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The Relationship Between Self-Reported Measures of Anxiety and Sensory Processing

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**THE RELATIONSHIP BETWEEN SELF-REPORTED MEASURES OF ANXIETY AND
SENSORY PROCESSING**

A Master's Thesis

Presented to

The Graduate College of
Missouri State University

In Partial Fulfillment

Of the Requirements for the Degree
Master of Science, Clinical Psychology

By

Elizabeth Rose Troutwine

July 2021

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THE RELATIONSHIP BETWEEN SELF-REPORTED MEASURES OF ANXIETY AND SENSORY PROCESSING

Psychology

Missouri State University, July 2021

Master of Science

Elizabeth Rose Troutwine

ABSTRACT

Auditory sensory gating, a type of sensory processing, is a physiological mechanism that allows the brain to filter out and respond less to redundant sensory information. Poor sensory gating has been found in clinical groups such as Alzheimer's dementia (Jessen et al., 2001), bipolar I disorder (Lijffijt et al., 2009), schizophrenia (Patterson et al., 2008), and other anxiety-related psychopathologies such as panic disorder (Ghisolfi et al., 2006), post-traumatic stress disorder (PTSD) (Orr et al., 2002), and obsessive-compulsive-disorder (OCD) (Hashimoto, 2007). Research is limited regarding effects of chronic worry and anxiety on sensory gating ability. This study will explore the relationship between anxiety and self-reported sensory gating ability. Questionnaires were administered in the form of a confidential online survey accessed through the SONA System, linked to Qualtrics. Participants completed the Sensory Gating Inventory (SGI), Highly Sensitive Person Scale (HSPS), Adult Sensory Processing Scale (ASPS), Beck Anxiety Inventory (BAI), Penn State Worry Questionnaire (PSWQ), State-Trait Anxiety Inventory Y2 Form (STAI-Y2), General Anxiety Disorder Scale (GAD7), along with a demographic questionnaire. This exploratory study provided evidence that anxiety and sensory processing are closely related. Results suggest individuals who report more sensory processing difficulties also experience more symptoms of anxiety.

KEYWORDS: sensory processing, sensory gating, electroencephalography, anxiety, self-report, Sensory Gating Inventory, Highly Sensitive Person Scale, Adult Sensory Processing Scale

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In the interest of academic freedom and the principle of free speech, approval of this thesis indicates the format is acceptable and meets the academic criteria for the discipline as determined by the faculty that constitute the thesis committee. The content and views expressed in this thesis are those of the student-scholar and are not endorsed by Missouri State University, its Graduate College, or its employees.

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INTRODUCTION

The mechanism underlying sensory gating and how sensory gating influences behavioral outcomes or psychopathology is not fully understood. Poor sensory gating has been found in clinical groups such as Alzheimer's dementia (Jessen et al., 2001), bipolar I disorder (Lijffijt et al., 2009), schizophrenia (Patterson et al., 2008), and other anxiety-related psychopathologies such as panic disorder (Ghisolfi et al., 2006), post-traumatic stress disorder (PTSD) (Orr et al., 2002), and obsessive-compulsive-disorder (OCD) (Hashimoto, 2007). Although there is a limited amount of literature on the relationship between some anxiety disorders and sensory gating, no known research has been conducted looking at the effects of chronic worry and anxiety on sensory gating ability. Sensory gating is typically measured in an event-related potential or ERP paradigm using electroencephalography. Alternative tools to measure neural-level sensory processing exist, but most rely on self-report questionnaires such as the Sensory Gating Inventory (SGI). It is unknown whether the SGI measures the same physiological construct as event related potentials. This study will explore the relationship between anxiety and self-reported sensory processing utilizing alternative measurements, like the SGI.

LITERATURE REVIEW

Auditory Sensory Gating

Auditory sensory gating, a type of sensory processing, is a physiological mechanism that allows the brain to filter out and respond less to redundant sensory information. For instance, we hear the humming of the air conditioner when it turns on. Over time, our brain responds less to the humming until eventually we do not attend to the sound. Sensory gating is thought of as a protective mechanism because it is essential to have the ability to quickly respond to new information and ignore repetitive information (Yadon, 2010). Researchers theorize that sensory gating allows for more cognitive resources to be allocated to new and important stimuli and fewer resources given to redundant stimuli. Research also suggests that poor sensory gating may lead to cognitive overload and diminished cognitive functioning (Lijffijt et al., 2009). There are two different types of sensory gating: gating in and gating out. Gating in is characterized by the brain's ability to favor novel stimuli and is most associated with the orienting response. Gating out consists of filtering out unimportant or repetitive stimuli (Yadon, 2010). Filtering is the most studied sensory gating mechanism; therefore, this thesis will only measure filtering using a classic sensory gating paradigm and will not include orienting.

Sensory gating ability is typically measured using electroencephalography. An electroencephalogram (EEG) is an electrophysiological tool that measures the electrical activity produced in the brain, colloquially termed brainwaves. This activity can be detected by metal electrodes and conductive media applied on the scalp (Teplan, 2002). Electrodes are also placed on the face to monitor artifacts that might interfere with EEG readings such as eye blinks and

electrical activity produced by facial muscles. EEGs are noninvasive and can be applied repeatedly with little to no known risk.

An EEG can be used to record the brain's response to specific sensory stimuli with millisecond precision. These neural responses are associated with a specific stimulus onset and are termed event-related potentials (ERPs). Luck (2005, p. 4) defined ERPs as "neural responses associated with specific sensory, cognitive, and motor events." Teplan (2002, p. 4) also described an ERP as a "significant positive or negative voltage fluctuations resulting from evoked neural activity." These voltage fluctuations produce predictable peaks and valleys called ERP components, which demonstrate the patterns of evoked neural activity. ERPs are thought to originate from postsynaptic potentials, which are produced when neurotransmitters bind to receptors resulting in a change in ion flow across the cell membrane (Luck, 2005). It is rare to present data from a single participant (Luck, 2005); therefore, ERP wave forms are created by averaging together the averaged waveforms of individual participants.

ERPs are described in terms of latency and amplitude. When plotting ERPs, the y-axis represents amplitude, or the strength of the brain's response, measured in microvolts (μV). The x-axis represents latency, or how quickly the brain responded to the stimuli, measured in milliseconds (ms). ERP components are named based on their positive or negative voltage fluctuation and their approximate latency in which they appear. For instance, a P300 component is a positive voltage fluctuation at approximately 300 ms after the stimulus onset.

Sensory gating ability is elicited using a paired-stimulus paradigm. In the classic paradigm, a brief auditory stimulus (click or short tone) is presented and then quickly followed by the identical stimulus. Several of these identical tone pairs are presented to participants throughout an experiment. A positive ERP component appears approximately 50 milliseconds

post-stimulus, a negative ERP component appears approximately 100 milliseconds post-stimulus, and another positive ERP component appears approximately 200 milliseconds post-stimulus (P50, N100, and P200, respectively) (Luck, 2005). These three components (P50, N100, and P200) are associated with auditory sensory gating and are considered mid-latency components and are believed to reflect different stages in sensory gating (Boutros et al., 2013). The P50 is the most common component used to measure sensory gating; however, researchers often report data from the N100 and P200 components in addition to the P50.

In a classic sensory gating paradigm, the amplitude in response to the second stimulus is usually less than the amplitude of the first stimulus. This reduction in amplitude to the second stimulus is termed P50 suppression or gating and reflects the gating out response. Sensory gating is typically reported as an amplitude ratio (Yadon & Daugherty, 2018), although a difference score is sometimes used (Smith et al., 1994). The amplitude associated with the second stimulus (S2) or “test” stimulus (T) is divided by the first amplitude (S1), or “conditioning” stimulus (C) ($\text{gating} = T/C$). This creates a T/C ratio where lower sensory gating ratios reflect higher sensory gating ability and higher T/C ratios reflect lower sensory gating ability.

In addition to neurophysiological tools, such as EEGs, alternative methods designed to capture neural-level sensory processing exist; however, most rely on self-report questionnaires. One such measure is the Sensory Gating Inventory (Hetrick et al., 2012). The Sensory Gating Inventory (SGI) is a self-report questionnaire designed to measure the experiential aspects of sensory gating traditionally measured by ERPs. Other self-report measures such as the Highly Sensitive Persons Scale (HSPS) and the Adult Sensory Processing Scale, aim to capture similar types of sensitivity. The HSPS is a 27-item designed to assess sensory processing sensitivity (SPS). Research suggests individuals who have higher SPS tend to have an increased awareness

of subtleties the environment, are easily overstimulated, have a stronger emotional response and possess an increased level of empathy (Acevedo et al., 2018). Increased SPS has been found in clinical groups such as autism spectrum disorders, particularly in children, post-traumatic stress disorder and schizophrenia (Acevedo et al., 2018).

The HSPS has been correlated with the SGI, as items on the HSPS seem to capture a similar construct as the SGI. Hetrick and colleagues compared the SGI and HSPS to establish convergent validity of SGI. They found a moderately positive correlation between the SGI and the HSPS, $r(219) = .65, p < .001$ (Hetrick et al., 2012). Aron and Aron (1997) also conducted a study examining the relationship between sensory processing and its relation to introversion and emotionality. This study established adequate reliability and content, convergent, and discriminant validity for the HSPS. It also offered connectivity between Introversion and sensory sensitivity. Although there is research connecting the HSPS to personality traits, there is minimal research regarding examining the relationship between the HSPS and anxiety.

The mechanism underlying sensory gating and how sensory gating influences behavioral outcomes or psychopathology is not fully understood. Neurotypical individuals typically display P50 suppression; however, it is not uncommon for neurotypical individuals to have poor sensory gating. Interestingly, 40% of neurotypical individuals have sensory gating ratios within one standard deviation of individuals with schizophrenia (Patterson et al., 2008). In addition, research has found reduced sensory gating ability is not always an unfavorable quality. For example, professional musicians have exhibited reduced sensory gating ability (Kizkin et al., 2006). In this study, sensory gating was compared between 34 professional musicians and 19 non-musicians. Results suggest musicians exhibited less P50 suppression compared to non-musicians.

Furthermore, higher levels of creativity have also been associated with reduced sensory gating (Zabelina et al., 2015).

Although a few studies have linked poor sensory gating to positive outcomes (those noted above), it is often associated with lower performance on measures of cognitive functioning (Truelove-Hill & Yadon, 2015). Poor sensory gating has been found in many clinical groups such as Alzheimer's dementia (Jessen et al., 2001), bipolar I disorder (Lijffijt et al., 2009), schizophrenia (Patterson et al., 2008), and other anxiety-related psychopathologies such as panic disorder (Ghisolfi et al., 2006), post-traumatic stress disorder (PTSD) (Orr et al., 2002), and obsessive-compulsive-disorder (OCD) (Hashimoto, 2007).

Yee and colleagues (1998) compared P50 suppression of 22 recent-onset schizophrenia patients to 11 normal controls. Results suggest that abnormal P50 suppression is present during the early stages of schizophrenia. Furthermore, P50 suppression covaried with clinical ratings of depression and anxiety and a relationship may exist between the degree of P50 suppression and the patient's attentional impairment. Potter and colleagues (2006) published a review article summarizing P50 suppression in individuals with schizophrenia. They established P50 abnormalities are present in a large portion of individuals with schizophrenia.

An fMRI study conducted by Acevedo and colleagues (2018) compared patterns of activation and deactivation for SPS, autism spectrum disorders, schizophrenia, and post-traumatic stress studies. The authors reported activation in the neural structures found with reward processing, physiological homeostasis and pain control, awareness, and self-control. Interestingly, individuals with schizophrenia displayed deactivation in the caudate, thalamus, amygdala, cingulate/anterior cingulate cortex, superior frontal gyrus, precuneus, medial temporal gyrus and super temporal lobe/gyrus while the other groups displayed activation.

A study conducted by Ghisolfi and colleagues (2006) compared 28 patients diagnosed with panic disorder to 28 healthy individuals. Subjects were presented with a double-click paradigm as a measure of sensory gating. Results demonstrated patients diagnosed with panic disorder displayed higher P50 ratios, or weaker gating ability, compared to healthy individuals. Individuals with panic disorder displayed larger N100 amplitudes for non-target tones when given a standard two-tone discrimination task (oddball task). This suggests an abnormality in early information processing. Furthermore, this study compared 28 control participants and 28 participants with schizophrenia to 28 participants with panic disorder. Results of this comparison suggested participants with panic disorder displayed weaker sensory gating compared to normal participants and exhibited a higher amplitude in response to the second stimulus (S2). Moreover, participants with schizophrenia displayed higher P50 ratios (weaker gating) compared to healthy controls and higher S2 amplitude. When compared to subjects with schizophrenia, there was no statistical difference compared to participants with panic disorder.

In addition to panic disorder, sensory gating abnormalities have been found in other anxiety-related psychopathologies. Hashimoto and colleagues (2007) compared the sensory gating ability of 26 participants diagnosed with OCD to 26 healthy controls. Results demonstrated higher P50 T/C ratios in participants diagnosed with OCD compared to healthy controls. Another study conducted by Orr and colleagues (2002) compared sensory gating abilities of male Vietnam combat veterans and female rape victims with PTSD to individuals without PTSD. Results showed that individuals diagnosed with PTSD did not display the typical reduction in the P50 response to the second stimulus. Furthermore, Gillette and colleagues (1997) discovered that weaker P50 suppression in male Vietnam combat veterans with PTSD was strongly correlated with higher intensity PTSD re-experiencing symptoms.

Fathy and colleagues (2015) examined the P50, N100, and P200 components in individuals with bipolar disorder with a comorbid anxiety disorder. This study compared P50 suppression in patients with a bipolar disorder and an anxiety disorder, patients with a bipolar disorder without an anxiety disorder, and healthy controls. The sample was divided into three groups: 30 patients with bipolar I disorder and comorbid anxiety, 30 patients with bipolar I disorder with no other comorbid (Axis I) diagnosis, and 30 control individuals. Results of this study revealed patients with bipolar disorder with comorbid anxiety demonstrated higher suppression ratios in P50, N100, and P200 than bipolar patients without comorbid anxiety. Furthermore, bipolar patients with comorbid anxiety showed higher suppression ratios compared to the control group. This study suggests that anxiety has a negative effect on sensory gating in bipolar disorder patients.

Anxiety and Stress

Although there has been research on different types of anxiety disorders, such as panic disorder and PTSD, there is little to no known research regarding the sensory gating ability of individuals who endorse symptoms of stress and anxiety, characterized by generalized anxiety disorder (GAD). According to the American Psychiatric Association's Diagnostic Statistical Manual of Mental Disorders, 5th Edition (DSM-5), anxiety disorders are characterized by excessive fear and anxiety and are related to behavioral disturbances. Fear is defined as the emotional response to a real or perceived imminent threat, while anxiety is defined as the anticipation of future threats. Fear and anxiety are often co-occurring; however, some anxiety disorders are differentiated based on the types of objects or situations that induce fear, anxiety, or avoidant behavior, and the associated ideation (DSM-5).

The DSM-5 defines GAD as excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least six months, about a number of events or activities (such as school or work performance). Six symptoms encompass this disorder: restlessness, being easily fatigued, difficulty concentrating, irritability, muscle tension, and sleep disturbance. Individuals must display three of these symptoms, and some symptoms must be present for more days than not for the past six months to be diagnosed with GAD. The diagnostic feature of GAD is the inability to control worry and to keep worrisome thoughts from interfering with the attention to tasks at hand. The DSM-5 further discusses the functional consequences of GAD. Many individuals who are diagnosed with GAD report that excessive worry impairs their ability to do things quickly and efficiently.

Symptoms of GAD occur mostly internally and lack overt behaviors (Mennin et al., 2009). Research suggests individuals with anxiety disorders have overactivity in the amygdala and underactivity in the prefrontal cortex (Carlson, 2014). Studies have shown that the prefrontal cortex contributes to the emotional reactions by inhibiting the activity of the amygdala (Kim et al., 2011). Furthermore, a study conducted by Kim and colleagues (2011) suggests an increase in connections between the amygdala and the prefrontal cortex is related to more beneficial outcomes regarding emotion regulation and anxiety.

The prefrontal cortex is the part of the brain necessary for higher-order or executive functioning. Executive functioning is defined as, “higher level cognitive processes of planning, decision making, problem solving, action sequencing, task assignment and organization, effortful and persistent goal pursuit, inhibition of competing impulses, flexibility in goal selection, and goal-conflict resolution” (American Psychological Association, 2020). Poor executive

functioning can result in difficulties with daily tasks such as organization, problem-solving, and time management.

The prefrontal cortex has also been associated as one of the important structures for the sensory gating mechanism and higher-order functioning (Funahashi & Andreau, 2013). Research suggests executive function performance is correlated with sensory gating measures. Better sensory gating was associated with better performance on tasks measuring executive functioning (Truelove-Hill & Yadon, 2015). Furthermore, Lijffijt and colleagues (2009) examined P50, N100, and P200 components in relation to behavioral inhibition, attention, and working memory. The study found P50, N100, and P200 components were correlated with fewer commission errors and slower reaction times on Delayed Memory Task (DMT), correction detections, and better discriminability on the Immediate Memory Task (IMT), respectively.

The amygdala is said to be the key area of the brain that regulates stress response, which in turn, affects stress-induced psychopathologies (Andolina & Borreca, 2017). Interestingly, there is a relationship between stress and sensory gating. Anxiety evoked by a novel situation can temporarily reduce sensory gating abilities (Orr et al., 2002). Research has also shown that a stressful stimulus can decrease sensory gating ability. For example, Johnson and Adler (1993) demonstrated that sensory gating can be temporarily reduced through an acute stressor using a cold pressor test that caused transit distress and pain accompanied by increased sympathetic activity. In this study, 10 participants immersed their hands in an ice water bath for two minutes. Prior to the cold pressor test, participants completed an EEG to establish a baseline. The second EEG recording began immediately after the cold pressor test. The baseline EEG was then compared to the EEG recording after the cold pressor test. Results suggested the

cold pressor task decreased the sensory gating ability in nine of the participants, suggesting an acute stressor can impair P50 ability.

A study conducted by White and Yee (1997) explored the role of psychological stress on P50 suppression. Participants were asked to complete two mental arithmetic tasks designed to maintain the same level of difficulty across tasks while changing the level of stress or anxiety. The first task was a silent mental arithmetic task, which has previously shown to have little to no effect on gating and produce only small effects on self-reported anxiety. The second task consisted of mental arithmetic performed aloud. This task has previously shown to be an effective acute psychological stressor. Results of this study indicate the impact of stress and anxiety on P50 amplitude and P50 suppression ratio can be significant depending on the psychological stressor. The second task, oral mental arithmetic, had a significant impact on P50 gating and self-report anxiety while the first task, a silent mental arithmetic task, had little to no effect on gating.

Present Study

Although there is a limited amount of literature on acute stressors, no known research has been conducted looking at the effects of chronic worry and anxiety on sensory gating ability. Anxiety disorders are the most widespread mental illness in the United States affecting almost 24.8 million individuals, with GAD being one of the most common mental disorders (Spitzer et al., 2006). This study will explore the relationship between anxiety and self-reported sensory processing utilizing alternative tools, like the SGI. A better understanding of the sensory gating mechanism will help us understand sensory gating in clinical groups and may improve our ability to design effective interventions and treatments for individuals affected by anxiety.

Treatments for anxiety exist, such as psychotherapies (i.e., Cognitive Behavioral Therapy and Acceptance and Commitment Therapy) and medication; however, elucidating the connectivity between sensory gating and anxiety may open a new avenue of treatment modalities specifically for individuals experiencing anxiety and sensory gating difficulties. Furthermore, it is possible that self-reported sensory processing assessments may assist with anxiety screenings and diagnosis in the future. Based on past research regarding the connectivity of poor sensory gating and anxiety-related psychopathologies, we propose that individuals who endorse more symptoms of anxiety will also score higher on measures of sensory processing, indicating weaker sensory processing. Furthermore, we propose that individuals who endorsed a diagnosis of GAD will endorse more difficulties with sensory processing compared to individuals who do not endorse a diagnosis of GAD.

METHODS

Participants

A total of 144 participants were recruited from Introductory Psychology courses at Missouri State University (ages 18-38, $M = 19.8$, $SD = 2.8$; females = 71, males = 66, non-binary/third gender = 1, prefer to self-describe = 5). Participants were given the option to utilize a text box when describing their gender. Four participants selected '*prefer to self-describe*' but declined to provide a text response. All participants were screened for abnormal hearing, a diagnosis or history of GAD, a history of traumatic brain injury or concussion (Yadon et al., 2015), a history of a serious neurological condition or bipolar disorder (Lijffijt, 2009), schizophrenia or first-degree relative with schizophrenia (Clementz et al., 1998), and any psychoactive drug use that has been found to affect P50 gating (Yadon et al., 2015). We excluded individuals who reported having seizures ($n = 1$), a concussion, or other head injuries from data analysis ($n = 15$) (Arciniegas et al., 2000). The final sample included a total of 128 participants (ages 18-38, $M = 19.8$, $SD = 3.0$; females = 60, males = 61, non-binary/third gender = 1, prefer to self-describe = 5). Although this study is a behavioral study, the questionnaires utilized are designed to measure the same physiological construct. It was decided to leave the exclusion criteria similar to studies using EEG as the criteria listed above affects physiological gating ability and thus might also affect self-reported sensory processing.

This study contained a demographic questionnaire that screened for a diagnosis of GAD. We provided a text box which stated, "Please explain, in as much detail as you feel comfortable, your present/past diagnosis of GAD 9 (e.g., who diagnosed you (primary care physician, counselor, therapist, etc.), when you were diagnosed, what age you were diagnosed, etc.)". Our

screening found 18.75% of our sample endorsed a diagnosis of GAD. A total of 24 participants endorsed a diagnosis of GAD given by a clinical provider (primary care physician = 17; psychiatrist = 1; therapist = 6). Ten participants reported their diagnosis being provided during the COVID pandemic (between 2020 and 2021). It is important to note that the percent of individuals who reported a diagnosis in our sample is unusually high considering the 12-month prevalence of GAD is 0.9% among adolescences and 2.9% among adults in the general population of the United States (DSM-5).

Measures

Generalized Anxiety Disorder 7- item (GAD-7) Scale. The GAD-7 is a 7-item self-report scale designed to assess the symptoms of GAD by asking participants to indicate how much they have been “bothered” by the symptom over the last two weeks (Spitzer et al., 2006). Items are rated on a 4-point Likert scale (0 = not at all sure; 1 = several days; 2 = over half the days; 3 = nearly every day). The amount of time to complete the GAD-7 is approximately five to ten minutes. Factors screened for include feeling nervous, anxious, or on edge, not being able to stop or control worrying, worrying too much about different things, trouble relaxing, being so restless it is hard to sit still, becoming easily annoyed or irritated, and feeling as if something awful might happen. The total score is calculated by adding the 4 column scores and can range from 0-21 with higher scores indicating more severe symptoms of GAD. The scoring is divided into four categories: minimal anxiety (0-4), mild anxiety (5-9), moderate anxiety (10-14), severe anxiety (15-21). The GAD-7 has been established as a reliable and valid screening tool used in clinical practice and research for assessing severity (Löwe et al., 2008) Furthermore, this assessment contains a high agreement between self-report measures and clinician administration.

Sample items include “Feeling afraid as if something awful might happen” and “Being so restless that it's hard to sit still”.

Beck Anxiety Inventory (BAI). The BAI is a 21-item self-report inventory aimed at measuring the severity of the symptoms of anxiety by asking the participants to indicate how much they have been “bothered” by the symptom during the past month, including today (Beck et al., 1998). Items are rated on a scale ranging from zero to three (0 = Not at all; 1 = mildly, but it didn’t bother me much; 2 = moderately- it wasn’t pleasant at the time; 3 = severely-it bothered me a lot). The amount of time to complete the BAI is approximately five to ten minutes. The total score is calculated by adding the 21 responses. A score between 0-7 indicates minimal levels of anxiety, 8-15 mild levels, 16-25 moderate levels, and 26-63 severe levels of anxiety. The BAI has shown high internal consistency and test-retest reliability (Beck et al., 1988). Furthermore, the BAI can discriminate between anxious clinical groups (GAD, panic disorder, etc.) from non-anxious clinical groups (major depression, dysthymic disorder, etc.). Sample items consist of “Numbness or tingling” and “Dizzy or lightheaded”.

Penn State Worry Questionnaire (PSWQ). The Penn State Worry Questionnaire (PSWQ) is a self-report measure designed to address the trait of worry characterized by symptoms of GAD. A study conducted by Meyer and colleagues (1990) found the PSWQ assesses anxiety as an independent construct, discriminating between depressive symptoms. Furthermore, researchers found the PSWQ significantly discriminated between individuals who met all, some, or none of the DSM-III-R diagnostic criteria for GAD and individuals who met criteria for GAD versus posttraumatic stress disorder. The PSWQ contains 16 items placed on a 5-point Likert scale ranging from 1 (“not at all typical of me”) to 5 (“very typical of me”). The PSW Q has shown high validity, internal consistency, and reliability (Meyer et al. 1990). The

development of the PSWQ utilized 1,580 college students, among them discriminated samples who met all, some or DSM-5 criteria and samples who met criteria for PTSD. The assessment successfully distinguished levels of diagnosable GAD and produced higher PSWQ scores compared to other PTSD cases (Meyer et al. 1990). Sample items include “If I do not have enough time to do everything, I do not worry about it” and “My worries overwhelm me”.

State-Trait Anxiety Inventory Form Y-2 (STAI-Y2). The STAI Form Y-2 (trait anxiety subscale) is an assessment used to measure the presence and severity of trait anxiety in adults by asking participants to indicate how they generally feel in response to a statement (Spielberger et al., 1983). Items are rated on a 4-point Likert scale (1 = almost never; 2 = sometimes; 3 = often; 4 = almost always). The STAI Form Y-2 consists of 20 questions with higher scores indicating higher anxiety. The amount of time to complete the STAI is approximately ten minutes. The trait anxiety subscale measures aspects of anxiety proneness while the state anxiety subscale measures the respondent’s current state of anxiety (Julian, 2011). According to the American Psychological Association (APA), the STAI can be used in clinical settings to diagnose anxiety and differentiate anxiety from depressive symptoms. The STAI has shown high internal consistency with coefficients ranging from .86 to .95 (Spielberger et al., 1983). STAI Form Y-2 sample items are as follows, “I feel nervous and restless” and “I am happy”.

Sensory Gating Inventory (SGI). The Sensory Gating Inventory (SGI) is a 36-item self-report assessment designed by Hetrick et al. (2012) designed to measure the dimensions of sensory gating from a phenomenological standpoint. Items are rated on a Likert-scale ranging from 0 = never true to 5 = always true. The scale is divided into five categories which include the SGI total score and four dimensions: Perceptual Modulation (PM), Distractibility, Over-inclusion

(OI), or hyper attention, and Fatigue & Stress Vulnerability (FSV). PM can be defined as increased sensory sensitivity and sensory overload while Distractibility refers to difficulties focusing attention. FSV is characterized by vulnerability to perceptual abnormalities during periods of fatigue and stress (Hetrick et al., 2012). The SGI has been shown to contain strong reliability and validity (Hetrick et al., 2012). Sample items include, “I have feelings of being flooded by visual experiences, sights, or colors” and “I hear sounds but I can’t make sense of them all because it’s like trying to do 2 or 3 things at once”

Highly Sensitive Person Scale (HSPS). The Highly Sensitive Person Scale (HSPS) is a 27-item designed to assess sensory processing sensitivity (SPS). The items presented contain both internal and external stimuli to successfully measure an individual’s SPS. Items are rated on a 7-point Likert scale, ranging from "not at all" to "extremely" (Aron & Aron, 1997). HSPS scores can range from 27 to 189 with higher scores indicating higher sensitivity to stimuli. Sample items include, “*Do other people's moods affect you?*”, “*Do you startle easily?*”, and “*Does being very hungry create a strong reaction in you, disrupting your concentration or mood?*” (Aron & Aron, 1997).

The Adult Sensory Processing Scale (ASPS). Adult Sensory Processing Scale (ASPS) is a self-report measure aimed at capturing challenges in sensory responsiveness on a behavioral level within sensory systems (i.e., auditory, visual, tactile, vestibular, and proprioceptive) Items are rated on a 5-point Likert scale, ranging from “*never*” to “*always*” (Blanche et al., 2014). ASPS scores are calculated in terms of patterns. Patterns refer to categories of stimuli (auditory, visual, etc.). The scale is divided into 11 patterns which include: Overresponsiveness to Vestibular Input, Overresponsiveness to Auditory Input, Overresponsiveness to Visual Input, Overresponsiveness to Tactile, Proprioceptive Seeking, Overall Underresponsiveness,

Underresponsiveness to Proprioceptive–Vestibular Input Affecting Postural–Motor Abilities, Underresponsiveness to Auditory Input, Underresponsiveness to Tactile, Overresponsiveness to Vestibular Input, and Overresponsiveness to Tactile. The ASPS has been found to contain adequate internal consistency, strong concurrent validity, and acceptable construct validity (Blanche et al., 2014). The sum of the pattern score is then compared to three number ranges categorized by “Typical range”, “Possible Difficulties”, and “Definite Difficulties” (Blanche et al., 2014). Sample items include, “*I tend to enjoy music at a higher than average volume*”, “*When I am stressed, I like to engage in activities like running, walking, or rock climbing to calm me down*”, and “*I become more irritated by certain sound due to lack of sleep*” (Blanche et al., 2014).

Procedure

All methodological procedures and protocols were approved by the Missouri State University Institutional Review Board (IRB-FY2020-486, Approval Date: March 31, 2021; See Appendix A). This study was administered in the form of a confidential online survey accessed through the SONA System, linked to Qualtrics. After providing informed consent (Appendix B), participants completed the Sensory Gating Inventory (SGI), Highly Sensitive Person Scale (HSPS), Adult Sensory Processing Scale (ASPS), Beck Anxiety Inventory (BAI), Penn State Worry Questionnaire (PSWQ), State-Trait Anxiety Inventory Y2 Form (STAI-Y2), and General Anxiety Disorder Scale (GAD7), along with a demographic questionnaire (Appendix C). To prevent order effects, questionnaires were presented in random order with the demographic questionnaire at the end. Participants were given a summary of the study with the lab contact information in case they have additional questions after completing the study.

RESULTS

Data Screening

Prior to analysis, data was screened for outliers. Only five data observations were identified as a univariate outlier and that data was removed for only that item. Two data points were excluded from the STAI-Y2 and one data point from ASPS P1, P6, and P9 subscale were excluded ($n = 5$). Criteria for outliers were based on z-scores. If a data point was ± 3 standard deviations from the mean, that data point was excluded from analysis (Cousineau & Chartier, 2010). No participants were identified as multivariate outliers.

Analysis

This study utilized IBM SPSS Statistics 28.0. Bivariate correlations were conducted to assess the relationship between self-reported anxiety and sensory gating ability. We examined the effects of anxiety on sensory gating at a group-level analysis by utilizing ANOVAs and independent t-tests. A multilinear regression analysis was also conducted to examine how self-reported levels of anxiety predicts sensory processing. Bonferroni corrections were applied as a conservative measure to mitigate error regarding multiple comparisons. Alpha was set for .05 for all other inferential tests. Table 1 consists of questionnaire descriptive statistics.

Table 1. Questionnaire descriptive statistics.

Measure	<i>n</i>	<i>M</i>	<i>SD</i>
Generalized Anxiety Disorder-7 item	128	9.17	5.97
Penn State Worry Questionnaire	128	55.23	13.83
Beck Anxiety Inventory	128	22.07	15.00
State-Trait Anxiety Inventory-Y2 Form	128	48.48	6.11
Sensory Gating Inventory			
SGI total score	128	79.20	41.34
Perceptual Modulation	128	30.19	19.19
Distractibility	128	20.23	10.59
Over Inclusion	128	16.86	8.44
Fatigue and Stress Vulnerability	128	11.91	6.55
Highly Sensitive Person Scale	128	4.03	1.13
Adult Sensory Processing Scale			
Over responsive to vestibular (P1)	128	14.67	4.23
Over responsive to auditory (P2)	128	20.84	7.06
Over responsive to visual (P3)	128	14.65	5.05
Over responsive to tactile (P4)	128	11.90	4.20
Proprioceptive seeker (under) (P5)	128	11.43	4.04
General under responsive (P6)	128	12.50	3.25
Vestibular-Proprioceptive	128	10.70	3.76
Motor/Postural (P7)			
Under responsive to auditory	128	6.59	2.28
(seeking) (P8)			
Under responsive to tactile (P9)	128	7.55	2.30
Over responsive to vestibular (P10)	128	8.87	2.82
Over responsive to tactile (clothing) (P11)	128	9.24	2.84

Relationships Between Anxiety Measures and Sensory Gating

Bivariate correlations were run to examine the relationship between anxiety measure and sensory gating. Assumptions of normality and equal variance were met. Results demonstrated a positive correlation between all anxiety assessments and SGI total scores as well as SGI's facets of sensory gating. Table 2 consists of each correlation output. SGI total scores were positively correlated with BAI scores, $r(128) = .66, p < .001$ (see Figure 1), GAD-7 scores, $r(128) = .56, p < .001$ (see Figure 2), PSWQ scores, $r(128) = .52, p < .001$ (see Figure 3), and STAI-Y2 scores, $r(128) = .40, p < .001$ (see Figure 4). The results indicate that individuals who scored higher on anxiety measures also scored higher on the SGI and its facets of sensory processing, which indicate weaker sensory gating ability.

Table 2. Correlations between anxiety measures and SGI scores.

	GAD-7	BAI	PSWQ	STAI-Y2
SGI total	$r(128) = .56,$ $p < .001^*$	$r(128) = .66,$ $p < .001^*$	$r(128) = .52,$ $p < .001^*$	$r(126) = .40,$ $p < .001^*$
PM	$r(128) = .53,$ $p < .001^*$	$r(128) = .65,$ $p < .001^*$	$r(128) = .46,$ $p < .001^*$	$r(126) = .38,$ $p < .001^*$
D	$r(128) = .53,$ $p < .001^*$	$r(128) = .60,$ $p < .001^*$	$r(128) = .55,$ $p < .001^*$	$r(126) = .37,$ $p < .001^*$
OI	$r(128) = .50,$ $p < .001^*$	$r(128) = .59,$ $p < .001^*$	$r(128) = .44,$ $p < .001^*$	$r(128=6) = .34,$ $p < .001^*$
FSV	$r(128) = .50,$ $p < .001^*$	$r(128) = .56,$ $p < .001^*$	$r(128) = .48,$ $p < .001^*$	$r(126) = .40,$ $p < .001^*$

*Significant after a Bonferroni correction of $p < .003$

Bivariate correlations were also run to examine the relationship between HSPS scores and anxiety measurements. Results showed a positive correlation between HSPS average scores and every anxiety questionnaire. HSPS average score positively correlated with BAI scores, $r(128) = .68, p < .001$ (see Figure 5), GAD-7 scores, $r(128) = .61, p < .001$ (see Figure 6), PSWQ scores, $r(128) = .62, p < .001$ (see Figure 7), and STAI-Y2 scores, $r(128) = .38, p < .001$ (see Figure 8).

Anxiety measurements were also correlated with patterns on the ASPS. The BAI was correlated with all ASPS patterns except for patterns 5 (Proprioceptive Seeking), 8 (Underresponsiveness to Auditory Seeking), and 9 (Underresponsiveness to Tactile Input). The GAD-7 was associated all ASPS patterns except for patterns 3 (Overresponsiveness to Visual Input), 5 (Proprioceptive Seeking), 8, and 9 (Under Responsiveness to Auditory Seeking). The PSWQ was correlated with all ASPS patterns except for patterns 3, 5, 6 (Overall Underresponsiveness), 8, and 9. The STAI-Y2 was associated with only patterns 2 (Overresponsiveness to Auditory Input), 7, 10 (Overresponsiveness to Vestibular Input), and 11 (Overresponsiveness to Tactile). Table 3 contains all ASPS and anxiety measurement correlation output.

A multilinear regression analysis was utilized to develop a model for predicting an individual's SGI scores from various anxiety assessment scores. Assumptions of normality, equal variance, autocorrelation (Durbin-Watson = 2.5), and multicollinearity (VIF < 2.77) were met. Table 4 illustrates a summary of these results. The predictor model yielded a significant effect of anxiety on SGI scores, $F_{(4, 125)} = 29.665, p < .001$ with anxiety accounting for 50% of the variance, $R^2 = .495$. Beta weights for the anxiety measures revealed scores on the PSWQ (β

= .658, $p = .014$), BAI ($\beta = 1.258$, $p < .001$), and STAI-Y2 ($\beta = 1.127$, $p = .029$) all significantly predicted SGI scores in this model. The GAD-7 did not reach significance ($\beta = 0.183$, $p = .803$).

Table 3. Correlations between anxiety measures and ASPS patterns.

<i>ASPS Pattern</i>	BAI	GAD-7	PSWQ	STAI-Y2
P1	$r(127) = .46$ $p < .001^*$	$r(127) = .37$ $p < .001^*$	$r(127) = .34$ $p < .001^*$	$r(126) = .22$ $p = .013$
P2	$r(128) = .52$ $p < .001^*$	$r(128) = .46$ $p < .001^*$	$r(128) = .43$ $p < .001^*$	$r(126) = .29$ $p = .001^*$
P3	$r(128) = .30$ $p < .001^*$	$r(128) = .17$ $p = .061$	$r(128) = .22$ $p = .012$	$r(126) = .24$ $p = .007$
P4	$r(128) = .38$ $p < .001^*$	$r(128) = .34$ $p < .001^*$	$r(128) = .29$ $p < .001^*$	$r(126) = .22$ $p = .014$
P5	$r(128) = -.12$ $p = .183$	$r(128) = -.06$ $p = .494$	$r(128) = -.20$ $p = .027$	$r(126) = .02$ $p = .798$
P6	$r(127) = .36$ $p < .001^*$	$r(127) = .33$ $p < .001^*$	$r(127) = .20$ $p = .033$	$r(125) = .24$ $p = .007$
P7	$r(128) = .55$ $p < .001^*$	$r(127) = .46$ $p < .001^*$	$r(128) = .45$ $p < .001^*$	$r(126) = .30$ $p < .001^*$
P8	$r(128) = .12$ $p = .162$	$r(128) = .21$ $p = .018$	$r(128) = .10$ $p = .243$	$r(126) = .22$ $p = .015$
P9	$r(127) = .25$ $p = .005$	$r(127) = .12$ $p = .175$	$r(127) = -.01$ $p = .951$	$r(126) = .20$ $p = .024$
P10	$r(128) = .45$ $p < .001^*$	$r(128) = .40$ $p < .001^*$	$r(128) = .35$ $p < .001^*$	$r(126) = .30$ $p < .001^*$
P11	$r(128) = .42$ $p < .001^*$	$r(128) = .34$ $p < .001^*$	$r(128) = .34$ $p < .001^*$	$r(126) = .34$ $p < .001^*$

*Significant after a Bonferroni correction of alpha $p < .001$

Table 4. Anxiety assessment regression models.

<i>SGI total scores</i>	β	p	F	df	p	$adj. R^2$
Anxiety measures						
Overall model			29.67	4, 125	< .001*	0.50
GAD-7	0.18	0.80				
PSWQ	0.66	.01*				
BAI	1.26	<.001*				
STAI-Y2	1.13	.03*				

*Significant at $p < .05$

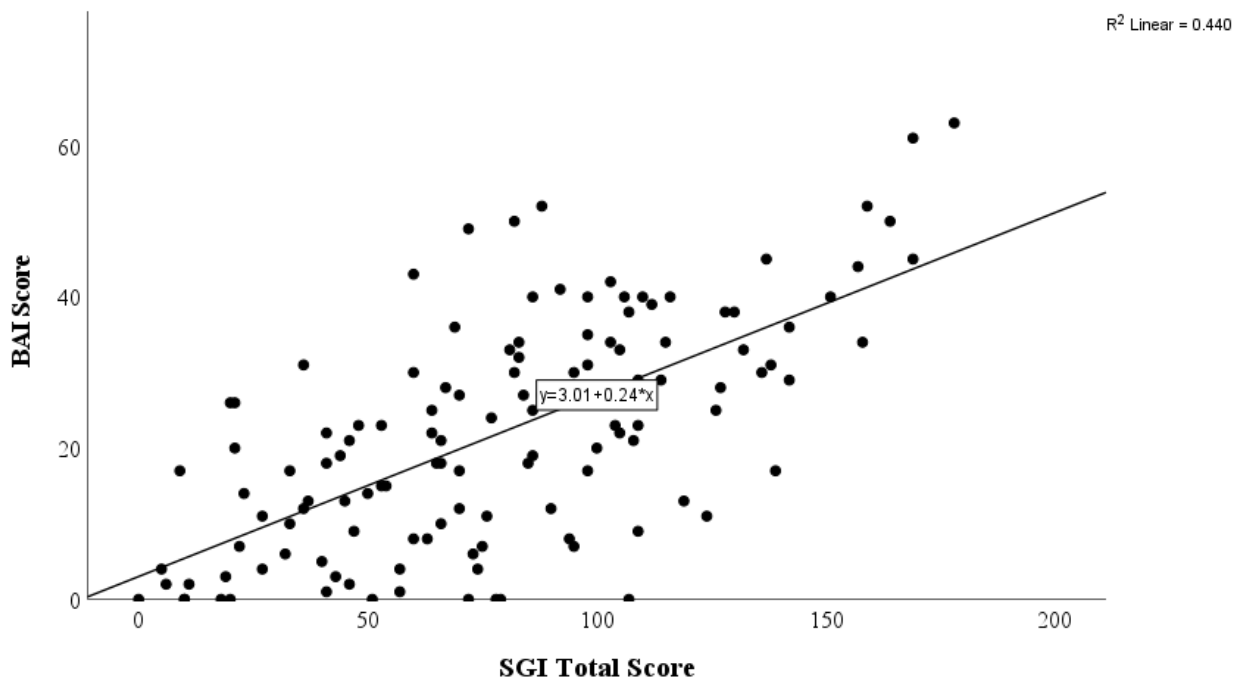


Figure 1. Relationship between SGI total score and BAI score, $r(128) = .66, p < .001$.

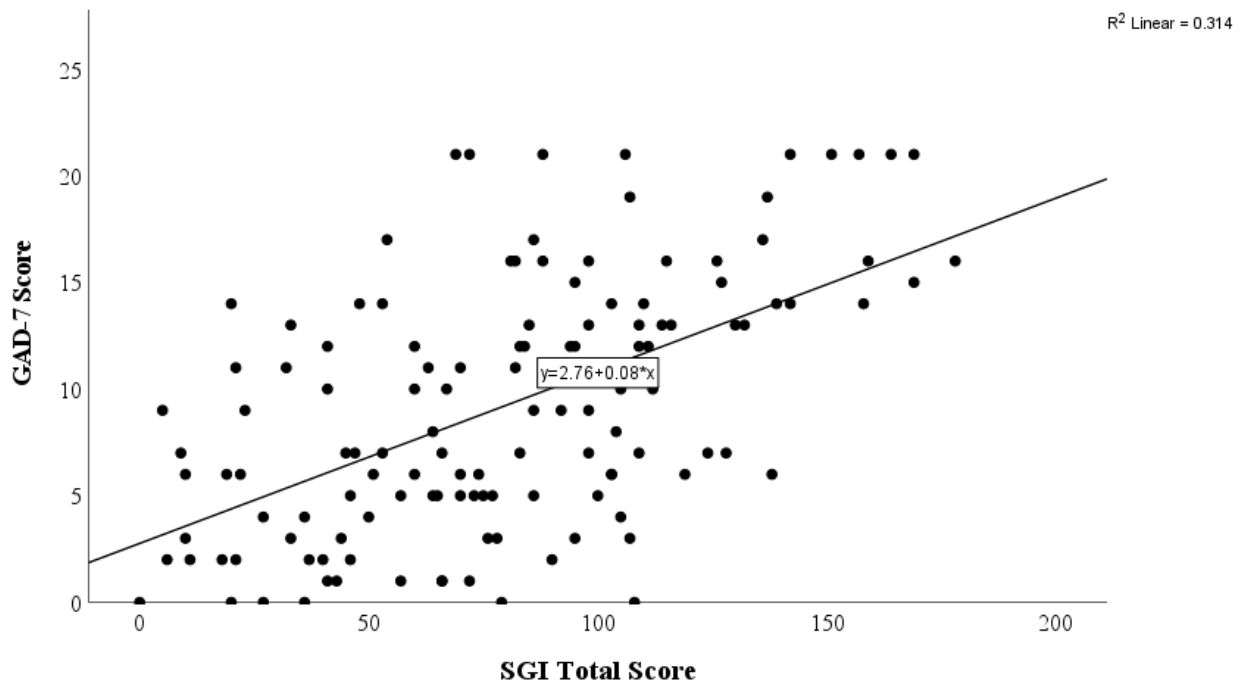


Figure 2. Relationship between SGI total score and GAD-7 score, $r(128) = .56, p < .001$.

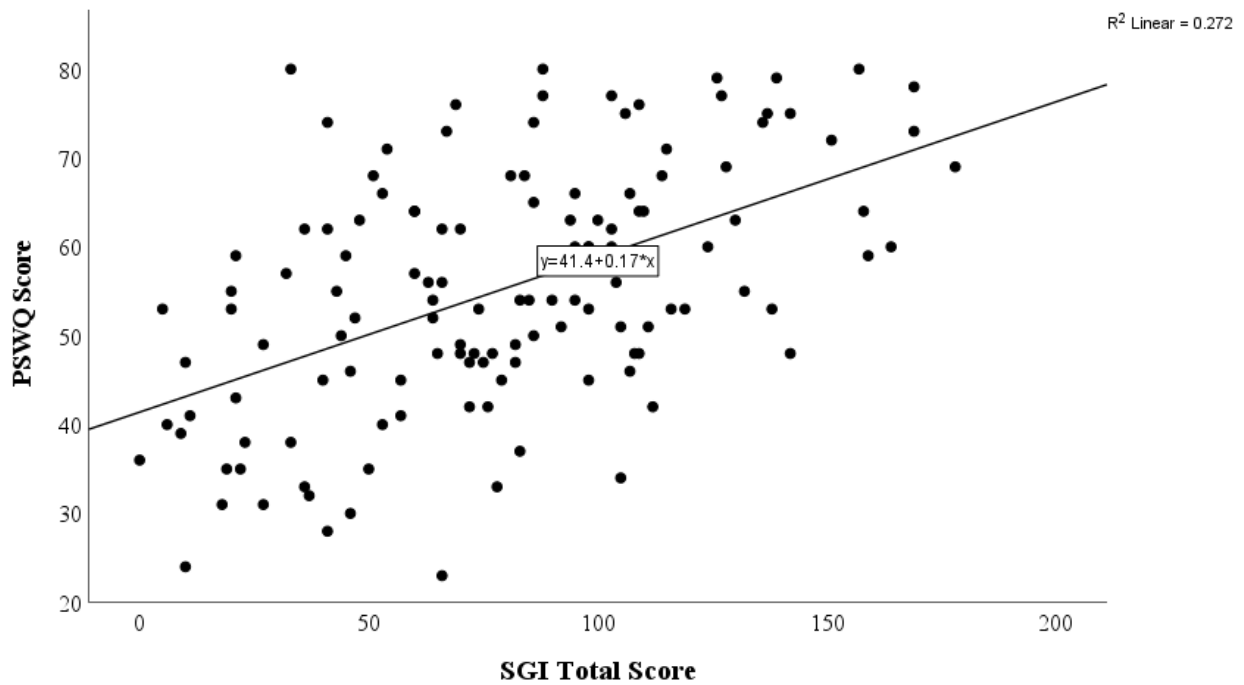


Figure 3. Relationship between SGI total score and PSWQ score, $r(128) = .52, p < .001$.

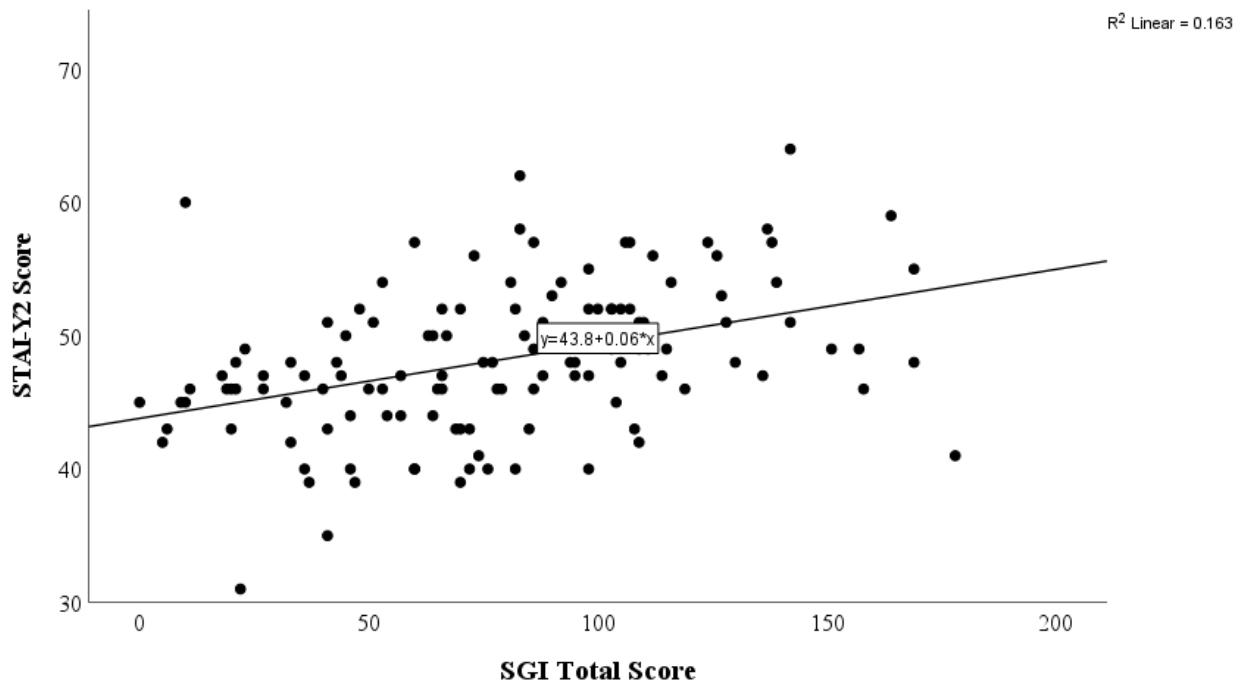


Figure 4. Relationship between SGI total score and STAI-Y2 score, $r(126) = .40, p < .001$.

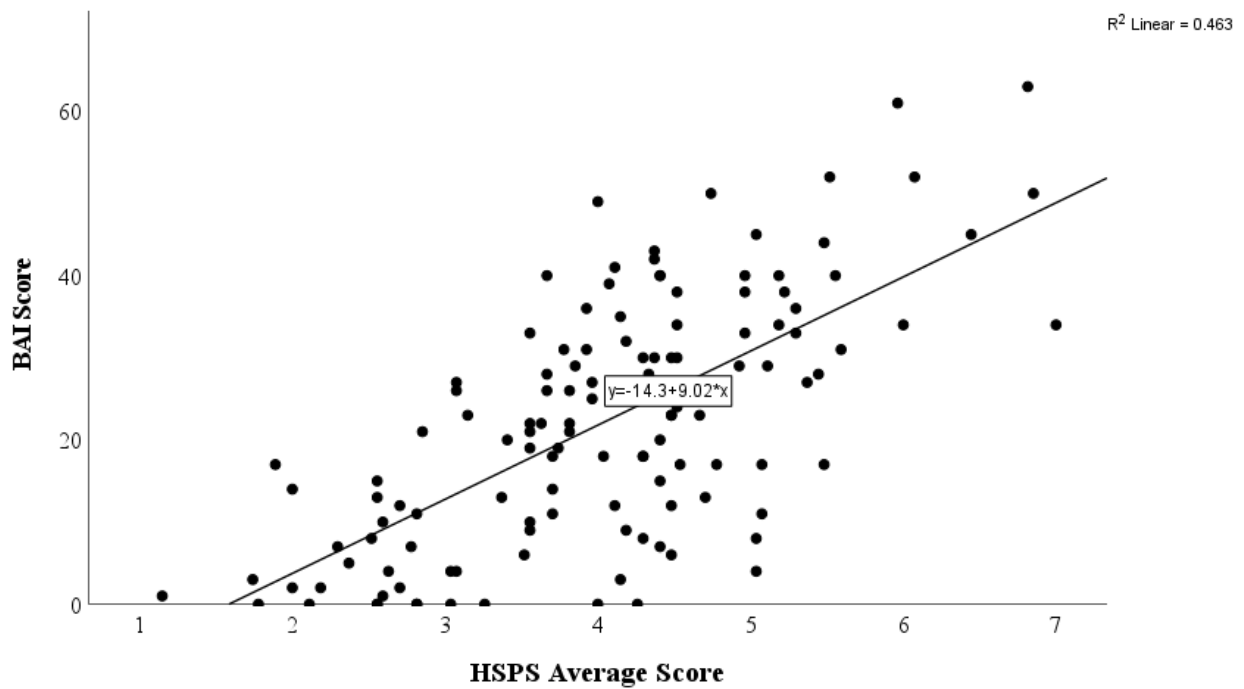


Figure 5. Relationship between HSPS average score and BAI score, $r(128) = .68, p < .001$

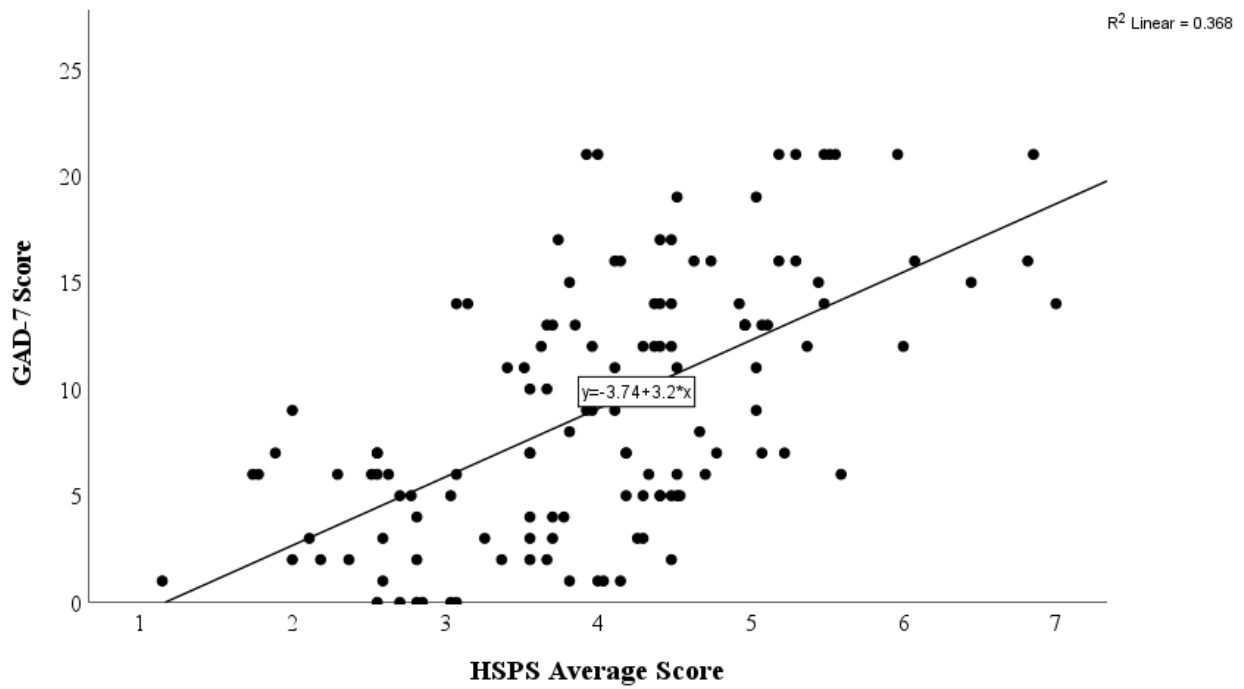


Figure 6. Relationship between HSPS average score and GAD-7 score, $r(128) = .61, p < .001$

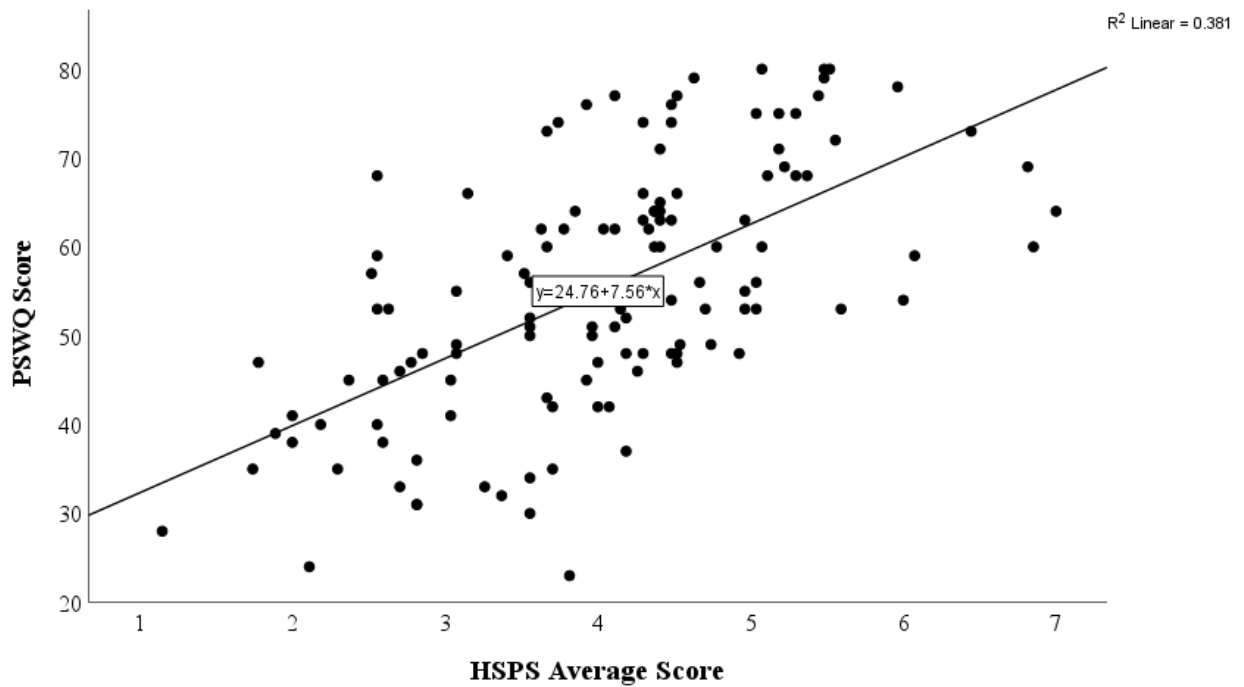


Figure 7. Relationship between HSPS average score and PSWQ score, $r(128) = .62, p < .001$

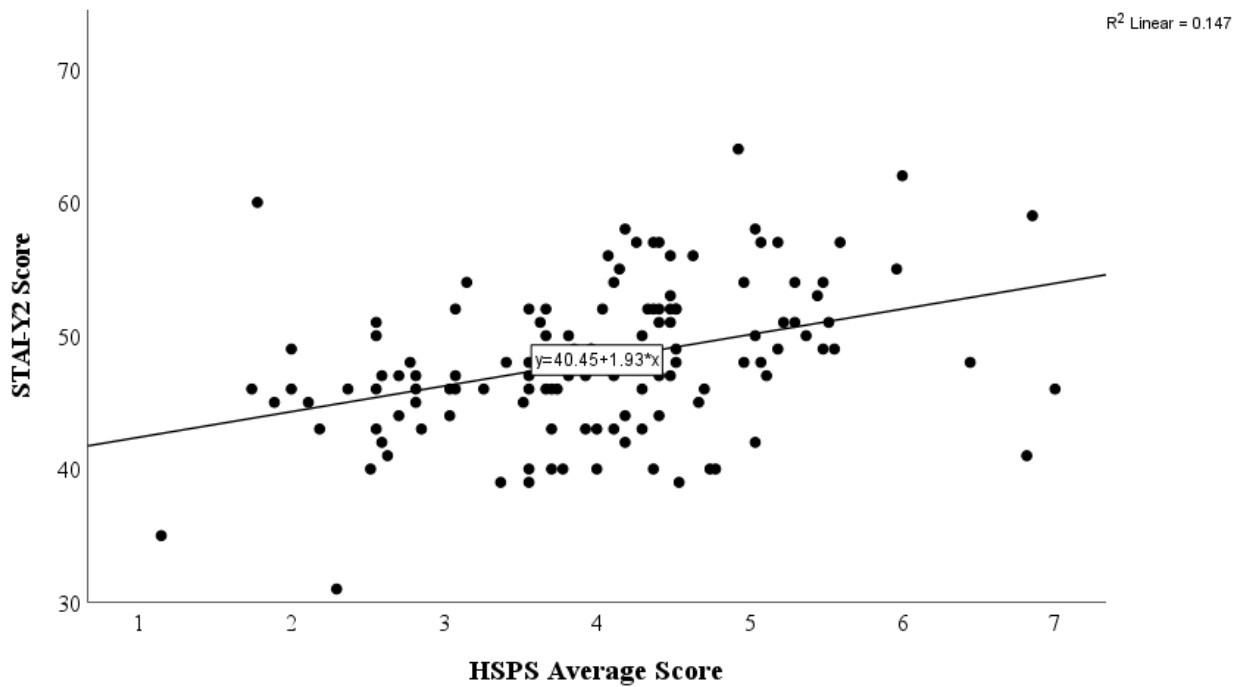


Figure 8. Relationship between HSPS average score and STAI-Y2 score, $r(128) = .34, p < .001$

Group-level Analysis for Anxiety

To assess the effects of anxiety level on sensory gating ability, participants were grouped together according to the assessment's scoring manual. Assumptions of normality and homogeneity of variance (Levene Statistics, $p > .05$) were met. BAI scoring is divided into four anxiety levels: minimal (0-7), mild (8-15), moderate (16-25), and severe (26-63) while GAD-7 scoring is divided into four categories: minimal (0-4), mild (5-9), moderate (10-14), and severe (15-21). The independent variable was SGI totals scores while the dependent variable was the four BAI levels. A one-way ANOVA revealed a significant difference in SGI total between four BAI levels scores ($F_{(3,124)} = 23.44, p < .001, \eta^2 = .37$). These results showed a significant main effect of anxiety levels on SGI total scores with 37% of variance being associated with anxiety levels. Tukey's Post-hoc comparisons indicate individuals who fell in the minimal range ($n = 27; M = 43.26, SD = 30.34$) endorsed significantly lower SGI scores than individuals who fell in the

severe range ($n = 51$; $M = 106.27$, $SD = 36.53$). These results suggest individuals who experience minimal anxiety report lower sensory gating difficulties. In addition, individuals who fell in the mild range ($n = 20$; $M = 63.80$, $SD = 29.93$) endorsed lower SGI total scores than those in the severe range. Lastly, individuals who fell in the moderate range ($n = 27$; $M = 72.37$, $SD = 32.16$) scored significantly lower than individuals who fell in the severe range ($M = 72.37$, $SD = 32.16$). A one-way ANOVA also revealed significant mean difference in SGI total scores ($F_{(3,127)} = 19.11$, $p < .001$, $\eta^2 = .32$) and four GAD-7 levels, with GAD-7 levels accounting for 32% of variance. A Tukey's Post-hoc comparison showed individuals who reported minimal anxiety ($n = 31$; $M = 49.84$, $SD = 31.16$) scored significantly lower on the SGI total score compared to individuals who reported moderate ($M = 85.88$, $SD = 36.58$, $n = 25$) and severe anxiety ($M = 118.24$, $SD = 36.47$, $n = 34$).

A group comparison between individuals who reported a diagnosis of GAD given by a clinical provider ($n = 24$) and individuals who did not endorse a GAD diagnosis ($n = 105$) was conducted. Assumptions of normality and equal variance (Levene's $p > .05$) were met. An independent samples t-test revealed individuals who reported a diagnosis of GAD scored significantly higher on the SGI total score ($t_{(126)} = 3.02$, $p = .003$, $d = .70$). This suggests that individuals who reported receiving a diagnosis of GAD endorsed more difficulty with sensory gating ($M = 102.04$, $SD = 44.03$) compared to individuals who do not report receiving a diagnosis ($M = 74.19$, $SD = 39.19$).

Another group comparison was run for the HSPS average score. Assumptions of normality and equal variance (Levene's $p > .05$) were met. An independent t-test yielded a significant difference between individuals who reported a diagnosis of GAD ($M = 4.6$, $SD = 1.26$) compared to individuals who did not ($M = 4.0$, $SD = 1.06$), $t_{(128)} = 2.88$, $p = .005$, $d = .66$.

This suggests individuals who report a diagnosis of GAD also experience significantly higher sensory sensitivity.

A one-way ANOVA was run examining group differences between anxiety severity, measured by the BAI, and ASPS patterns. The independent variable was ASPS patterns while the dependent variable was four BAI anxiety levels (minimal, mild, moderate, and severe).

Assumptions of normality and homogeneity of variance (Levene Statistics, $p > .05$) were met.

Significant mean differences were found in the following ASPS patterns: Pattern 1 ($F_{(1,3)} = 10.94, p < .001, \eta^2 = .22$), Pattern 2 ($F_{(1,3)} = 12.23, p < .001, \eta^2 = .23$), Pattern 4 ($F_{(1,3)} = 8.02, p < .001, \eta^2 = .17$), Pattern 6 ($F_{(1,3)} = 7.85, p < .001, \eta^2 = .16$), Pattern 7 ($F_{(1,3)} = 12.86, p < .001, \eta^2 = .24$), Pattern 9 ($F_{(1,3)} = 2.86, p = .04, \eta^2 = .07$), Pattern 10 ($F_{(1,3)} = 7.07, p < .001, \eta^2 = .15$), and Pattern 11 ($F_{(1,3)} = 9.37, p < .001, \eta^2 = .19$). Results demonstrated a significant main effect of anxiety of ASPS pattern scores, with anxiety accounting for 8%-24% of variance depending on the pattern. A Tukey's Post-hoc comparison revealed individuals who fell in the minimal range on the BAI ($M = 12.81, SD = 3.83$) and mild range ($M = 12.50, SD = 2.93$) scored significantly lower ($p < .001$) than individuals who fell in the severe range ($M = 16.80, SD = 4.08$) regarding Pattern 1 (Overresponsive to Vestibular). Differences were also found in Pattern 2 (Overresponsive to Auditory) where individuals who fell in the minimal ($M = 17.15, SD = 5.91$) and mild range ($M = 17.90, SD = 4.88$) endorsed significantly lower pattern scores compared to individuals who fell in the severe range ($M = 24.90, SD = 7.12$). Pattern 4 (Overresponsive to Tactile) scores were significantly different for individuals who fell in the minimal ($M = 10.30, SD = 4.06$), moderate ($M = 10.19, SD = 4.02$), and severe ($M = 13.94, SD = 4.07$). Individuals who endorsed minimal anxiety scores significantly lower compared to individuals in the severe range. In addition, individuals who scored in the moderate range also endorsed lower pattern

scores compared to individuals in the severe range. A significant mean difference was also found in Pattern 6 (General Underresponsive). Individuals who endorsed moderate levels of anxiety ($M = 10.96$, $SD = 2.50$) reported lower pattern scores than individuals who endorsed severe levels of anxiety ($M = 13.86$, $SD = 2.99$). Pattern 7 (Underresponsive to Proprioceptive-Vestibular) found similar results to Pattern 1 where individuals who fell in the minimal range ($M = 8.30$, $SD = 3.17$) scores significantly lower pattern scores compared to individuals who reported severe levels of anxiety ($M = 12.84$, $SD = 3.65$). Results for Pattern 10 (Overresponsive to Vestibular) were like Pattern 7. Individuals who fell in the minimal range ($M = 7.33$, $SD = 2.34$) scored reported lower pattern scores compared to individuals who fell in the severe range ($M = 9.84$, $SD = 3.11$). Similar to Pattern 10, individuals who endorsed minimal ($M = 7.78$, $SD = 2.28$) anxiety and moderate anxiety ($M = 8.26$, $SD = 3.03$) reported significantly lower pattern scores than individuals who endorsed severe anxiety ($M = 10.67$, $SD = 2.37$).

A one-way ANOVA was run examining group differences between anxiety severity, measured by the GAD-7, and ASPS patterns. Assumptions of normality and homogeneity of variance (Levene Statistics, $p > .05$) were met. An ANOVA revealed significant main effect of anxiety levels on the following ASPS patterns: Pattern 1 ($F_{(1,3)} = 9.89$, $p < .001$, $\eta^2 = .19$), Pattern 2 ($F_{(1,3)} = 12.76$, $p < .001$, $\eta^2 = .24$), Pattern 4 ($F_{(1,3)} = 6.63$, $p < .001$, $\eta^2 = .14$), Pattern 6 ($F_{(1,3)} = 3.86$, $p < .001$, $\eta^2 = .09$), Pattern 7 ($F_{(1,3)} = 13.40$, $p < .001$, $\eta^2 = .25$), Pattern 10 ($F_{(1,3)} = 8.25$, $p < .001$, $\eta^2 = .17$), and Pattern 11 ($F_{(1,3)} = 6.77$, $p < .001$, $\eta^2 = .14$), with anxiety levels accounting for 9%-20% of variance depending on the pattern. A Tukey's Post-hoc comparison revealed significant differences between minimal anxiety ($M = 12.35$, $SD = 3.05$) and severe anxiety ($M = 17.12$, $SD = 3.92$) regarding Pattern 1 (Overresponsiveness to Vestibular). Individuals who endorsed mild anxiety ($M = 19.76$, $SD = 6.07$) also reported lower Pattern 2

(Overresponsiveness to Auditory) scores compared to individuals who endorsed severe anxiety ($M = 26.36, SD = 7.16$). Similar to Pattern 1, Pattern 4 (Overresponsiveness to Tactile) and Pattern 10 (Overresponsiveness to Vestibular) showed similar results where individuals who reported minimal anxiety ($M = 9.39, SD = 3.69$ and $M = 7.55, SD = 2.46$, respectively) scored a lower pattern score compared to individuals who reported severe anxiety ($M = 13.72, SD = 4.12$ and $M = 10.56, SD = 3.48$, respectively). Pattern 7 (Underresponsiveness to Vestibular-Proprioceptive Motor/Postural Abilities) revealed a significant difference between minimal ($M = 9.39, SD = 3.13$) and severe levels ($M = 14.28, SD = 3.45$), severe and mild levels ($M = 9.39, SD = 3.60$), and moderate ($M = 10.71, SD = 2.99$) and severe levels of anxiety. Pattern 11 (Overresponsiveness to Tactile (clothing)) found significant differences between minimal ($M = 8.29, SD = 2.61$) and severe ($M = 11.16, SD = 2.29$) and mild ($M = 8.50, SD = 2.81$) and severe levels of anxiety.

A group comparison analyzed the ASPS patterns between groups to examine if differences exist among individuals who reported a diagnosis of GAD compared to individuals who did not (Table 5). Assumptions of normality and equal variance (Levene's $p > .05$) were met. An independent t-test demonstrated a significance difference between Pattern 7 (Underresponsiveness to Vestibular-Proprioceptive Motor/Postural Abilities). Individuals who reported receiving diagnosis of GAD scored significantly lower ($p < .001$) on ASPS's pattern of Underresponsiveness to Vestibular-Proprioceptive Motor/Postural Abilities ($M = 13.22, SD = 3.94$) compared to their counterparts ($M = 10.14, SD = 3.50$). This indicates individuals who reported a GAD diagnosis are more unresponsive to their balance and body position (i.e., leaning on furniture, slouching, etc.). Only Pattern 7 reached statistical significance.

Table 5. ASPS patterns between groups.

	<i>F</i>	<i>t</i>	<i>df</i>	<i>p</i>	<i>d</i>
P1	.560	-.059	125	.953	-.013
P2	.000	.768	126	.444	.177
P3	.124	1.705	126	.091	.392
P4	1.618	1.171	126	.244	.270
P5	.114	-2.253	126	.026	-.519
P6	2.505	.177	125	.860	.041
P7	.053	3.733	126	<.001*	.859
P8	.644	.556	126	.579	.128
P9	.302	-.874	125	.384	-.201
P10	.337	1.381	126	.170	.318
P11	.057	1.670	126	.097	.384

*Significant at $p < .001$

DISCUSSION

This study explored the relationship between anxiety and self-reported sensory processing utilizing alternative measures, like the SGI. Our exploratory study provided evidence that anxiety and sensory processing are closely related. Bivariate correlations revealed individuals who reported more sensory processing difficulties also experienced more symptoms of anxiety. Group comparisons demonstrated individuals who reported a diagnosis of GAD endorsed significantly higher sensory processing difficulties. These findings were consistent for both the SGI and the HSPS, but not the ASPS. Interestingly, results for the ASPS were inconsistent. Anxiety measurements correlated with most of the 11 ASPS patterns except for the STAI-Y2 questionnaire. The STAI-Y2 was correlated with only four of the 11 factors which include following: P2 (Overresponsiveness to Auditory), P7 (Underresponsiveness to Proprioceptive-Vestibular), P10 (Overresponsiveness to Vestibular), P11 (Overresponsiveness to Tactile). The STAI-Y2 has been found to contain poor discriminant validity between anxious and depressed states. It is possible this inconsistency is due to the lack of discriminability in the STAI-Y2. Group comparisons revealed a significant difference between only Pattern 1, Underresponsiveness to Vestibular-Proprioceptive Motor/Postural Abilities, which is not often associated with anxiety.

Furthermore, a multilinear regression demonstrated differences between the anxiety measures when predicting SGI total scores. The GAD-7 and the PSWQ are both designed to screen for GAD while the BAI and STAI-Y2 aim at measuring anxiety and anxiety proneness, respectively. Although the GAD-7 was significantly correlated with the SGI, it did not significantly predict SGI total scores. The GAD-7 items mainly focus on worry and controlling

worry, which is a key feature to GAD; however, the GAD-7 does not contain items focusing on physiological symptoms of anxiety. BAI is designed to measure the intensity of somatic anxiety symptoms; however, it does not assess other main symptoms of anxiety, such as worry.

Interestingly, the BAI had the strongest correlation with the SGI total scores and was the strongest predictor in the multilinear regression analysis. It is possible that sensory processing and physiological symptoms of anxiety are related more than cognitive symptoms. Our results are similar to past research examining poor sensory gating in anxiety psychopathologies. Orr and colleagues (2002) compared sensory gating abilities of male Vietnam combat veterans and female rape victims with PTSD to individuals without PTSD. Results showed that individuals diagnosed with PTSD did not display the typical reduction in the P50 response to the second stimulus. Furthermore, poor sensory gating has also been found in individuals diagnosed with PTSD. Gillette and colleagues (1997) discovered that weaker P50 suppression in male Vietnam combat veterans with PTSD was strongly correlated with higher intensity PTSD re-experiencing. Both panic disorder and PTSD contain diagnostic criteria that are more physiologically oriented compared to GAD.

The DSM-5 noted GAD symptoms may include somatic symptoms (i.e., sweating, nausea, diarrhea) and exaggerated startle response; however, autonomic hyperarousal (i.e., increased heart rate, shortness of breath, dizziness) are less prominent in GAD. Patriquin and Mathew (2017) posited that psychophysiological symptoms of GAD may be context specific and are not universally found. White and Yee (1997) also found a difference in the role of various psychological stressors on sensory gating ability. They found a silent math task did not suppress sensory gating ability; however, an oral math task significantly affected gating ability. It seems the impact of stress and anxiety on sensory gating ability can be vary depending on the

psychological stressor. In order to meet criteria for GAD, individuals must display three of the six symptoms: restlessness, being easily fatigued, difficulty concentrating, irritability, muscle tension, and sleep disturbance. This study screened for overall presence of anxiety rather than specific symptoms. A closer look at specific GAD symptoms, more specifically physiological symptoms, and how they may relate to sensory processing might help explain the variability in psychological stressors.

Based on self-report assessment, our study provided evidence that sensory gating and anxiety are related. Overall, individuals who report weaker sensory gating ability also endorse more symptoms of anxiety. A better understanding of the sensory gating mechanism will help us understand sensory gating in clinical groups and may improve our ability to design effective interventions and treatments for individuals affected by anxiety and sensory sensitivity. Treatments for anxiety exist, however most therapy modalities focus on the cognitive symptoms of anxiety such as uncontrollable worry and reframing cognitive thinking. One such therapy intervention includes grounding techniques (e.g., placing hands in water, breathing deeply, notice 5 things, picking up or touching an item close by, etc.) which are aimed at distracting or shifting focus from distressing experiences. It is possible therapy interventions designed to help block out or reduce focus on sensory stimuli in the environment will assist individuals affected by anxiety more than reframing cognitive thinking. Another intervention is mindfulness. Mindfulness is defined as the quality of awareness and attention being placed on a current experience without passing judgement (Baer et.al., 2004; Brown & Ryan, 2003).

Research suggests that individuals who are more mindful tend to display healthier responses to stressful situations, indicating that mindfulness effectively assists in emotion regulation (Kadziolka et. al., 2015). Research suggests that mindfulness practices may improve trait

mindfulness and thus impact everyday sensory and emotional processing (Brown & Ryan, 2003; Quaglia et al., 2016).

Kadziolka and colleagues stated the being mindful depends on shifting attention from distractions back to the present moment. Anxiety has been related to attention control and perception of anticipatory threat. Najmi and colleagues (2014, p. 1) defined attention control as “the ability to use cognitive resources selectively to inhibit the processing of certain stimuli.” Research supports a strong relationship between anxiety and attention. Low attentional control has been associated with characteristics of GAD, such as worry and rumination. White and colleagues (2009) founds those anxious individuals tend to show automatic, involuntary attentional bias toward threatening information compared to non-anxious individuals. Sensory gating has been correlated with attention. For example, Guterman and colleagues (1992) conducted a study looking at attentional influence on the P50 component. Their findings indicate the auditory P50 response can be modified by task-relevant factors; however, these findings are highly debated as the P50 component is seen as mostly pre-attentive while the N100 component may be modified by attention. Furthermore, Wan (2008) found P50 suppression was positively correlated with better performance on attention tasks. Yadon and colleagues (2009) also found a relationship between N100 component and neuropsychological measures of attention and deficits in N100 filtering seem to relate to attentional problems. Higher-level attention tasks were also better predictors of gating for the N100 component. Examining the neurological processes in attention such as bottom-up and top-down processing may assist in connecting the physiological processes of anxiety and sensory processing.

Limitations

There are several limitations in this study, one being that our study relied on self-report responses for all completed measurements. The validity of this study relied on participant insight when providing responses. Furthermore, researchers did not complete a clinical interview to screen or diagnose GAD in the sample. Most of the diagnoses reported were given by a primary care physician ($n = 17$) rather than a clinical provider trained in screening and assessment of mental disorders. Results and findings should be interpreted with some degree of caution as it is possible reported GAD diagnoses were not accurate or current. Another important limitation is the lack of a physiological component. Social distancing due to COVID-19 prevented participants from completing an EEG session. This study collected data from college aged students attending a Midwestern university. It is possible results are not generalizable due to lack of diversity in our sample. Finally, this study was exploratory thus no cause-and-effect relationships can be established.

Future Direction

Overall, this study warrants future research examining anxiety and sensory processing. Although there is a limited amount of literature on acute stressors, no known research has been conducted looking at the effects of chronic worry and anxiety on sensory gating ability. Furthermore, limited research exists addressing the differential and biological aspects of GAD (Patriquin and Mathew, 2017). Hetrick and colleagues (2012) stated, “It seems prudent to keep in mind that any given psychological or neuropsychological construct is distinct from its operationalization and may never be fully captured by any 1 measurement” (p. 189). Kisley and colleagues (2004) conducted a comparison of sensory gating to mismatched negativity and self-

reported perceptual phenomena, measured by the SGI, however research is limited comparing ERPs with self-report questionnaire. Additional research examining the differences between self-reported and physiological measures of sensory gating, specifically utilizing event-related potentials (ERPs) through EEGs the may close gaps between experiential and physiological constructs of sensory gating. Furthermore, electrophysiological measurements for sensory gating are resource intensive. Establishing a time and cost-effective measurement similar to an EEG may benefit both researchers and clinicians who do not have access to electrophysiological tools.

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APPENDICES

Appendix A: IRB Approval

Date: 3-31-2021

IRB #: IRB-FY2020-486
Title: GAD and Auditory Sensory Gating
Creation Date: 1-9-2020
End Date:
Status: Approved
Principal Investigator: Carly Yadon
Review Board: MSU
Sponsor:

Study History

Submission Type	Initial	Review Type	Expedited	Decision	Approved
Submission Type	Modification	Review Type	Expedited	Decision	Approved

Key Study Contacts

Member	Carly Yadon	Role	Principal Investigator	Contact	carlyyadon@missouristate.edu
Member	Elizabeth Troutwine	Role	Primary Contact	Contact	elizabeth345@live.missouristate.edu

Appendix B: Informed Consent Form



Introduction: You are invited to participate in a research project conducted in the Psychology Department at Missouri State University. The purpose of this study is to explore the relationship between how you are feeling and sensory processing. Carefully read the information in this document. If you have any questions or concerns related to this study, please contact one of the people listed below.

Participation: Student participants will complete various surveys and a demographic data form (approximately 45 minutes will be required).

Risks: The demographic form does request the participant to include their student name and identification number (M-number). As with collecting any identifying information, there is a slight risk of a break in confidentiality. See below for methods to reduce this risk.

Procedures for Minimizing Risks/Confidentiality of Records: All the information you provide will be kept strictly confidential and maintained on Qualtrics. This is protected by a secure log-in known only to the Principal Investigator and research assistants. Only researchers directly involved in this study will have access to participant information. Results of the project may be presented at conferences or in publications; however, your identity will not be disclosed as the results from this study will always be presented in group format.

Benefits: Results from this project will increase understanding the connection emotion and sensory processing. You may not benefit directly from this study. However, the information from this study may help you think about how your brain works, specifically how your brain processes sensory information and how that may be related to other behaviors. The information you provide will expand the knowledge in the field and because of that has the potential to positively affect others.

Voluntary Participation: This study is based on voluntary participation. If you feel uncomfortable answering any question(s) within the survey, feel free to skip the question(s) and continue to the next item. Additionally, if you feel uncomfortable completing the survey, you may withdraw from the study at any time. There is no penalty for discontinuing participation.

Contact Persons: Further information regarding this project may be obtained by contacting Dr. Carly Yadon or Elizabeth Troutwine

Appendix C: Demographic Questionnaire

Demographic Questionnaire

What is your name?

What is your M Number?

1. What is your age?
2. What is your assigned sex at birth?
3. What is your gender?
 - Male
 - Female
 - Non-binary/third gender
 - Prefer to self-describe
4. What is your handedness?
 - Right
 - Left
 - Ambidextrous (both)
5. What is your highest degree completed?
6. Do you currently have a sleep disorder?
 - Yes
 - No (redirect to #8)
7. If yes, please explain:
8. Have you ever had a brain injury?
 - Yes
 - No (redirect to #13)
9. If so, please explain the type of injury:
10. Did you lose consciousness?
 - Yes
 - No (redirect to #12)
11. Approximately how long were you unconscious?
12. Were you hospitalized?
 - Yes
 - No
13. Have you been diagnosed with ADD or ADHD?
 - Yes
 - No
14. Have you ever been diagnosed with any type of neurological disorder (e.g., seizures, epilepsy, tic disorder, anxiety, depression)?
 - Yes
 - No (redirect to #16).
15. Please explain your neurological disorder

16. Do you currently have a diagnosis of Generalized Anxiety Disorder (GAD)?
Yes
No
17. Have you had a diagnosis of GAD in the past?
Yes
No
18. Please explain, in as much detail as you feel comfortable, your present/past diagnosis of GAD 9e.g. who diagnosed you (primary care physician, counselor, therapist, etc.), when you were diagnosed, what age you were diagnosed, etc.). (If not applicable, please right NA)
19. Does anyone in your family have schizophrenia?
Yes
No (redirect to #21)
20. Please explain which family member
21. Are you currently taking any prescription medications?
Yes
No (redirect to #23)
22. Please list the type of medication that you take:
23. Have you been prescribed medical marijuana?
Yes
No (redirect to #26)
24. When was the last time you consumed medical marijuana?
25. How much medical marijuana did you consume?
26. When was the last time you consumed caffeine?
27. What was it (e.g. coffee, tea, NoDoz)?
How much did you consume? [Please be as specific as possible, e.g., 2 cups of coffee at 7am].
28. When was the last time you consumed alcohol? About how much?
29. Do you use nicotine products (e.g., smokeless tobacco, cigarettes, cigars, vape, Juul)?
Yes
No (redirect to #31)
30. How many hours has it been since you last used one of the listed nicotine products?

31. Did you get less sleep last night than is usual for you?

Yes

No

32. Do you have normal vision?

Yes

No

33. Do you have vision loss?

Yes

No (redirect to #35)

34. Please explain your vision loss:

35. Do you have corrected vision (e.g., glasses or contacts)?

Yes

No

36. Are you colorblind?

Yes

No

37. Do you have normal hearing?

Yes (redirect to #40)

No

38. Do you have hearing loss?

Yes

No (redirect to #40)

39. Please explain your hearing loss:

40. Do you have corrected hearing?

Yes

No

Thank you for participating in our study! The reason for this study is to explore how the brain processes sensory information and how that may relate to stress and anxiety. Sensory gating is conceptualized as the ability of neurons to stop responding to constant stimuli. We experience this phenomenon every day and each of our senses displays this property. For example, when you first put on your watch in the morning you notice it is there and can feel pressure against your skin. After a few minutes, however, you do not feel your watch anymore. This is because the sensory neurons on your wrist send the same information to your brain, which begins to actively "ignore" this redundant information. This type of sensory gating is thought to protect us from sensory overload. The pathology of some clinical conditions such as schizophrenia and autistic spectrum disorders is thought to involve impaired sensory gating. Therefore, it is important to study the phenomenon in healthy individuals to learn more about the mechanism. We are particularly interested in how sensory processing may relate to stress and anxiety.