarely
    - Occasionally
    - Fairly Often
    - Almost Always

12. I experience moments of inner peace and ease, even when things get hectic and stressful.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

13. I am impatient with myself and with others.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

14. I am able to smile when I notice how I sometimes make life difficult.

- ○ Rarely
- ○ Occasionally
- ○ Fairly Often
- ○ Almost Always

APPENDIX H. MINDFUL MEDITATION EXERCISE SCRIPT

Adapted from Segal et al., 2002

(Experimenter Notes: Read slowly and clearly. Inform participant that they don't need to respond to you throughout the training session.)

Now we're going to do a meditation exercise for a few minutes. First, settle into a comfortable sitting position, with your back straight against the back of the chair, your legs uncrossed, your feet flat on the floor <pause> and your hands in your lap. Now close your eyes.

As you begin this exercise, acknowledge that this practice is sometimes pleasant and sometimes not and requires discipline and motivation. Prepare your mind for this exercise by recalling its purpose, to really pay attention to the present moment, finding a sense of calm in everyday experience.

PAUSE FOR 1 or 2 BREATHS

Ask yourself, "What is my experience right now? What am I thinking about? What am I feeling emotionally? What sensations are present in my body?" Just observe your experience, whatever it is.

PAUSE FOR 2 or 3 BREATHS

Now bring your attention to the changing physical sensations in your lower abdomen as the breath moves in and out of your body. To help you pay attention to your breathing, place your hand on your lower abdomen, and become aware of the changing sensations where your hand makes contact with your belly. When you've tuned in to these sensations, you can remove your hand if you like, and continue to observe the sensations in your belly.

PAUSE FOR 2 BREATHS

As you attend to your breathing, allow yourself to feel that the breath represents a source of calm, focus and energy within you. It may help to remind yourself of the way the breath sustains your body every moment of every day.

PAUSE FOR 2 BREATHS

Continuing to follow the breath, focus your awareness on the sensations of slight stretching as the belly rises with each in-breath, and of gentle deflation as it falls with each outbreath, perhaps also noticing the slight pause at the end of the in-breath, <pause> and the slight pause between the end of one outbreath <pause> and the beginning of the next in-breath. <pause> There is no need to control the breathing in any way—allow it to be just as it is. As best you can, also bring this sense of allowing to the rest of your experience, even to those aspects of your experience that may be unpleasant.

PAUSE FOR 5 BREATHS

Sooner or later your mind will wander away from the focus on the breath to thoughts, feelings, or daydreams. This is perfectly OK, it's what minds do. When you notice that your awareness is no longer on the breath, acknowledge briefly where the mind has been, perhaps using a quiet label, like thinking, or feeling, or itchy. Then, gently bring your awareness back to the breath.

PAUSE FOR 5 BREATHS

You may find that your mind often wanders. If a certain thought or feeling persists, don't push it away. Just keep acknowledging, and gently returning to the breath. Remember there is no need to be frustrated. This wandering and returning is part of the technique. As your attention leaves the breath and returns, see if you can settle into a sense of calm and clarity, all around you, moving through you, with every breath in, and every breath out.

PAUSE FOR 5 BREATHS

Simply continue to follow the breath, noting what is on your mind when it wanders, and then gently bringing it back to the breath. I'll let you know when to open your eyes.

PAUSE FOR 5 BREATHS

This concludes the meditation exercise. Thank you.

APPENDIX I. PROGRESSIVE MUSCLE RELAXATION SCRIPT

Adapted from Inner Health Studio website, 2012

(Experimenter Notes: Read slowly and clearly. Inform participant that they don't need to respond to you throughout the training session.)

Now we're going to do a progressive muscle relaxation exercise for a few minutes. Begin by finding a comfortable sitting position. You can change positions any time during this progressive muscle relaxation exercises to make yourself more comfortable.

We're going to work on relaxing the muscles of your body. Start with the large muscles of your legs. Tighten all the muscles of your legs. <pause> Tense the muscles further <pause> and hold onto this tension. <pause> Continue to hold this tension. <pause> Feel the muscles wanting to give up this tension. <pause> Hold it for a few moments more <pause> and now relax. Let all the tension go. Feel the muscles in your legs relax. <pause> Notice how relaxed the muscles feel now. <pause> Feel the difference between tension and relaxation.

PAUSE FOR 1 or 2 BREATHS

Now focus on the muscles in your arms. Tighten your shoulders, upper arms, lower arms, and hands. Squeeze your hands into tight fists. Tense the muscles in your arms and hands as tightly as you can. <pause> Hold the tension in your arms, shoulders, and hands. <pause> Feel the tension in these muscles. <pause> Hold it for a few moments more <pause> and now release. Let the muscles of your shoulders, arms, and hands relax. Feel the relaxation as your shoulders lower into a comfortable position and your hands relax at your sides. <pause> Allow the muscles in your arms to relax completely.

PAUSE FOR 1 or 2 BREATHS

Tighten the muscles of your back now. <pause> Feel your back tightening, pulling your shoulders back and tensing the muscles along your spine. <pause> Arch your back slightly as you tighten these muscles. Tense the muscles further <pause> and hold onto this tension. <pause> Continue to hold this tension. <pause> Feel the muscles wanting to give up this tension. <pause> Hold it for a few moments more <pause> and now relax. Let all the tension go. Feel your back comfortably relaxing into a good and healthy posture.

PAUSE FOR 1 or 2 BREATHS

Turn your attention now to the muscles of your chest and stomach. Tighten and tense these muscles. <pause> Tense the muscles further <pause> and hold onto this tension. <pause> Continue to hold this tension. <pause> Feel the muscles wanting to give up this tension. <pause> Hold it for a few moments more <pause> and now relax. Let all the tension go. Relax the muscles of your chest and stomach.

PAUSE FOR 1 or 2 BREATHS

Finally, tighten the muscles of your face. <pause> Scrunch your eyes shut tightly, wrinkle your nose, and tighten your cheeks and chin. <pause> Tense the muscles further <pause> and hold onto this tension. <pause> Continue to hold this tension. <pause> Feel the muscles wanting to give up this tension. <pause> Hold it for a few moments more <pause> and now relax. Let all the tension go. Feel how relaxed your face is.

PAUSE FOR 1 or 2 BREATHS

Notice all of the muscles in your body. Notice how relaxed your muscles feel. Allow any last bits of tension to drain away. <pause> Notice your calm breathing… your relaxed muscles…Enjoy the relaxation for a few moments.

PAUSE FOR 1 or 2 BREATHS

When you are ready to return to your usual level of alertness and awareness, slowly begin to re-awaken your body. <pause> Stretch, if you like.

PAUSE FOR 1 or 2 BREATHS

This concludes the muscle relaxation exercise. Thank you.

APPENDIX J. JUNG TYPOLOGY TEST

Adapted from HumanMetrics website, 2012

Participant Instructions: Please answer Yes or No to the following statements. When responding to the statements, please choose the one you agree with most. If you are not sure how to answer, make your choice based on your most typical response or feeling in the given situation.

1. You are almost never late for your appointments
   ○ YES ○ NO
2. You like to be engaged in an active and fast-paced job
   ○ YES ○ NO
3. You enjoy having a wide circle of acquaintances
   ○ YES ○ NO
4. You feel involved when watching TV soaps
   ○ YES ○ NO
5. You are usually the first to react to a sudden event, such as the telephone ringing or unexpected question
   ○ YES ○ NO
6. You are more interested in a general idea than in the details of its realization
   ○ YES ○ NO
7. You tend to be unbiased even if this might endanger your good relations with people
   ○ YES ○ NO
8. Strict observance of the established rules is likely to prevent a good outcome
   ○ YES ○ NO
9. It's difficult to get you excited
   ○ YES ○ NO
10. It is in your nature to assume responsibility
    ○ YES ○ NO
11. You often think about humankind and its destiny
    ○ YES ○ NO
12. You believe the best decision is one that can be easily changed
    ○ YES ○ NO
13. Objective criticism is always useful in any activity
    ○ YES ○ NO
14. You prefer to act immediately rather than speculate about various options
    ○ YES ○ NO
15. You trust reason rather than feelings
    ○ YES ○ NO

16. You are inclined to rely more on improvisation than on careful planning
    ○ YES ○ NO
17. You spend your leisure time actively socializing with a group of people, attending parties, shopping, etc.
    ○ YES ○ NO
18. You usually plan your actions in advance
    ○ YES ○ NO
19. Your actions are frequently influenced by emotions
    ○ YES ○ NO
20. You are a person somewhat reserved and distant in communication
    ○ YES ○ NO
21. You know how to put every minute of your time to good purpose
    ○ YES ○ NO
22. You readily help people while asking nothing in return
    ○ YES ○ NO
23. You often contemplate the complexity of life
    ○ YES ○ NO
24. After prolonged socializing you feel you need to get away and be alone
    ○ YES ○ NO
25. You often do jobs in a hurry
    ○ YES ○ NO
26. You easily see the general principle behind specific occurrences
    ○ YES ○ NO
27. You frequently and easily express your feelings and emotions
    ○ YES ○ NO
28. You find it difficult to speak loudly
    ○ YES ○ NO
29. You get bored if you have to read theoretical books
    ○ YES ○ NO
30. You tend to sympathize with other people
    ○ YES ○ NO
31. You value justice higher than mercy
    ○ YES ○ NO
32. You rapidly get involved in the social life of a new workplace
    ○ YES ○ NO
33. The more people with whom you speak, the better you feel
    ○ YES ○ NO

34. You tend to rely on your experience rather than on theoretical alternatives
    ○ YES ○ NO
35. You like to keep a check on how things are progressing
    ○ YES ○ NO
36. You easily empathize with the concerns of other people
    ○ YES ○ NO
37. You often prefer to read a book than go to a party
    ○ YES ○ NO
38. You enjoy being at the center of events in which other people are directly involved
    ○ YES ○ NO
39. You are more inclined to experiment than to follow familiar approaches
    ○ YES ○ NO
40. You avoid being bound by obligations
    ○ YES ○ NO
41. You are strongly touched by stories about people's troubles
    ○ YES ○ NO
42. Deadlines seem to you to be of relative, rather than absolute, importance
    ○ YES ○ NO
43. You prefer to isolate yourself from outside noises
    ○ YES ○ NO
44. It's essential for you to try things with your own hands
    ○ YES ○ NO
45. You think that almost everything can be analyzed
    ○ YES ○ NO
46. You do your best to complete a task on time
    ○ YES ○ NO
47. You take pleasure in putting things in order
    ○ YES ○ NO
48. You feel at ease in a crowd
    ○ YES ○ NO
49. You have good control over your desires and temptations
    ○ YES ○ NO
50. You easily understand new theoretical principles
    ○ YES ○ NO
51. The process of searching for a solution is more important to you than the solution itself
    ○ YES ○ NO

52. You usually place yourself nearer to the side than in the center of a room
    ○ YES ○ NO
53. When solving a problem you would rather follow a familiar approach than seek a new one
    ○ YES ○ NO
54. You try to stand firmly by your principles
    ○ YES ○ NO
55. A thirst for adventure is close to your heart
    ○ YES ○ NO
56. You prefer meeting in small groups over interaction with lots of people
    ○ YES ○ NO
57. When considering a situation you pay more attention to the current situation and less to a possible sequence of events
    ○ YES ○ NO
58. You consider the scientific approach to be the best
    ○ YES ○ NO
59. You find it difficult to talk about your feelings
    ○ YES ○ NO
60. You often spend time thinking of how things could be improved
    ○ YES ○ NO
61. Your decisions are based more on the feelings of a moment than on the careful planning
    ○ YES ○ NO
62. You prefer to spend your leisure time alone or relaxing in a tranquil atmosphere
    ○ YES ○ NO
63. You feel more comfortable sticking to conventional ways
    ○ YES ○ NO
64. You are easily affected by strong emotions
    ○ YES ○ NO
65. You are always looking for opportunities
    ○ YES ○ NO
66. Your desk, workbench, etc. is usually neat and orderly
    ○ YES ○ NO
67. As a rule, current preoccupations worry you more than your future plans
    ○ YES ○ NO
68. You get pleasure from solitary walks
    ○ YES ○ NO

69. It is easy for you to communicate in social situations

YES NO

70. You are consistent in your habits

YES NO

71. You willingly involve yourself in matters which engage your sympathies

YES NO

72. You easily perceive various ways in which events could develop

YES NO

APPENDIX K. CORRELATION TABLES

Table 5. Fz Site T/C Ratio, S1 (C), and S2 (T) Correlations

		Fz Pre-Training Baseline Ratio	Fz Pre-Training Post-Stress Ratio	Fz Post-Training Baseline Ratio	Fz Post-Training Post-Stress Ratio	Fz Pre-Training Baseline S1	Fz Pre-Training Baseline S2	Fz Pre-Training Post-Stress S1	Fz Pre-Training Post-Stress S2	Fz Post-Training Baseline S1	Fz Post-Training Baseline S2	Fz Post-Training Post-Stress S1	Fz Post-Training Post-Stress S2
Fz	r	1	.387*	0.023	.470**	-0.058	0.302	-0.079	-0.049	-0.343	-0.333	-0.283	-0.222
Pre-Training	p		0.035	0.905	0.009	0.762	0.105	0.678	0.797	0.064	0.072	0.13	0.239
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	.387*	1	-0.15	.506**	-0.265	-0.107	-0.248	-0.038	-.617**	-.623**	-.495**	-.435*
Pre-Training	p	0.035		0.43	0.004	0.157	0.575	0.186	0.842	0	0	0.005	0.016
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.023	-0.15	1	0.107	-0.026	-0.013	0.083	-0.008	0.207	0.288	0.172	0.185
Post-Training	p	0.905	0.43		0.575	0.891	0.946	0.663	0.967	0.272	0.122	0.364	0.328
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	.470**	.506**	0.107	1	-0.174	0.01	-0.265	-0.195	-0.133	-0.123	-0.012	0.111
Post-Training	p	0.009	0.004	0.575		0.357	0.96	0.157	0.301	0.484	0.518	0.952	0.559
Post=Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.058	-0.265	-0.026	-0.174	1	.927**	.749**	.703**	-0.01	-0.008	-0.258	-0.284
Pre-Training	p	0.762	0.157	0.891	0.357		0	0	0	0.956	0.965	0.168	0.129
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.302	-0.107	-0.013	0.01	.927**	1	.675**	.660**	-0.13	-0.125	-0.322	-0.323
Pre-Training	p	0.105	0.575	0.946	0.96	0		0	0	0.494	0.51	0.083	0.082
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.079	-0.248	0.083	-0.265	.749**	.675**	1	.952**	-0.07	-0.052	-0.342	-.370*
Pre-Training	p	0.678	0.186	0.663	0.157	0	0		0	0.714	0.785	0.065	0.044
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.049	-0.038	-0.008	-0.195	.703**	.660**	.952**	1	-0.188	-0.177	-.416*	-.436*
Pre-Training	p	0.797	0.842	0.967	0.301	0	0	0		0.32	0.351	0.022	0.016
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.343	-.617**	0.207	-0.133	-0.01	-0.13	-0.07	-0.188	1	.994**	.913**	.884**
Post=Training	p	0.064	0	0.272	0.484	0.956	0.494	0.714	0.32		0	0	0
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.333	-.623**	0.288	-0.123	-0.008	-0.125	-0.052	-0.177	.994**	1	.902**	.875**
Post-Training	p	0.072	0	0.122	0.518	0.965	0.51	0.785	0.351	0		0	0
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.283	-.495**	0.172	-0.012	-0.258	-0.322	-0.342	-.416*	.913**	.902**	1	.992**
Post-Training	p	0.13	0.005	0.364	0.952	0.168	0.083	0.065	0.022	0	0		0
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.222	-.435*	0.185	0.111	-0.284	-0.323	-.370*	-.436*	.884**	.875**	.992**	1
Post-Training	p	0.239	0.016	0.328	0.559	0.129	0.082	0.044	0.016	0	0	0	
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 6. Pz Site T/C Ratio, S1 (C), and S2 (T) Correlations

		Pz Pre-Training Baseline Ratio	Pz Pre-Training Post-Stress Ratio	Pz Post-Training Baseline Ratio	Pz Post-Training Post-Stress Ratio	Pz Pre-Training Baseline S1	Pz Pre-Training Baseline S2	Pz Pre-Training Post-Stress S1	Pz Pre-Training Post-Stress S2	Pz Post-Training Baseline S1	Pz Post-Training Baseline S2	Pz Post-Training Post-Stress S1	Pz Post-Training Post-Stress S2
Pz	r	1	0.263	0.212	0.336	-0.318	0.063	-.366*	-0.329	-0.077	0.126	0.122	-0.014
Pre-Training	p		0.161	0.261	0.069	0.087	0.742	0.047	0.076	0.685	0.507	0.519	0.943
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.263	1	0.05	.483**	-.463**	-.372*	-.554**	-0.307	-0.217	-0.145	0.007	-0.145
Pre-Training	p	0.161		0.793	0.007	0.01	0.043	0.001	0.099	0.25	0.445	0.97	0.444
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.212	0.05	1	0.102	-0.189	-0.112	-0.2	-0.206	-0.335	.502**	0.09	-0.013
Post-Training	p	0.261	0.793		0.592	0.318	0.555	0.288	0.276	0.07	0.005	0.638	0.944
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.336	.483**	0.102	1	-.439*	-0.335	-.496**	-.408*	-0.217	-0.089	0.114	-0.317
Post-Training	p	0.069	0.007	0.592		0.015	0.07	0.005	0.025	0.249	0.64	0.548	0.088
Post=Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.318	-.463**	-0.189	-.439*	1	.923**	.951**	.930**	.444*	0.185	-0.244	-0.008
Pre-Training	p	0.087	0.01	0.318	0.015		0	0	0	0.014	0.327	0.194	0.965
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.063	-.372*	-0.112	-0.335	.923**	1	.849**	.847**	.450*	0.265	-0.206	0
Pre-Training	p	0.742	0.043	0.555	0.07	0		0	0	0.013	0.157	0.274	0.999
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-.366*	-.554**	-0.2	-.496**	.951**	.849**	1	.961**	.507**	0.237	-0.144	0.076
Pre-Training	p	0.047	0.001	0.288	0.005	0	0		0	0.004	0.207	0.449	0.688
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.329	-0.307	-0.206	-.408*	.930**	.847**	.961**	1	.508**	0.235	-0.144	0.059
Pre-Training	p	0.076	0.099	0.276	0.025	0	0	0		0.004	0.212	0.447	0.758
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.077	-0.217	-0.335	-0.217	.444*	.450*	.507**	.508**	1	.623**	.418*	.659**
Post=Training	p	0.685	0.25	0.07	0.249	0.014	0.013	0.004	0.004		0	0.021	0
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.126	-0.145	.502**	-0.089	0.185	0.265	0.237	0.235	.623**	1	.441*	.604**
Post-Training	p	0.507	0.445	0.005	0.64	0.327	0.157	0.207	0.212	0		0.015	0
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.122	0.007	0.09	0.114	-0.244	-0.206	-0.144	-0.144	.418*	.441*	1	.831**
Post-Training	p	0.519	0.97	0.638	0.548	0.194	0.274	0.449	0.447	0.021	0.015		0
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.014	-0.145	-0.013	-0.317	-0.008	0	0.076	0.059	.659**	.604**	.831**	1
Post-Training	p	0.943	0.444	0.944	0.088	0.965	0.999	0.688	0.758	0	0	0	
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 7a. Fz and Pz Site T/C Ratio, S1 (C), and S2 (T) Correlations

		Fz Pre-Training Baseline Ratio	Fz Pre-Training Post-Stress Ratio	Fz Post-Training Baseline Ratio	Fz Post-Training Post-Stress Ratio	Fz Pre-Training Baseline S1	Fz Pre-Training Baseline S2	Fz Pre-Training Post-Stress S1	Fz Pre-Training Post-Stress S2	Fz Post-Training Baseline S1	Fz Post-Training Baseline S2	Fz Post-Training Post-Stress S1	Fz Post-Training Post-Stress S2
Pz	r	0.207	0.022	0.097	.493**	0.148	0.205	0.224	0.203	-0.015	0.01	-0.016	0.052
Pre-Training	p	0.273	0.91	0.61	0.006	0.435	0.277	0.234	0.283	0.939	0.958	0.933	0.785
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.012	0.123	0.162	0.257	-0.146	-0.103	-0.068	0.03	0.047	0.066	0.199	0.238
Pre-Training	p	0.948	0.519	0.393	0.17	0.441	0.587	0.723	0.873	0.804	0.728	0.293	0.206
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.05	-0.033	0.238	0.019	-0.03	-0.057	0.179	0.252	0.068	0.103	0.002	0.002
Post-Training	p	0.794	0.862	0.205	0.922	0.876	0.766	0.344	0.178	0.723	0.589	0.991	0.992
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	0.009	0.227	0.205	0.356	-0.27	-0.241	-0.291	-0.216	-0.152	-0.124	0.033	0.079
Post-Training	p	0.961	0.228	0.277	0.053	0.149	0.2	0.119	0.252	0.422	0.512	0.863	0.678
Post=Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-.366*	-.431*	-0.074	-.510**	0.256	0.109	0.297	0.181	-0.076	-0.09	-0.238	-0.292
Pre-Training	p	0.047	0.017	0.697	0.004	0.172	0.566	0.112	0.339	0.69	0.637	0.206	0.117
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.311	-.456*	-0.044	-0.355	0.359	0.221	.442*	0.309	-0.099	-0.102	-0.269	-0.3
Pre-Training	p	0.094	0.011	0.816	0.054	0.051	0.239	0.014	0.096	0.604	0.593	0.15	0.107
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.355	-.442*	-0.095	-.530**	0.23	0.078	0.29	0.168	-0.02	-0.05	-0.189	-0.249
Pre-Training	p	0.054	0.014	0.616	0.003	0.221	0.683	0.12	0.375	0.918	0.793	0.317	0.184
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-.404*	-.462*	-0.052	-.544**	0.211	0.047	0.326	0.216	-0.024	-0.052	-0.17	-0.229
Pre-Training	p	0.027	0.01	0.785	0.002	0.264	0.804	0.079	0.251	0.9	0.787	0.368	0.224
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-.414*	-.613**	-0.049	-.382*	.487**	0.32	0.214	0.088	0.361	0.327	0.304	0.261
Post=Training	p	0.023	0	0.798	0.037	0.006	0.085	0.256	0.644	0.05	0.078	0.102	0.164
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-.431*	-.608**	0.156	-0.339	.369*	0.193	0.332	0.271	.381*	.384*	0.295	0.256
Post-Training	p	0.017	0	0.41	0.067	0.045	0.307	0.073	0.147	0.038	0.036	0.113	0.172
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.123	-0.207	0.114	-0.165	.461*	0.347	0.228	0.165	0.351	0.344	0.175	0.139
Post-Training	p	0.517	0.272	0.547	0.383	0.01	0.06	0.225	0.382	0.057	0.063	0.356	0.464
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Pz	r	-0.2	-.473**	0.049	-.399*	.519**	.392*	0.311	0.194	.487**	.477**	0.307	0.25
Post-Training	p	0.29	0.008	0.796	0.029	0.003	0.032	0.095	0.304	0.006	0.008	0.098	0.182
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).

Table 7b. Fz and Pz Site T/C Ratio, S1 (C), and S2 (T) Correlations

		Pz Pre-Training Baseline Ratio	Pz Pre-Training Post-Stress Ratio	Pz Post-Training Baseline Ratio	Pz Post-Training Post-Stress Ratio	Pz Pre-Training Baseline S1	Pz Pre-Training Baseline S2	Pz Pre-Training Post-Stress S1	Pz Pre-Training Post-Stress S2	Pz Post-Training Baseline S1	Pz Post-Training Baseline S2	Pz Post-Training Post-Stress S1	Pz Post-Training Post-Stress S2
Fz	r	0.207	0.012	-0.05	0.009	-.366*	-0.311	-0.355	-.404*	-.414*	-.431*	-0.123	-0.2
Pre-Training	p	0.273	0.948	0.794	0.961	0.047	0.094	0.054	0.027	0.023	0.017	0.517	0.29
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.022	0.123	-0.033	0.227	-.431*	-.456*	-.442*	-.462*	-.613**	-.608**	-0.207	-.473**
Pre-Training	p	0.91	0.519	0.862	0.228	0.017	0.011	0.014	0.01	0	0	0.272	0.008
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.097	0.162	0.238	0.205	-0.074	-0.044	-0.095	-0.052	-0.049	0.156	0.114	0.049
Post-Training	p	0.61	0.393	0.205	0.277	0.697	0.816	0.616	0.785	0.798	0.41	0.547	0.796
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	.493**	0.257	0.019	0.356	-.510**	-0.355	-.530**	-.544**	-.382*	-0.339	-0.165	-.399*
Post-Training	p	0.006	0.17	0.922	0.053	0.004	0.054	0.003	0.002	0.037	0.067	0.383	0.029
Post=Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.148	-0.146	-0.03	-0.27	0.256	0.359	0.23	0.211	.487**	.369*	.461*	.519**
Pre-Training	p	0.435	0.441	0.876	0.149	0.172	0.051	0.221	0.264	0.006	0.045	0.01	0.003
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.205	-0.103	-0.057	-0.241	0.109	0.221	0.078	0.047	0.32	0.193	0.347	.392*
Pre-Training	p	0.277	0.587	0.766	0.2	0.566	0.239	0.683	0.804	0.085	0.307	0.06	0.032
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.224	-0.068	0.179	-0.291	0.297	.442*	0.29	0.326	0.214	0.332	0.228	0.311
Pre-Training	p	0.234	0.723	0.344	0.119	0.112	0.014	0.12	0.079	0.256	0.073	0.225	0.095
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.203	0.03	0.252	-0.216	0.181	0.309	0.168	0.216	0.088	0.271	0.165	0.194
Pre-Training	p	0.283	0.873	0.178	0.252	0.339	0.096	0.375	0.251	0.644	0.147	0.382	0.304
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.015	0.047	0.068	-0.152	-0.076	-0.099	-0.02	-0.024	0.361	.381*	0.351	.487**
Post=Training	p	0.939	0.804	0.723	0.422	0.69	0.604	0.918	0.9	0.05	0.038	0.057	0.006
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.01	0.066	0.103	-0.124	-0.09	-0.102	-0.05	-0.052	0.327	.384*	0.344	.477**
Post-Training	p	0.958	0.728	0.589	0.512	0.637	0.593	0.793	0.787	0.078	0.036	0.063	0.008
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	-0.016	0.199	0.002	0.033	-0.238	-0.269	-0.189	-0.17	0.304	0.295	0.175	0.307
Post-Training	p	0.933	0.293	0.991	0.863	0.206	0.15	0.317	0.368	0.102	0.113	0.356	0.098
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30
Fz	r	0.052	0.238	0.002	0.079	-0.292	-0.3	-0.249	-0.229	0.261	0.256	0.139	0.25
Post-Training	p	0.785	0.206	0.992	0.678	0.117	0.107	0.184	0.224	0.164	0.172	0.464	0.182
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 8a. Psychological Inventory Correlations

		Pre-Training Pain Score	Post-Training Pain Score	Pre-Training Stress Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood	Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Pre-Training	r	1	.390*	.565**	.387*	-0.076	0.084	0.037	-0.19	0.112	0.056	0.015	0.226	-0.185	-0.119	-0.17	0.193	-0.337	-0.137	0.091	0.17
Pain Score	p		0.033	0.004	0.042	0.694	0.666	0.851	0.333	0.557	0.767	0.937	0.23	0.336	0.556	0.379	0.315	0.341	0.705	0.802	0.638
	N	30	30	24	28	29	29	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	.390*	1	0.056	.717**	0.024	0.122	0.042	-.396*	0.092	0.077	0.063	0.049	0.082	-0.069	-0.08	0.148	-0.093	0.018	0.164	0.181
Pain Score	p	0.033		0.796	0	0.901	0.53	0.833	0.037	0.629	0.685	0.742	0.799	0.671	0.733	0.68	0.444	0.798	0.961	0.651	0.616
	N	30	30	24	28	29	29	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-Training	r	.565**	0.056	1	0.212	-0.006	0.058	0.183	0.011	0.199	-0.065	0.172	0.09	0.101	0.077	0.041	0.077	-0.024	-0.057	0.357	0.408
Stress Score	p	0.004	0.796		0.331	0.979	0.788	0.393	0.959	0.351	0.764	0.421	0.676	0.638	0.72	0.848	0.728	0.95	0.885	0.346	0.275
	N	24	24	24	23	24	24	24	24	24	24	24	24	24	24	24	23	9	9	9	9
Post-Training	r	.387*	.717**	0.212	1	-0.111	0.085	0.261	-0.307	0.145	0.243	0.122	0.124	0.207	-0.37	0.177	-0.013	-0.234	0.01	0.328	0.383
Stress Score	p	0.042	0	0.331		0.58	0.673	0.198	0.127	0.462	0.213	0.536	0.528	0.301	0.068	0.377	0.947	0.515	0.978	0.355	0.274
	N	28	28	23	28	27	27	26	26	28	28	28	28	27	25	27	27	10	10	10	10
Pre-Training	r	-0.076	0.024	-0.006	-0.111	1	-0.148	.668**	0.046	.693**	-.456*	.600**	-.372*	-0.06	0.131	-0.367	-0.341	.779**	.717*	-0.184	-0.286
Baseline	p	0.694	0.901	0.979	0.58		0.444	0	0.815	0	0.013	0.001	0.047	0.761	0.516	0.055	0.076	0.008	0.02	0.611	0.423
Positive Mood	N	29	29	24	27	29	29	28	28	29	29	29	29	28	27	28	28	10	10	10	10
Pre-Training	r	0.084	0.122	0.058	0.085	-0.148	1	-0.043	0.252	-0.232	0.27	-0.272	0.216	.554**	.601**	.562**	.556**	-0.544	-0.515	0.321	0.463
Baseline	p	0.666	0.53	0.788	0.673	0.444		0.829	0.195	0.226	0.157	0.153	0.261	0.002	0.001	0.002	0.002	0.104	0.128	0.366	0.178
Negative Mood	N	29	29	24	27	29	29	28	28	29	29	29	29	28	27	28	28	10	10	10	10
Pre-Training	r	0.037	0.042	0.183	0.261	.668**	-0.043	1	0.133	.805**	-0.125	.793**	-0.147	0.105	0.01	-0.003	-0.265	0.614	0.469	0.313	0.166
Post-Stress	p	0.851	0.833	0.393	0.198	0	0.829		0.501	0	0.527	0	0.456	0.603	0.962	0.988	0.182	0.059	0.171	0.378	0.646
Positive Mood	N	28	28	24	26	28	28	28	28	28	28	28	28	27	27	28	27	10	10	10	10
Pre-Training	r	-0.19	-.396*	0.011	-0.307	0.046	0.252	0.133	1	0.075	0.047	0.036	0.346	0.236	.563**	0.102	0.233	0.067	-0.087	0.131	0.279
Post-Stress	p	0.333	0.037	0.959	0.127	0.815	0.195	0.501		0.705	0.812	0.857	0.071	0.236	0.002	0.606	0.242	0.854	0.812	0.718	0.435
Negative Mood	N	28	28	24	26	28	28	28	28	28	28	28	28	27	27	28	27	10	10	10	10
Post-Training	r	0.112	0.092	0.199	0.145	.693**	-0.232	.805**	0.075	1	-.407*	.873**	-0.212	-0.043	0	-0.356	-0.114	0.538	0.526	0.023	-0.051
Baseline	p	0.557	0.629	0.351	0.462	0	0.226	0	0.705		0.025	0	0.26	0.824	0.999	0.058	0.556	0.109	0.118	0.951	0.888
Positive Mood	N	30	30	24	28	29	29	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	0.056	0.077	-0.065	0.243	-.456*	0.27	-0.125	0.047	-.407*	1	-0.256	.653**	0.357	0.046	.646**	0.274	-0.105	0.07	0.392	.672*
Baseline	p	0.767	0.685	0.764	0.213	0.013	0.157	0.527	0.812	0.025		0.173	0	0.057	0.821	0	0.15	0.772	0.848	0.262	0.033
Negative Mood	N	30	30	24	28	29	29	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	0.015	0.063	0.172	0.122	.600**	-0.272	.793**	0.036	.873**	-0.256	1	-0.299	0.138	-0.027	-0.142	-0.19	0.477	0.373	0.077	-0.077
Post-Stress	p	0.937	0.742	0.421	0.536	0.001	0.153	0	0.857	0	0.173		0.108	0.475	0.894	0.463	0.323	0.163	0.288	0.832	0.832
Positive Mood	N	30	30	24	28	29	29	28	28	30	30	30	30	29	27	29	29	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 8b. Psychological Inventory Correlations

		Pre-Training Pain Score	Post-Training Pain Score	Pre-Training Stress Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood	Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Post-Training	r	0.226	0.049	0.09	0.124	-.372*	0.216	-0.147	0.346	-0.212	.653**	-0.299	1	0.17	0.307	0.34	.547**	-0.087	-0.04	0.132	0.429
Post-Stress	p	0.23	0.799	0.676	0.528	0.047	0.261	0.456	0.071	0.26	0	0.108		0.377	0.119	0.071	0.002	0.812	0.913	0.715	0.217
Negative Mood	N	30	30	24	28	29	29	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-training	r	-0.185	0.082	0.101	0.207	-0.06	.554**	0.105	0.236	-0.043	0.357	0.138	0.17	1	.505**	.680**	0.34	-0.221	-0.136	0.385	.680*
Baseline SAI	p	0.336	0.671	0.638	0.301	0.761	0.002	0.603	0.236	0.824	0.057	0.475	0.377		0.008	0	0.077	0.569	0.728	0.306	0.044
	N	29	29	24	27	28	28	27	27	29	29	29	29	29	26	28	28	9	9	9	9
Pre-Training	r	-0.119	-0.069	0.077	-0.37	0.131	.601**	0.01	.563**	0	0.046	-0.027	0.307	.505**	1	0.313	.566**	-0.228	-0.263	0.29	0.531
Post-Stress SAI	p	0.556	0.733	0.72	0.068	0.516	0.001	0.962	0.002	0.999	0.821	0.894	0.119	0.008		0.112	0.003	0.526	0.462	0.416	0.114
	N	27	27	24	25	27	27	27	27	27	27	27	27	26	27	27	26	10	10	10	10
Post-training	r	-0.17	-0.08	0.041	0.177	-0.367	.562**	-0.003	0.102	-0.356	.646**	-0.142	0.34	.680**	0.313	1	0.283	-0.26	-0.294	0.603	0.607
Baseline SAI	p	0.379	0.68	0.848	0.377	0.055	0.002	0.988	0.606	0.058	0	0.463	0.071	0	0.112		0.144	0.468	0.41	0.065	0.063
	N	29	29	24	27	28	28	28	28	29	29	29	29	28	27	29	28	10	10	10	10
Post-Training	r	0.193	0.148	0.077	-0.013	-0.341	.556**	-0.265	0.233	-0.114	0.274	-0.19	.547**	0.34	.566**	0.283	1	-0.348	-0.168	0.079	0.374
Post-Stress SAI	p	0.315	0.444	0.728	0.947	0.076	0.002	0.182	0.242	0.556	0.15	0.323	0.002	0.077	0.003	0.144		0.324	0.643	0.827	0.287
	N	29	29	23	27	28	28	27	27	29	29	29	29	28	26	28	29	10	10	10	10
Pre-Training	r	-0.337	-0.093	-0.024	-0.234	.779**	-0.544	0.614	0.067	0.538	-0.105	0.477	-0.087	-0.221	-0.228	-0.26	-0.348	1	.853**	-0.215	-0.295
FMI Score	p	0.341	0.798	0.95	0.515	0.008	0.104	0.059	0.854	0.109	0.772	0.163	0.812	0.569	0.526	0.468	0.324		0.002	0.551	0.408
	N	10	10	9	10	10	10	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Post-Training	r	-0.137	0.018	-0.057	0.01	.717*	-0.515	0.469	-0.087	0.526	0.07	0.373	-0.04	-0.136	-0.263	-0.294	-0.168	.853**	1	-0.345	-0.226
FMI Score	p	0.705	0.961	0.885	0.978	0.02	0.128	0.171	0.812	0.118	0.848	0.288	0.913	0.728	0.462	0.41	0.643	0.002		0.328	0.531
	N	10	10	9	10	10	10	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Pre-Training	r	0.091	0.164	0.357	0.328	-0.184	0.321	0.313	0.131	0.023	0.392	0.077	0.132	0.385	0.29	0.603	0.079	-0.215	-0.345	1	.845**
MAAS Score	p	0.802	0.651	0.346	0.355	0.611	0.366	0.378	0.718	0.951	0.262	0.832	0.715	0.306	0.416	0.065	0.827	0.551	0.328		0.002
	N	10	10	9	10	10	10	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Post-Training	r	0.17	0.181	0.408	0.383	-0.286	0.463	0.166	0.279	-0.051	.672*	-0.077	0.429	.680*	0.531	0.607	0.374	-0.295	-0.226	.845**	1
MAAS Score	p	0.638	0.616	0.275	0.274	0.423	0.178	0.646	0.435	0.888	0.033	0.832	0.217	0.044	0.114	0.063	0.287	0.408	0.531	0.002	
	N	10	10	9	10	10	10	10	10	10	10	10	10	9	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9a. Fz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Fz Pre-Training Baseline S1	Fz Pre-Training Baseline S2	Fz Pre-Training Baseline Ratio	Fz Pre-Training Post-Stress S1	Fz Pre-Training Post-Stress S2	Fz Pre-Training Post-Stress Ratio	Fz Post-Training Baseline S1	Fz Post-Training Baseline S2	Fz Post-Training Baseline Ratio	Fz Post-Training Post-Stress S1	Fz Post-Training Post-Stress S2	Fz Post-Training Post-Stress Ratio	Pre-Training McGill Pain Scale	Post-Training McGill Pain Scale	Pre-Training Pain Score	Pre-Training Stress Score	Post-Training Pain Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood
Fz	r	1	.927**	-.058	.749**	.703**	-.265	-.010	-.008	-.026	-.258	-.284	-.174	-.198	-.240	-.208	.156	-.029	-.074	.113	.137
Pre-Training	p		.000	.762	.000	.000	.157	.956	.965	.891	.168	.129	.357	.295	.201	.329	.468	.882	.709	.561	.478
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	.927**	1	.302	.675**	.660**	-.107	-.130	-.125	-.013	-.322	-.323	.010	-.249	-.308	-.300	.052	-.157	-.161	.080	.048
Pre-Training	p	.000		.105	.000	.000	.575	.494	.510	.946	.083	.082	.960	.184	.098	.154	.810	.425	.412	.678	.807
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.058	.302	1	-.079	-.049	.387*	-.343	-.333	.023	-.283	-.222	.470**	-.124	-.172	-.275	-.327	-.337	-.228	-.068	-.152
Pre-Training	p	.762	.105		.678	.797	.035	.064	.072	.905	.130	.239	.009	.515	.365	.194	.119	.080	.243	.728	.433
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	.749**	.675**	-.079	1	.952**	-.248	-.070	-.052	.083	-.342	-.370*	-.265	-.345	-.398*	-.277	.084	-.055	-.327	.260	.028
Pre-Training	p	.000	.000	.678		.000	.186	.714	.785	.663	.065	.044	.157	.062	.029	.191	.697	.782	.090	.174	.886
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	.703**	.660**	-.049	.952**	1	-.038	-.188	-.177	-.008	-.416*	-.436*	-.195	-.399*	-.471**	-.344	.045	-.069	-.384*	.216	-.047
Pre-Training	p	.000	.000	.797	.000		.842	.320	.351	.967	.022	.016	.301	.029	.009	.100	.833	.727	.044	.261	.809
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.265	-.107	.387*	-.248	-.038	1	-.617**	-.623**	-.150	-.495**	-.435*	.506**	-.309	-.151	-.281	-.462*	-.120	-.155	-.213	-.164
Pre-Training	p	.157	.575	.035	.186	.842		.000	.000	.430	.005	.016	.004	.097	.426	.183	.023	.541	.432	.268	.394
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.010	-.130	-.343	-.070	-.188	-.617**	1	.994**	.207	.913**	.884**	-.133	.439*	.188	.280	.283	.186	.320	.369*	.111
Post-Training	p	.956	.494	.064	.714	.320	.000		.000	.272	.000	.000	.484	.015	.320	.185	.180	.343	.097	.049	.566
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.008	-.125	-.333	-.052	-.177	-.623**	.994**	1	.288	.902**	.875**	-.123	.412*	.184	.257	.274	.192	.328	.392*	.117
Post-Training	p	.965	.510	.072	.785	.351	.000	.000		.122	.000	.000	.518	.024	.331	.226	.194	.328	.088	.036	.544
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.026	-.013	.023	.083	-.008	-.150	.207	.288	1	.172	.185	.107	-.246	-.227	-.323	-.183	-.152	-.003	.141	-.148
Post-Training	p	.891	.946	.905	.663	.967	.430	.272	.122		.364	.328	.575	.191	.229	.123	.391	.440	.987	.465	.444
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.258	-.322	-.283	-.342	-.416*	-.495**	.913**	.902**	.172	1	.992**	-.012	.455*	.245	.290	.240	.174	.376*	.290	.004
Post-Training	p	.168	.083	.130	.065	.022	.005	.000	.000	.364		.000	.952	.011	.192	.169	.258	.374	.049	.127	.984
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Fz	r	-.284	-.323	-.222	-.370*	-.436*	-.435*	.884**	.875**	.185	.992**	1	.111	.421*	.253	.281	.188	.185	.394*	.303	-.041
Post-Training	p	.129	.082	.239	.044	.016	.016	.000	.000	.328	.000		.559	.021	.178	.184	.378	.346	.038	.110	.831
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9b. Fz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Fz	r	.214	.469*	.174	-.055	.028	.107	.177	.236	.089	.085	.392	.175	.301	.194
Pre-Training	p	.275	.012	.357	.772	.883	.573	.358	.236	.646	.659	.262	.629	.399	.591
Baseline S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.160	.509**	.071	-.046	-.094	.041	.007	.146	.019	-.074	.491	.265	.181	.139
Pre-Training	p	.417	.006	.708	.811	.623	.830	.973	.467	.920	.703	.150	.459	.617	.702
Baseline S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	-.115	.036	-.290	.065	-.357	-.170	-.441*	-.200	-.090	-.382*	.453	.378	-.279	-.166
Pre-Training	p	.558	.855	.120	.732	.053	.368	.017	.317	.641	.041	.189	.281	.434	.647
Baseline Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.112	.565**	.115	-.191	.087	.019	.183	.450*	.029	.038	.358	.181	.263	.205
Pre-Training	p	.570	.002	.545	.312	.647	.921	.343	.018	.881	.845	.310	.617	.463	.569
Post-Stress S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.057	.621**	.083	-.189	.042	.032	.131	.379	-.042	-.006	.408	.230	.223	.193
Pre-Training	p	.773	.000	.664	.318	.827	.868	.497	.052	.828	.975	.241	.523	.536	.593
Post-Stress S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	-.231	.029	-.148	.144	-.202	.065	-.127	-.134	.002	-.050	.174	.246	-.158	-.019
Pre-Training	p	.236	.885	.436	.447	.285	.733	.512	.506	.990	.798	.631	.493	.662	.958
Post-Stress Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.459*	-.076	.408*	-.184	.433*	-.189	.012	-.120	-.002	-.122	-.070	-.157	.238	-.084
Post-Training	p	.014	.701	.025	.332	.017	.318	.949	.552	.990	.530	.849	.666	.508	.817
Baseline S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.491**	-.075	.428*	-.170	.463*	-.201	.040	-.114	.028	-.116	-.029	-.091	.276	-.005
Post-Training	p	.008	.704	.018	.370	.010	.286	.835	.570	.886	.549	.937	.803	.441	.989
Baseline S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.261	-.183	.193	.014	.373*	-.347	.137	-.265	.200	-.177	.138	.405	.239	.369
Post-Training	p	.180	.351	.307	.940	.042	.060	.479	.181	.298	.359	.704	.246	.506	.294
Baseline Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.431*	-.139	.364*	-.110	.442*	-.191	.035	-.265	-.059	-.233	-.215	-.304	.008	-.393
Post-Training	p	.022	.482	.048	.563	.015	.313	.858	.181	.760	.224	.551	.392	.982	.262
Post-Stress S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Fz	r	.458*	-.150	.372*	-.089	.442*	-.193	.006	-.308	-.073	-.273	-.109	-.206	-.012	-.444
Post-Training	p	.014	.447	.043	.638	.014	.306	.975	.118	.706	.153	.763	.568	.975	.198
Post-Stress S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9c. Fz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Fz Pre-Training Baseline S1	Fz Pre-Training Baseline S2	Fz Pre-Training Baseline Ratio	Fz Pre-Training Post-Stress S1	Fz Pre-Training Post-Stress S2	Fz Pre-Training Post-Stress Ratio	Fz Post-Training Baseline S1	Fz Post-Training Baseline S2	Fz Post-Training Baseline Ratio	Fz Post-Training Post-Stress S1	Fz Post-Training Post-Stress S2	Fz Post-Training Post-Stress Ratio	Pre-Training McGill Pain Scale	Post-Training McGill Pain Scale	Pre-Training Pain Score	Pre-Training Stress Score	Post-Training Pain Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood
Fz	r	-.174	.010	.470**	-.265	-.195	.506**	-.133	-.123	.107	-.012	.111	1	-.231	.038	-.031	-.427*	.071	.159	.117	-.368*
Post-Training	p	.357	.960	.009	.157	.301	.004	.484	.518	.575	.952	.559		.219	.842	.884	.037	.721	.419	.547	.050
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pre-Training	r	-.198	-.249	-.124	-.345	-.399*	-.309	.439*	.412*	-.246	.455*	.421*	-.231	1	.390*	.699**	.565**	.244	.387*	-.076	.084
McGill Pain Scale	p	.295	.184	.515	.062	.029	.097	.015	.024	.191	.011	.021	.219		.033	.000	.004	.211	.042	.694	.666
	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	-.240	-.308	-.172	-.398*	-.471**	-.151	.188	.184	-.227	.245	.253	.038	.390*	1	.564**	.056	.812**	.717**	.024	.122
McGill Pain Scale	p	.201	.098	.365	.029	.009	.426	.320	.331	.229	.192	.178	.842	.033		.004	.796	.000	.000	.901	.530
	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pre-Training	r	-.208	-.300	-.275	-.277	-.344	-.281	.280	.257	-.323	.290	.281	-.031	.699**	.564**	1	.409*	.417*	.292	.224	-.117
Pain Score	p	.329	.154	.194	.191	.100	.183	.185	.226	.123	.169	.184	.884	.000	.004		.047	.048	.177	.292	.587
	N	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	23	23	24	24
Pre-Training	r	.156	.052	-.327	.084	.045	-.462*	.283	.274	-.183	.240	.188	-.427*	.565**	.056	.409*	1	.129	.212	-.006	.058
Stress Score	p	.468	.810	.119	.697	.833	.023	.180	.194	.391	.258	.378	.037	.004	.796	.047		.558	.331	.979	.788
	N	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	23	23	24	24
Post-Training	r	-.029	-.157	-.337	-.055	-.069	-.120	.186	.192	-.152	.174	.185	.071	.244	.812**	.417*	.129	1	.652**	.072	.090
Pain Score	p	.882	.425	.080	.782	.727	.541	.343	.328	.440	.374	.346	.721	.211	.000	.048	.558		.000	.720	.656
	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	23	23	28	28	27	27
Post-Training	r	-.074	-.161	-.228	-.327	-.384*	-.155	.320	.328	-.003	.376*	.394*	.159	.387*	.717**	.292	.212	.652**	1	-.111	.085
Stress Score	p	.709	.412	.243	.090	.044	.432	.097	.088	.987	.049	.038	.419	.042	.000	.177	.331	.000		.580	.673
	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	23	23	28	28	27	27
Pre-Training	r	.113	.080	-.068	.260	.216	-.213	.369*	.392*	.141	.290	.303	.117	-.076	.024	.224	-.006	.072	-.111	1	-.148
Baseline	p	.561	.678	.728	.174	.261	.268	.049	.036	.465	.127	.110	.547	.694	.901	.292	.979	.720	.580		.444
Positive Mood	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	29	29
Pre-Training	r	.137	.048	-.152	.028	-.047	-.164	.111	.117	-.148	.004	-.041	-.368*	.084	.122	-.117	.058	.090	.085	-.148	1
Baseline	p	.478	.807	.433	.886	.809	.394	.566	.544	.444	.984	.831	.050	.666	.530	.587	.788	.656	.673	.444	
Negative Mood	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	29	29
Pre-Training	r	.214	.160	-.115	.112	.057	-.231	.459*	.491**	.261	.431*	.458*	.241	.037	.042	.088	.183	.103	.261	.668**	-.043
Post-Stress	p	.275	.417	.558	.570	.773	.236	.014	.008	.180	.022	.014	.216	.851	.833	.682	.393	.616	.198	.000	.829
Positive Mood	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	24	24	26	26	28	28
Pre-Training	r	.469*	.509**	.036	.565**	.621**	.029	-.076	-.075	-.183	-.139	-.150	-.146	-.190	-.396*	-.403	.011	-.144	-.307	.046	.252
Post-Stress	p	.012	.006	.855	.002	.000	.885	.701	.704	.351	.482	.447	.460	.333	.037	.051	.959	.484	.127	.815	.195
Negative Mood	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	24	24	26	26	28	28

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9d. Fz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Fz	r	.241	-.146	.118	.134	.015	-.043	-.307	-.405*	-.114	-.359	.563	.532	-.059	-.284
Post-Training	p	.216	.460	.536	.481	.939	.822	.105	.036	.556	.056	.090	.113	.871	.426
Post-Stress Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-Training	r	.037	-.190	.112	.056	.015	.226	-.185	-.119	-.170	.193	-.337	-.137	.091	.170
McGill Pain Scale	p	.851	.333	.557	.767	.937	.230	.336	.556	.379	.315	.341	.705	.802	.638
	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	.042	-.396*	.092	.077	.063	.049	.082	-.069	-.080	.148	-.093	.018	.164	.181
McGill Pain Scale	p	.833	.037	.629	.685	.742	.799	.671	.733	.680	.444	.798	.961	.651	.616
	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-Training	r	.088	-.403	.387	-.309	.154	.075	-.273	-.022	-.472*	.121	-.206	.101	.125	.017
Pain Score	p	.682	.051	.062	.142	.474	.727	.197	.920	.020	.581	.595	.797	.749	.966
	N	24	24	24	24	24	24	24	24	24	23	9	9	9	9
Pre-Training	r	.183	.011	.199	-.065	.172	.090	.101	.077	.041	.077	-.024	-.057	.357	.408
Stress Score	p	.393	.959	.351	.764	.421	.676	.638	.720	.848	.728	.950	.885	.346	.275
	N	24	24	24	24	24	24	24	24	24	23	9	9	9	9
Post-Training	r	.103	-.144	.122	.159	.166	.023	.241	-.011	-.030	.144	.046	.117	.559	.477
Pain Score	p	.616	.484	.537	.420	.398	.907	.226	.959	.883	.475	.899	.748	.093	.163
	N	26	26	28	28	28	28	27	25	27	27	10	10	10	10
Post-Training	r	.261	-.307	.145	.243	.122	.124	.207	-.370	.177	-.013	-.234	.010	.328	.383
Stress Score	p	.198	.127	.462	.213	.536	.528	.301	.068	.377	.947	.515	.978	.355	.274
	N	26	26	28	28	28	28	27	25	27	27	10	10	10	10
Pre-Training	r	.668**	.046	.693**	-.456*	.600**	-.372*	-.060	.131	-.367	-.341	.779**	.717*	-.184	-.286
Baseline	p	.000	.815	.000	.013	.001	.047	.761	.516	.055	.076	.008	.020	.611	.423
Positive Mood	N	28	28	29	29	29	29	28	27	28	28	10	10	10	10
Pre-Training	r	-.043	.252	-.232	.270	-.272	.216	.554**	.601**	.562**	.556**	-.544	-.515	.321	.463
Baseline	p	.829	.195	.226	.157	.153	.261	.002	.001	.002	.002	.104	.128	.366	.178
Negative Mood	N	28	28	29	29	29	29	28	27	28	28	10	10	10	10
Pre-Training	r	1	.133	.805**	-.125	.793**	-.147	.105	.010	-.003	-.265	.614	.469	.313	.166
Post-Stress	p		.501	.000	.527	.000	.456	.603	.962	.988	.182	.059	.171	.378	.646
Positive Mood	N	28	28	28	28	28	28	27	27	28	27	10	10	10	10
Pre-Training	r	.133	1	.075	.047	.036	.346	.236	.563**	.102	.233	.067	-.087	.131	.279
Post-Stress	p	.501		.705	.812	.857	.071	.236	.002	.606	.242	.854	.812	.718	.435
Negative Mood	N	28	28	28	28	28	28	27	27	28	27	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9e. Fz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Fz Pre-Training Baseline S1	Fz Pre-Training Baseline S2	Fz Pre-Training Baseline Ratio	Fz Pre-Training Post-Stress S1	Fz Pre-Training Post-Stress S2	Fz Pre-Training Post-Stress Ratio	Fz Post-Training Baseline S1	Fz Post-Training Baseline S2	Fz Post-Training Baseline Ratio	Fz Post-Training Post-Stress S1	Fz Post-Training Post-Stress S2	Fz Post-Training Post-Stress Ratio	Pre-Training McGill Pain Scale	Post-Training McGill Pain Scale	Pre-Training Pain Score	Pre-Training Stress Score	Post-Training Pain Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood
Post-Training	r	.174	.071	-.290	.115	.083	-.148	.408*	.428*	.193	.364*	.372*	.118	.112	.092	.387	.199	.122	.145	.693**	-.232
Baseline	p	.357	.708	.120	.545	.664	.436	.025	.018	.307	.048	.043	.536	.557	.629	.062	.351	.537	.462	.000	.226
Positive Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	-.055	-.046	.065	-.191	-.189	.144	-.184	-.170	.014	-.110	-.089	.134	.056	.077	-.309	-.065	.159	.243	-.456*	.270
Baseline	p	.772	.811	.732	.312	.318	.447	.332	.370	.940	.563	.638	.481	.767	.685	.142	.764	.420	.213	.013	.157
Negative Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	.028	-.094	-.357	.087	.042	-.202	.433*	.463*	.373*	.442*	.442*	.015	.015	.063	.154	.172	.166	.122	.600**	-.272
Post-Stress	p	.883	.623	.053	.647	.827	.285	.017	.010	.042	.015	.014	.939	.937	.742	.474	.421	.398	.536	.001	.153
Positive Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	.107	.041	-.170	.019	.032	.065	-.189	-.201	-.347	-.191	-.193	-.043	.226	.049	.075	.090	.023	.124	-.372*	.216
Post-Stress	p	.573	.830	.368	.921	.868	.733	.318	.286	.060	.313	.306	.822	.230	.799	.727	.676	.907	.528	.047	.261
Negative Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pre-training	r	.177	.007	-.441*	.183	.131	-.127	.012	.040	.137	.035	.006	-.307	-.185	.082	-.273	.101	.241	.207	-.060	.554**
Baseline SAI	p	.358	.973	.017	.343	.497	.512	.949	.835	.479	.858	.975	.105	.336	.671	.197	.638	.226	.301	.761	.002
	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	28	28
Pre-Training	r	.236	.146	-.200	.450*	.379	-.134	-.120	-.114	-.265	-.265	-.308	-.405*	-.119	-.069	-.022	.077	-.011	-.370	.131	.601**
Post-Stress SAI	p	.236	.467	.317	.018	.052	.506	.552	.570	.181	.181	.118	.036	.556	.733	.920	.720	.959	.068	.516	.001
	N	27	27	27	27	27	27	27	27	27	27	27	27	27	27	24	24	25	25	27	27
Post-training	r	.089	.019	-.090	.029	-.042	.002	-.002	.028	.200	-.059	-.073	-.114	-.170	-.080	-.472*	.041	-.030	.177	-.367	.562**
Baseline SAI	p	.646	.920	.641	.881	.828	.990	.990	.886	.298	.760	.706	.556	.379	.680	.020	.848	.883	.377	.055	.002
	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	28	28
Post-Training	r	.085	-.074	-.382*	.038	-.006	-.050	-.122	-.116	-.177	-.233	-.273	-.359	.193	.148	.121	.077	.144	-.013	-.341	.556**
Post-Stress SAI	p	.659	.703	.041	.845	.975	.798	.530	.549	.359	.224	.153	.056	.315	.444	.581	.728	.475	.947	.076	.002
	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	23	23	27	27	28	28
Pre-Training	r	.392	.491	.453	.358	.408	.174	-.070	-.029	.138	-.215	-.109	.563	-.337	-.093	-.206	-.024	.046	-.234	.779**	-.544
FMI Score	p	.262	.150	.189	.310	.241	.631	.849	.937	.704	.551	.763	.090	.341	.798	.595	.950	.899	.515	.008	.104
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10
Post-Training	r	.175	.265	.378	.181	.230	.246	-.157	-.091	.405	-.304	-.206	.532	-.137	.018	.101	-.057	.117	.010	.717*	-.515
FMI Score	p	.629	.459	.281	.617	.523	.493	.666	.803	.246	.392	.568	.113	.705	.961	.797	.885	.748	.978	.020	.128
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10
Pre-Training	r	.301	.181	-.279	.263	.223	-.158	.238	.276	.239	.008	-.012	-.059	.091	.164	.125	.357	.559	.328	-.184	.321
MAAS Score	p	.399	.617	.434	.463	.536	.662	.508	.441	.506	.982	.975	.871	.802	.651	.749	.346	.093	.355	.611	.366
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10
Post-Training	r	.194	.139	-.166	.205	.193	-.019	-.084	-.005	.369	-.393	-.444	-.284	.170	.181	.017	.408	.477	.383	-.286	.463
MAAS Score	p	.591	.702	.647	.569	.593	.958	.817	.989	.294	.262	.198	.426	.638	.616	.966	.275	.163	.274	.423	.178
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9f. Fz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Post-Training	r	.805**	.075	1	-.407*	.873**	-.212	-.043	.000	-.356	-.114	.538	.526	.023	-.051
Baseline	p	.000	.705		.025	.000	.260	.824	.999	.058	.556	.109	.118	.951	.888
Positive Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	-.125	.047	-.407*	1	-.256	.653**	.357	.046	.646**	.274	-.105	.070	.392	.672*
Baseline	p	.527	.812	.025		.173	.000	.057	.821	.000	.150	.772	.848	.262	.033
Negative Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	.793**	.036	.873**	-.256	1	-.299	.138	-.027	-.142	-.190	.477	.373	.077	-.077
Post-Stress	p	.000	.857	.000	.173		.108	.475	.894	.463	.323	.163	.288	.832	.832
Positive Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	-.147	.346	-.212	.653**	-.299	1	.170	.307	.340	.547**	-.087	-.040	.132	.429
Post-Stress	p	.456	.071	.260	.000	.108		.377	.119	.071	.002	.812	.913	.715	.217
Negative Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-training	r	.105	.236	-.043	.357	.138	.170	1	.505**	.680**	.340	-.221	-.136	.385	.680*
Baseline SAI	p	.603	.236	.824	.057	.475	.377		.008	.000	.077	.569	.728	.306	.044
	N	27	27	29	29	29	29	29	26	28	28	9	9	9	9
Pre-Training	r	.010	.563**	.000	.046	-.027	.307	.505**	1	.313	.566**	-.228	-.263	.290	.531
Post-Stress SAI	p	.962	.002	.999	.821	.894	.119	.008		.112	.003	.526	.462	.416	.114
	N	27	27	27	27	27	27	26	27	27	26	10	10	10	10
Post-training	r	-.003	.102	-.356	.646**	-.142	.340	.680**	.313	1	.283	-.260	-.294	.603	.607
Baseline SAI	p	.988	.606	.058	.000	.463	.071	.000	.112		.144	.468	.410	.065	.063
	N	28	28	29	29	29	29	28	27	29	28	10	10	10	10
Post-Training	r	-.265	.233	-.114	.274	-.190	.547**	.340	.566**	.283	1	-.348	-.168	.079	.374
Post-Stress SAI	p	.182	.242	.556	.150	.323	.002	.077	.003	.144		.324	.643	.827	.287
	N	27	27	29	29	29	29	28	26	28	29	10	10	10	10
Pre-Training	r	.614	.067	.538	-.105	.477	-.087	-.221	-.228	-.260	-.348	1	.853**	-.215	-.295
FMI Score	p	.059	.854	.109	.772	.163	.812	.569	.526	.468	.324		.002	.551	.408
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Post-Training	r	.469	-.087	.526	.070	.373	-.040	-.136	-.263	-.294	-.168	.853**	1	-.345	-.226
FMI Score	p	.171	.812	.118	.848	.288	.913	.728	.462	.410	.643	.002		.328	.531
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Pre-Training	r	.313	.131	.023	.392	.077	.132	.385	.290	.603	.079	-.215	-.345	1	.845**
MAAS Score	p	.378	.718	.951	.262	.832	.715	.306	.416	.065	.827	.551	.328		.002
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Post-Training	r	.166	.279	-.051	.672*	-.077	.429	.680*	.531	.607	.374	-.295	-.226	.845**	1
MAAS Score	p	.646	.435	.888	.033	.832	.217	.044	.114	.063	.287	.408	.531	.002	
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 10a. Pz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pz Pre-Training Baseline S1	Pz Pre-Training Baseline S2	Pz Pre-Training Baseline Ratio	Pz Pre-Training Post-Stress S1	Pz Pre-Training Post-Stress S2	Pz Pre-Training Post-Stress Ratio	Pz Post-Training Baseline S1	Pz Post-Training Baseline S2	Pz Post-Training Baseline Ratio	Pz Post-Training Post-Stress S1	Pz Post-Training Post-Stress S2	Pz Post-Training Post-Stress Ratio	Pre-Training McGill Pain Scale	Post-Training McGill Pain Scale	Pre-Training Pain Score	Pre-Training Stress Score	Post-Training Pain Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood
Pz	r	1	.923**	-.318	.951**	.930**	-.463**	.444*	.185	-.189	-.244	-.008	-.439*	-.103	.176	.120	.004	.072	-.133	-.049	.243
Pre-Training	p		.000	.087	.000	.000	.010	.014	.327	.318	.194	.965	.015	.588	.351	.577	.984	.716	.501	.802	.204
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	.923**	1	.063	.849**	.847**	-.372*	.450*	.265	-.112	-.206	.000	-.335	-.102	.214	.107	-.001	.227	-.060	-.016	.243
Pre-Training	p	.000		.742	.000	.000	.043	.013	.157	.555	.274	.999	.070	.591	.257	.617	.998	.245	.760	.936	.204
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	-.318	.063	1	-.366*	-.329	.263	-.077	.126	.212	.122	-.014	.336	.092	.113	.012	-.009	.386*	.231	.016	-.054
Pre-Training	p	.087	.742		.047	.076	.161	.685	.507	.261	.519	.943	.069	.630	.551	.955	.967	.043	.238	.936	.779
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	.951**	.849**	-.366*	1	.961**	-.554**	.507**	.237	-.200	-.144	.076	-.496**	-.092	.089	.104	-.050	-.012	-.228	-.084	.165
Pre-Training	p	.000	.000	.047		.000	.001	.004	.207	.288	.449	.688	.005	.629	.639	.627	.817	.953	.244	.666	.393
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	.930**	.847**	-.329	.961**	1	-.307	.508**	.235	-.206	-.144	.059	-.408*	-.109	.060	.054	.023	.001	-.145	-.131	.154
Pre-Training	p	.000	.000	.076	.000		.099	.004	.212	.276	.447	.758	.025	.565	.751	.802	.917	.998	.461	.497	.427
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	-.463**	-.372*	.263	-.554**	-.307	1	-.217	-.145	.050	.007	-.145	.483**	-.032	-.062	-.186	.276	.092	.403*	-.077	-.125
Pre-Training	p	.010	.043	.161	.001	.099		.250	.445	.793	.970	.444	.007	.867	.745	.384	.191	.643	.033	.693	.518
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	.444*	.450*	-.077	.507**	.508**	-.217	1	.623**	-.335	.418*	.659**	-.217	.245	.022	.140	.181	-.031	.088	.044	.156
Post-Training	p	.014	.013	.685	.004	.004	.250		.000	.070	.021	.000	.249	.192	.907	.515	.396	.876	.658	.821	.418
Baseline S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	.185	.265	.126	.237	.235	-.145	.623**	1	.502**	.441*	.604**	-.089	.247	-.118	.141	.307	.084	-.055	.220	-.031
Post-Training	p	.327	.157	.507	.207	.212	.445	.000		.005	.015	.000	.640	.188	.533	.511	.145	.670	.781	.252	.872
Baseline S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	-.189	-.112	.212	-.200	-.206	.050	-.335	.502**	1	.090	-.013	.102	.011	-.115	.004	.093	.189	-.106	.192	-.189
Post-Training	p	.318	.555	.261	.288	.276	.793	.070	.005		.638	.944	.592	.953	.545	.985	.665	.334	.592	.318	.325
Baseline Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	-.244	-.206	.122	-.144	-.144	.007	.418*	.441*	.090	1	.831**	.114	.371*	-.239	.015	.249	-.133	.187	-.062	.146
Post-Training	p	.194	.274	.519	.449	.447	.970	.021	.015	.638		.000	.548	.044	.203	.944	.240	.500	.341	.750	.451
Post-Stress S1	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pz	r	-.008	.000	-.014	.076	.059	-.145	.659**	.604**	-.013	.831**	1	-.317	.426*	-.219	.021	.351	-.174	.051	.085	.227
Post-Training	p	.965	.999	.943	.688	.758	.444	.000	.000	.944	.000		.088	.019	.245	.923	.092	.376	.798	.662	.237
Post-Stress S2	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 10b. Pz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Pz	r	-.359	.018	-.171	-.052	-.217	.166	.096	.379	-.041	.432*	-.406	-.260	-.273	-.147
Pre-Training	p	.061	.927	.365	.786	.249	.381	.619	.051	.835	.019	.244	.469	.446	.686
Baseline S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	-.298	.118	-.173	.017	-.218	.206	.175	.424*	-.017	.437*	-.213	-.158	-.074	.052
Pre-Training	p	.123	.550	.361	.927	.248	.274	.363	.028	.931	.018	.555	.662	.839	.887
Baseline S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.125	.170	-.017	.171	-.025	.066	.093	-.002	.025	-.016	.367	.192	.427	.409
Pre-Training	p	.526	.386	.930	.365	.896	.730	.630	.992	.897	.932	.297	.596	.219	.240
Baseline Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	-.394*	-.035	-.194	-.125	-.195	.071	.015	.309	-.083	.347	-.398	-.382	-.282	-.399
Pre-Training	p	.038	.859	.303	.509	.302	.711	.940	.116	.670	.065	.254	.275	.430	.254
Post-Stress S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	-.363	-.023	-.209	-.092	-.171	.075	.078	.273	-.030	.266	-.451	-.416	-.156	-.231
Pre-Training	p	.057	.906	.267	.630	.368	.692	.689	.168	.877	.163	.190	.232	.668	.521
Post-Stress S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.293	.015	.072	.132	.161	-.047	.148	-.319	.152	-.439*	-.010	.021	.474	.602
Pre-Training	p	.131	.941	.704	.487	.397	.806	.443	.105	.432	.017	.979	.954	.166	.065
Post-Stress Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.172	.192	.222	-.076	.185	.092	.203	.094	-.074	.173	-.251	-.368	-.032	-.225
Post-Training	p	.381	.327	.239	.689	.328	.629	.291	.639	.702	.369	.485	.295	.930	.533
Baseline S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.236	.082	.235	-.118	.311	-.066	.197	.007	-.104	.169	.447	.445	-.025	-.168
Post-Training	p	.227	.678	.212	.534	.094	.728	.307	.971	.591	.380	.196	.198	.944	.642
Baseline S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.031	-.169	.000	-.068	.102	-.216	-.005	-.169	-.073	-.004	.646*	.767**	-.040	-.033
Post-Training	p	.874	.389	.999	.720	.593	.252	.979	.398	.708	.985	.044	.010	.912	.929
Baseline Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.242	.031	.190	-.014	.108	.027	.106	-.112	.179	.081	-.084	-.102	.382	.171
Post-Training	p	.214	.874	.314	.940	.570	.889	.582	.577	.353	.675	.817	.780	.276	.636
Post-Stress S1	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pz	r	.309	.220	.239	-.036	.212	.066	.186	.122	.209	.155	.127	.068	.302	.144
Post-Training	p	.110	.262	.204	.852	.260	.730	.335	.546	.277	.423	.726	.852	.396	.691
Post-Stress S2	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 10c. Pz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pz Pre-Training Baseline S1	Pz Pre-Training Baseline S2	Pz Pre-Training Baseline Ratio	Pz Pre-Training Post-Stress S1	Pz Pre-Training Post-Stress S2	Pz Pre-Training Post-Stress Ratio	Pz Post-Training Baseline S1	Pz Post-Training Baseline S2	Pz Post-Training Baseline Ratio	Pz Post-Training Post-Stress S1	Pz Post-Training Post-Stress S2	Pz Post-Training Post-Stress Ratio	Pre-Training McGill Pain Scale	Post-Training McGill Pain Scale	Pre-Training Pain Score	Pre-Training Stress Score	Post-Training Pain Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood
Pz	r	-.439*	-.335	.336	-.496**	-.408*	.483**	-.217	-.089	.102	.114	-.317	1	.035	.066	.076	-.013	.101	.371	-.187	-.150
Post-Training	p	.015	.070	.069	.005	.025	.007	.249	.640	.592	.548	.088		.854	.729	.722	.951	.608	.052	.331	.438
Post-Stress Ratio	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pre-Training	r	-.103	-.102	.092	-.092	-.109	-.032	.245	.247	.011	.371*	.426*	.035	1	.390*	.699**	.565**	.244	.387*	-.076	.084
McGill Pain Scale	p	.588	.591	.630	.629	.565	.867	.192	.188	.953	.044	.019	.854		.033	.000	.004	.211	.042	.694	.666
	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	.176	.214	.113	.089	.060	-.062	.022	-.118	-.115	-.239	-.219	.066	.390*	1	.564**	.056	.812**	.717**	.024	.122
McGill Pain Scale	p	.351	.257	.551	.639	.751	.745	.907	.533	.545	.203	.245	.729	.033		.004	.796	.000	.000	.901	.530
	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pre-Training	r	.120	.107	.012	.104	.054	-.186	.140	.141	.004	.015	.021	.076	.699**	.564**	1	.409*	.417*	.292	.224	-.117
Pain Score	p	.577	.617	.955	.627	.802	.384	.515	.511	.985	.944	.923	.722	.000	.004		.047	.048	.177	.292	.587
	N	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	23	23	24	24
Pre-Training	r	.004	-.001	-.009	-.050	.023	.276	.181	.307	.093	.249	.351	-.013	.565**	.056	.409*	1	.129	.212	-.006	.058
Stress Score	p	.984	.998	.967	.817	.917	.191	.396	.145	.665	.240	.092	.951	.004	.796	.047		.558	.331	.979	.788
	N	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	24	23	23	24	24
Post-Training	r	.072	.227	.386*	-.012	.001	.092	-.031	.084	.189	-.133	-.174	.101	.244	.812**	.417*	.129	1	.652**	.072	.090
Pain Score	p	.716	.245	.043	.953	.998	.643	.876	.670	.334	.500	.376	.608	.211	.000	.048	.558		.000	.720	.656
	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	23	23	28	28	27	27
Post-Training	r	-.133	-.060	.231	-.228	-.145	.403*	.088	-.055	-.106	.187	.051	.371	.387*	.717**	.292	.212	.652**	1	-.111	.085
Stress Score	p	.501	.760	.238	.244	.461	.033	.658	.781	.592	.341	.798	.052	.042	.000	.177	.331	.000		.580	.673
	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	23	23	28	28	27	27
Pre-Training	r	-.049	-.016	.016	-.084	-.131	-.077	.044	.220	.192	-.062	.085	-.187	-.076	.024	.224	-.006	.072	-.111	1	-.148
Baseline	p	.802	.936	.936	.666	.497	.693	.821	.252	.318	.750	.662	.331	.694	.901	.292	.979	.720	.580		.444
Positive Mood	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	29	29
Pre-Training	r	.243	.243	-.054	.165	.154	-.125	.156	-.031	-.189	.146	.227	-.150	.084	.122	-.117	.058	.090	.085	-.148	1
Baseline	p	.204	.204	.779	.393	.427	.518	.418	.872	.325	.451	.237	.438	.666	.530	.587	.788	.656	.673	.444	
Negative Mood	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	29	29
Pre-Training	r	-.359	-.298	.125	-.394*	-.363	.293	.172	.236	.031	.242	.309	.059	.037	.042	.088	.183	.103	.261	.668**	-.043
Post-Stress	p	.061	.123	.526	.038	.057	.131	.381	.227	.874	.214	.110	.765	.851	.833	.682	.393	.616	.198	.000	.829
Positive Mood	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	24	24	26	26	28	28
Pre-Training	r	.018	.118	.170	-.035	-.023	.015	.192	.082	-.169	.031	.220	-.192	-.190	-.396*	-.403	.011	-.144	-.307	.046	.252
Post-Stress	p	.927	.550	.386	.859	.906	.941	.327	.678	.389	.874	.262	.327	.333	.037	.051	.959	.484	.127	.815	.195
Negative Mood	N	28	28	28	28	28	28	28	28	28	28	28	28	28	28	24	24	26	26	28	28

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 10d. Pz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Pz	r	.059	-.192	.043	.072	-.002	.003	.045	-.411*	-.115	-.101	-.527	-.316	.055	.311
Post-Training	p	.765	.327	.822	.705	.993	.989	.816	.033	.551	.602	.117	.374	.880	.381
Post-Stress Ratio	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-Training	r	.037	-.190	.112	.056	.015	.226	-.185	-.119	-.170	.193	-.337	-.137	.091	.170
McGill Pain Scale	p	.851	.333	.557	.767	.937	.230	.336	.556	.379	.315	.341	.705	.802	.638
	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	.042	-.396*	.092	.077	.063	.049	.082	-.069	-.080	.148	-.093	.018	.164	.181
McGill Pain Scale	p	.833	.037	.629	.685	.742	.799	.671	.733	.680	.444	.798	.961	.651	.616
	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-Training	r	.088	-.403	.387	-.309	.154	.075	-.273	-.022	-.472*	.121	-.206	.101	.125	.017
Pain Score	p	.682	.051	.062	.142	.474	.727	.197	.920	.020	.581	.595	.797	.749	.966
	N	24	24	24	24	24	24	24	24	24	23	9	9	9	9
Pre-Training	r	.183	.011	.199	-.065	.172	.090	.101	.077	.041	.077	-.024	-.057	.357	.408
Stress Score	p	.393	.959	.351	.764	.421	.676	.638	.720	.848	.728	.950	.885	.346	.275
	N	24	24	24	24	24	24	24	24	24	23	9	9	9	9
Post-Training	r	.103	-.144	.122	.159	.166	.023	.241	-.011	-.030	.144	.046	.117	.559	.477
Pain Score	p	.616	.484	.537	.420	.398	.907	.226	.959	.883	.475	.899	.748	.093	.163
	N	26	26	28	28	28	28	27	25	27	27	10	10	10	10
Post-Training	r	.261	-.307	.145	.243	.122	.124	.207	-.370	.177	-.013	-.234	.010	.328	.383
Stress Score	p	.198	.127	.462	.213	.536	.528	.301	.068	.377	.947	.515	.978	.355	.274
	N	26	26	28	28	28	28	27	25	27	27	10	10	10	10
Pre-Training	r	.668**	.046	.693**	-.456*	.600**	-.372*	-.060	.131	-.367	-.341	.779**	.717*	-.184	-.286
Baseline	p	.000	.815	.000	.013	.001	.047	.761	.516	.055	.076	.008	.020	.611	.423
Positive Mood	N	28	28	29	29	29	29	28	27	28	28	10	10	10	10
Pre-Training	r	-.043	.252	-.232	.270	-.272	.216	.554**	.601**	.562**	.556**	-.544	-.515	.321	.463
Baseline	p	.829	.195	.226	.157	.153	.261	.002	.001	.002	.002	.104	.128	.366	.178
Negative Mood	N	28	28	29	29	29	29	28	27	28	28	10	10	10	10
Pre-Training	r	1	.133	.805**	-.125	.793**	-.147	.105	.010	-.003	-.265	.614	.469	.313	.166
Post-Stress	p		.501	.000	.527	.000	.456	.603	.962	.988	.182	.059	.171	.378	.646
Positive Mood	N	28	28	28	28	28	28	27	27	28	27	10	10	10	10
Pre-Training	r	.133	1	.075	.047	.036	.346	.236	.563**	.102	.233	.067	-.087	.131	.279
Post-Stress	p	.501		.705	.812	.857	.071	.236	.002	.606	.242	.854	.812	.718	.435
Negative Mood	N	28	28	28	28	28	28	27	27	28	27	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 10e. Pz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pz Pre-Training Baseline S1	Pz Pre-Training Baseline S2	Pz Pre-Training Baseline Ratio	Pz Pre-Training Post-Stress S1	Pz Pre-Training Post-Stress S2	Pz Pre-Training Post-Stress Ratio	Pz Post-Training Baseline S1	Pz Post-Training Baseline S2	Pz Post-Training Baseline Ratio	Pz Post-Training Post-Stress S1	Pz Post-Training Post-Stress S2	Pz Post-Training Post-Stress Ratio	Pre-Training McGill Pain Scale	Post-Training McGill Pain Scale	Pre-Training Pain Score	Pre-Training Stress Score	Post-Training Pain Score	Post-Training Stress Score	Pre-Training Baseline Positive Mood	Pre-Training Baseline Negative Mood
Post-Training	r	-.171	-.173	-.017	-.194	-.209	.072	.222	.235	.000	.190	.239	.043	.112	.092	.387	.199	.122	.145	.693**	-.232
Baseline	p	.365	.361	.930	.303	.267	.704	.239	.212	.999	.314	.204	.822	.557	.629	.062	.351	.537	.462	.000	.226
Positive Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	-.052	.017	.171	-.125	-.092	.132	-.076	-.118	-.068	-.014	-.036	.072	.056	.077	-.309	-.065	.159	.243	-.456*	.270
Baseline	p	.786	.927	.365	.509	.630	.487	.689	.534	.720	.940	.852	.705	.767	.685	.142	.764	.420	.213	.013	.157
Negative Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	-.217	-.218	-.025	-.195	-.171	.161	.185	.311	.102	.108	.212	-.002	.015	.063	.154	.172	.166	.122	.600**	-.272
Post-Stress	p	.249	.248	.896	.302	.368	.397	.328	.094	.593	.570	.260	.993	.937	.742	.474	.421	.398	.536	.001	.153
Positive Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Post-Training	r	.166	.206	.066	.071	.075	-.047	.092	-.066	-.216	.027	.066	.003	.226	.049	.075	.090	.023	.124	-.372*	.216
Post-Stress	p	.381	.274	.730	.711	.692	.806	.629	.728	.252	.889	.730	.989	.230	.799	.727	.676	.907	.528	.047	.261
Negative Mood	N	30	30	30	30	30	30	30	30	30	30	30	30	30	30	24	24	28	28	29	29
Pre-training	r	.096	.175	.093	.015	.078	.148	.203	.197	-.005	.106	.186	.045	-.185	.082	-.273	.101	.241	.207	-.060	.554**
Baseline SAI	p	.619	.363	.630	.940	.689	.443	.291	.307	.979	.582	.335	.816	.336	.671	.197	.638	.226	.301	.761	.002
	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	28	28
Pre-Training	r	.379	.424*	-.002	.309	.273	-.319	.094	.007	-.169	-.112	.122	-.411*	-.119	-.069	-.022	.077	-.011	-.370	.131	.601**
Post-Stress SAI	p	.051	.028	.992	.116	.168	.105	.639	.971	.398	.577	.546	.033	.556	.733	.920	.720	.959	.068	.516	.001
	N	27	27	27	27	27	27	27	27	27	27	27	27	27	27	24	24	25	25	27	27
Post-training	r	-.041	-.017	.025	-.083	-.030	.152	-.074	-.104	-.073	.179	.209	-.115	-.170	-.080	-.472*	.041	-.030	.177	-.367	.562**
Baseline SAI	p	.835	.931	.897	.670	.877	.432	.702	.591	.708	.353	.277	.551	.379	.680	.020	.848	.883	.377	.055	.002
	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	24	24	27	27	28	28
Post-Training	r	.432*	.437*	-.016	.347	.266	-.439*	.173	.169	-.004	.081	.155	-.101	.193	.148	.121	.077	.144	-.013	-.341	.556**
Post-Stress SAI	p	.019	.018	.932	.065	.163	.017	.369	.380	.985	.675	.423	.602	.315	.444	.581	.728	.475	.947	.076	.002
	N	29	29	29	29	29	29	29	29	29	29	29	29	29	29	23	23	27	27	28	28
Pre-Training	r	-.406	-.213	.367	-.398	-.451	-.010	-.251	.447	.646*	-.084	.127	-.527	-.337	-.093	-.206	-.024	.046	-.234	.779**	-.544
FMI Score	p	.244	.555	.297	.254	.190	.979	.485	.196	.044	.817	.726	.117	.341	.798	.595	.950	.899	.515	.008	.104
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10
Post-Training	r	-.260	-.158	.192	-.382	-.416	.021	-.368	.445	.767**	-.102	.068	-.316	-.137	.018	.101	-.057	.117	.010	.717*	-.515
FMI Score	p	.469	.662	.596	.275	.232	.954	.295	.198	.010	.780	.852	.374	.705	.961	.797	.885	.748	.978	.020	.128
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10
Pre-Training	r	-.273	-.074	.427	-.282	-.156	.474	-.032	-.025	-.040	.382	.302	.055	.091	.164	.125	.357	.559	.328	-.184	.321
MAAS Score	p	.446	.839	.219	.430	.668	.166	.930	.944	.912	.276	.396	.880	.802	.651	.749	.346	.093	.355	.611	.366
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10
Post-Training	r	-.147	.052	.409	-.399	-.231	.602	-.225	-.168	-.033	.171	.144	.311	.170	.181	.017	.408	.477	.383	-.286	.463
MAAS Score	p	.686	.887	.240	.254	.521	.065	.533	.642	.929	.636	.691	.381	.638	.616	.966	.275	.163	.274	.423	.178
	N	10	10	10	10	10	10	10	10	10	10	10	10	10	10	9	9	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

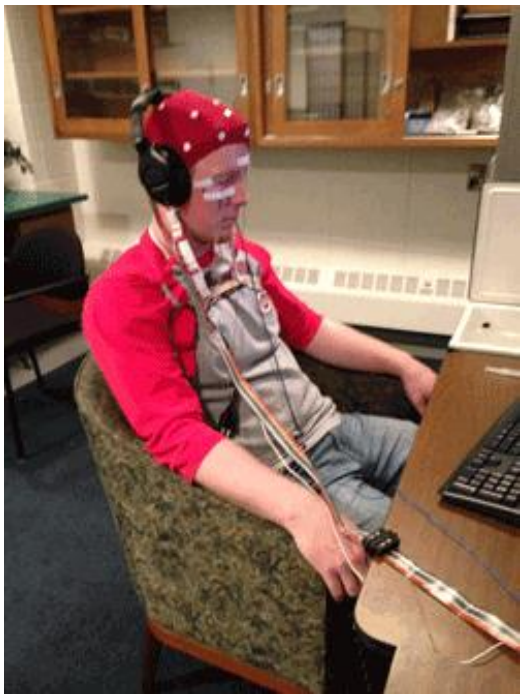
Table 10f. Pz Site T/C Ratio, S1 (C), S2 (T), Psychological Inventory Correlations

		Pre-Training Post-Stress Positive Mood	Pre-Training Post-Stress Negative Mood	Post-Training Baseline Positive Mood	Post-Training Baseline Negative Mood	Post-Training Post-Stress Positive Mood	Post-Training Post-Stress Negative Mood	Pre-Training Baseline SAI	Pre-Training Post-Stress SAI	Post-Training Baseline SAI	Post-Training Post-StressSAI	Pre-Training FMI Score	Post-Training FMI Score	Pre-Training MAAS Score	Post-Training MAAS Score
Post-Training	r	.805**	.075	1	-.407*	.873**	-.212	-.043	.000	-.356	-.114	.538	.526	.023	-.051
Baseline	p	.000	.705		.025	.000	.260	.824	.999	.058	.556	.109	.118	.951	.888
Positive Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	-.125	.047	-.407*	1	-.256	.653**	.357	.046	.646**	.274	-.105	.070	.392	.672*
Baseline	p	.527	.812	.025		.173	.000	.057	.821	.000	.150	.772	.848	.262	.033
Negative Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	.793**	.036	.873**	-.256	1	-.299	.138	-.027	-.142	-.190	.477	.373	.077	-.077
Post-Stress	p	.000	.857	.000	.173		.108	.475	.894	.463	.323	.163	.288	.832	.832
Positive Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Post-Training	r	-.147	.346	-.212	.653**	-.299	1	.170	.307	.340	.547**	-.087	-.040	.132	.429
Post-Stress	p	.456	.071	.260	.000	.108		.377	.119	.071	.002	.812	.913	.715	.217
Negative Mood	N	28	28	30	30	30	30	29	27	29	29	10	10	10	10
Pre-training	r	.105	.236	-.043	.357	.138	.170	1	.505**	.680**	.340	-.221	-.136	.385	.680*
Baseline SAI	p	.603	.236	.824	.057	.475	.377		.008	.000	.077	.569	.728	.306	.044
	N	27	27	29	29	29	29	29	26	28	28	9	9	9	9
Pre-Training	r	.010	.563**	.000	.046	-.027	.307	.505**	1	.313	.566**	-.228	-.263	.290	.531
Post-Stress SAI	p	.962	.002	.999	.821	.894	.119	.008		.112	.003	.526	.462	.416	.114
	N	27	27	27	27	27	27	26	27	27	26	10	10	10	10
Post-training	r	-.003	.102	-.356	.646**	-.142	.340	.680**	.313	1	.283	-.260	-.294	.603	.607
Baseline SAI	p	.988	.606	.058	.000	.463	.071	.000	.112		.144	.468	.410	.065	.063
	N	28	28	29	29	29	29	28	27	29	28	10	10	10	10
Post-Training	r	-.265	.233	-.114	.274	-.190	.547**	.340	.566**	.283	1	-.348	-.168	.079	.374
Post-Stress SAI	p	.182	.242	.556	.150	.323	.002	.077	.003	.144		.324	.643	.827	.287
	N	27	27	29	29	29	29	28	26	28	29	10	10	10	10
Pre-Training	r	.614	.067	.538	-.105	.477	-.087	-.221	-.228	-.260	-.348	1	.853**	-.215	-.295
FMI Score	p	.059	.854	.109	.772	.163	.812	.569	.526	.468	.324		.002	.551	.408
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Post-Training	r	.469	-.087	.526	.070	.373	-.040	-.136	-.263	-.294	-.168	.853**	1	-.345	-.226
FMI Score	p	.171	.812	.118	.848	.288	.913	.728	.462	.410	.643	.002		.328	.531
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Pre-Training	r	.313	.131	.023	.392	.077	.132	.385	.290	.603	.079	-.215	-.345	1	.845**
MAAS Score	p	.378	.718	.951	.262	.832	.715	.306	.416	.065	.827	.551	.328		.002
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10
Post-Training	r	.166	.279	-.051	.672*	-.077	.429	.680*	.531	.607	.374	-.295	-.226	.845**	1
MAAS Score	p	.646	.435	.888	.033	.832	.217	.044	.114	.063	.287	.408	.531	.002	
	N	10	10	10	10	10	10	9	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

APPENDIX L. ELECTROENCEPHALOGRAPHY SETUP



APPENDIX M. CONSENT FORM

BGSU®

BOWLING GREEN STATE UNIVERSITY

Department of Psychology

Title of experiment: Ameliorating Stress-Induced Inhibitory Gating Deficits
Principal investigator: Rachel Atchley, Graduate Student Department of Psychology, Bowling Green State University Email: atchler@bgsu.edu Phone: (419) 372-4375

We are conducting a research study to learn more about normal brain function. We are specifically interested in a brain process that involves information filtering. To participate in this study, you must meet the qualifying criteria: 1) 18 or older; 2) Not currently diagnosed with a mental illness; 3) Never diagnosed with a neurological disease; 4) Not currently receiving treatment for a medical condition; 5) No circulatory problems; 6) No hearing problems; 7) Willing to abstain from alcohol for 24 hours before your appointments; 8) Willing to abstain from non-prescription drugs for 24 hours before your appointments; 9) Willing to abstain from nicotine for 30 minutes before your appointments; 10) Able to remove any metal jewelry worn in the ears or on the face.
If you agree to participate, the following will happen:
You will attend 4 appointments over 4 days. For Appointments 1 and 4, you will wear an electrode cap and three electrodes will be placed around your forehead and right eye. Two electrodes will be clipped onto your earlobes. The electrodes will be filled with an easily washable gel. You'll wear headphones, listen to clicks, and keep your eyes closed for approximately 7 minutes. Next, you will be asked to place one hand in cold water for up to 2 minutes. You will then listen to clicks through headphones for another 7 minutes. The cap and electrodes will be removed. You will then be guided through a relaxation training session. At the end of the experiment, you'll be given questionnaires on your mood, stress level, and experiences with the tasks described above.
In the next two days, you'd come in for two more relaxation training sessions (Appointments 2 and 3). These appointments are much shorter and only last about 15 minutes. Please note that you need to attend all of your appointments to get full credit.
You will earn experimental course credit or cash for your participation in this project. You'll receive 1 SONA credit for every half hour of participation or $5 for attending each appointment. Appointments 1 and 4 should last approximately 1 hour and 15 minutes, and Appointments 2 and 3 should last 15 minutes. In total, the experiment will take approximately 3 hours to complete over 4 sessions. If you withdraw before the experiment is complete, you will receive SONA credit equivalent to however long you participated or $5 for attending your appointment.
Information about your participation will be held in the strictest confidence. Records other than this form will have a number rather than your name on them. Only the experimenter and HSRB-approved research assistants will have access to your data. All records and data will be stored in locked cabinets and password-protected computers within locked laboratories.

BGSU HSRB - APPROVED FOR USE
IRBNet ID # _414801
EFFECTIVE __04/17/2013
EXPIRES __02/10/2014

Participation in this research is entirely voluntary. You may refuse to participate or withdraw at any time without penalty. Your grades, class standing, and relationship with the university will not be affected should you choose to participate, refuse to participate, or withdraw from the experiment. We will end the experiment early without your consent only if there is an equipment failure or unforeseen technical problem. If we have to end the experiment, you will still receive credit for the amount of time you participated.

The risks of participation are no greater than those experienced in daily life. The cold water task may be slightly uncomfortable, but you are free to stop the task at any time. An experimenter will always be present to address any concerns you may have. There are no direct benefits to you for participating in this experiment, although some people find the procedures interesting. We hope that this project will help us understand more about how the human brain processes auditory information. If you have any questions, please ask them before signing this form. If you would like more information in the future, please contact Rachel Atchley using the phone number or email address at the top of this form. You may also contact the project advisor, Dr. Casey Cromwell, at (419) 372-9408 or hcc@bgsu.edu. If you have questions regarding the conduct of the study or concerns regarding your rights as a research participant, you may contact the chair of the Human Subjects Review Board at Bowling Green State University at (419) 372-7716 or hsrb@bgsu.edu.

Please save this form for your records.