THESIS

EXAMINING SENSORY GATING AND PROCESSING SPEED IN ADULTS WITH AUTISM USING EEG

Submitted by

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ABSTRACT

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Objectives. Most individuals with Autism Spectrum Disorder (ASD) experience sensory deficits in their auditory processing (Tomchek & Dunn, 2007). These deficits can further impact their ability to participate in their physical and social environments. One way to increase understanding of these deficits is through use of electroencephalography (EEG), which measures brain activity in real-time and is able to distinguish brain processes such as sensory processing and the deficits that might be occurring during this process (Davies & Gavin, 2007). This study's purpose is to understand how processing speed and ability to filter out irrelevant stimuli impacts adults with ASD compared to their neurotypical (NT) peers through measurements of latency of prominent brain activity following presentation of an auditory stimulus and sensory gating. This study also analyzed how active and passive attention states impact sensory gating and latency. Methods. 24 adults with autism (M = 23.3 years, SD = 3.8) and 24 neurotypical adults (M = 23.7 years, SD = 3.5) participated in this study. They completed a sensory gating paradigm in both an active and a passive listening condition. In the active condition they were asked to press a button when they heard a single click, and in the passive condition they simply stared at a static image on a screen while the auditory stimuli were presented to them. Results. The results showed that there are no significantly different sensory gating responses between the ASD and NT groups. Individuals with ASD had delayed processing speed as measured through latency as early as 100 milliseconds following an auditory stimulus. Both groups experienced

slower processing in the passive condition starting at approximately 200 milliseconds post-stimulus onset. As expected, more gating was observed for both groups in the passive condition at early components, where-as the active condition - which required attention to the stimulus that is usually suppressed in this task - resulted in less gating. In the latest component analyzed, approximately 200 milliseconds post-stimulus, both groups showed more gating in the active attention state, which was the opposite of the expected results, and the possible reason for this unexpected result needs further exploration. Conclusions. These findings suggest that individuals with autism do not have deficits in the ability to filter out irrelevant stimuli, however, they are likely more impacted by delayed processing speeds. Implications for practice include allowing more time to process auditory information for individuals with autism, and using compensatory strategies to influence neural processing speeds and amount of gating in response to auditory stimuli through the use of activity demands to create either passive or active attention states.

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CHAPTER 1 – INTRODUCTION

This study intends to further explain the differences in auditory processing in adults with high functioning autism and neurotypical adults. Neurological processing of auditory stimuli was observed using brain imaging and discrete moments within auditory processing were analyzed and reported. This study was interested in both the speed of processing after an auditory stimulus, and the ability to filter out redundant information. This study also investigated how a participant's state of attention affects their neurological auditory processing, with comparisons between an active listening state and a passive listening state.

Autism Spectrum Disorders

Autism was first described and categorized by Leo Kanner in 1943. He reported on several case studies in which he observed children with repetitive movements and speech, limited spontaneous activity, rigidity to contextual changes, sensitivity to invasive sounds and movements, and obsessive behaviors (Kanner, 1943). While our understanding of autism has changed over the decades, many of those parameters still exist in the current definition of autism.

The Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) outlines the current criteria for the diagnosis of Autism Spectrum Disorder (ASD) (American Psychiatric Association, 2013). The first set of criteria addresses deficits in social interaction and communication and includes deficits in social-emotional reciprocity, deficits in non-verbal communication, and difficulties in forming and maintaining relationships. The second set of criteria addresses a presence of restricted and repetitive behaviors, interests, or activities and includes repetitive speech, movement, and use of objects; strong adherence to rituals or excessive resistance to change; highly restrictive and abnormally obsessive interests; and

excessive or limited reactions to sensory input from the environment. A person must meet all three areas of the first set of criteria and two out of the four areas of the second set of criteria to meet the DSM-5 standards for a diagnosis of ASD (APA, 2013).

The most recent reports by the Centers for Disease Control and Prevention (CDC) report that approximately 1 in 68 children have ASD (2012). This ratio has continually increased from their original report of 1 in 150 children having ASD in the year 2000. While these reports are specifically looking at children, these increasing numbers also apply to adults as the population ages, since ASD is a developmental disorder that affects each person for life. One of the areas of common deficit for individuals with autism is in processing the sensory experiences of their environment (Case-Smith & O'Brien, 2015). While these deficits in processing sensory information can occur for any individuals, they are found to occur at a higher rate for individuals with autism (Davies & Tucker, 2010) and can further impact the person's ability to communicate and participate in their physical and social environments.

Sensory Processing in ASD

Sensory processing is "the act of perceiving and using stimuli presented to the sensory systems for integration with past and present experience and meaning for a behavior response" (Llorens, 1986, pp. 104). More simply put, it is the process by which the brain organizes sensory input from the environment for functional use (Su & Parham, 2014). There are six distinct systems recognized in sensory processing: visual, auditory, tactile, vestibular, proprioceptive, and taste/smell (Miller, Anzalone, Lane, Cermak, & Osten, 2007). These systems can function improperly and provide inaccurate information to the body about the stimuli that is being experienced, which in response can cause impairment in daily routines such as dressing, bathing, and going out into the community (Su & Parham, 2014). When these daily

routines are impacted by a dysfunction in sensory processing, it is called a Sensory Processing Disorder (Miller et al., 2007), or Sensory Integration Dysfunction (Ayres, 1989). While these terms both reference deficits in the ability to process and use sensory information (Schaaf & Davies, 2010), they come from different origins, with sensory integration dysfunction born of the sensory integration theory established by Ayres (1969), and sensory processing disorder more recently presented as a category for diagnostic use by Miller et al. (2007). While some people use these terms interchangeably, for this paper we will be using Sensory Processing Disorder to refer to deficits in the processing and use of sensory input.

Since sensory processing disorder (SPD) was first identified, it has been associated with ASD (Ayres & Tickle, 1980; Lovaas, Koegel, & Schreibman, 1979; Minshew & Hobson, 2008; Ornitz, 1976; Tomchek & Dunn, 2007). Research has consistently shown that those with a diagnosis of ASD tend to also have dysfunction in at least one of their sensory processing systems (Minshew & Hobson, 2008; Su & Parham, 2014; Tomchek & Dunn, 2007). A study performed by Tomchek & Dunn (2007) compared sensory processing deficits in children with ASD to typically developing peers and found that 77.6% of children with ASD had abnormal auditory processing. Specifically, they found that these children tended to "tune out" spoken language and had trouble functioning when there are distractions or background noise.

Research has shown that sensory processing can be observed through both observable behaviors and neural processing (Davies & Gavin, 2007; Griefe, 2013). Any time that sensory processing occurs, it involves neurons in the central nervous system (CNS) which produce an electrical signal to inform the brain of the received stimulus (Lane, 2002). This electrical signal can be detected and recorded using a technique called electroencephalography (EEG). The results of these recordings can be used to analyze and interpret how a person's brain was

processing the sensory information on a neural level and can show when dysfunction in the process is occurring (Davies & Gavin, 2007).

Introduction to EEG and ERPs

EEG is both a medical imaging technique and research tool that uses metal electrodes placed on the surface of the scalp to pick up electrical activity from the underlying brain structures (Teplan, 2002). This electrical activity is then amplified and recorded and is often used to explore and explain neurological processes (Teplan, 2002). EEG has advantages in that it is taking direct measurements of the brain's activity in real-time and is sensitive enough to pick up electrical activity from processes such as sensory processing (Banaschewsk & Brandeis, 2007; Davies & Gavin, 2007)

One way that we examine and interpret EEG results is by looking at event-related potentials, or, ERPs. ERPs are the brain's immediate response to stimuli (internal or external) and can be captured in both amplitudes and latencies (Davies & Gavin, 2007). They occur amidst many other brain processes, so to be able to identify them multiple accounts of the response must be recorded and then averaged to find the pattern of response. Amplitude is the amount of voltage output by the brain in response to the stimuli. When a larger amplitude is observed in an ERP component, it often indicates that more processing is occurring in the brain at that moment in time (Gavin et al., 2011). Latency is the amount of time (in milliseconds) that has elapsed between the stimulus onset and the maximum peak amplitude in the ERP component being studied (Gavin et al., 2011). Thus, latency provides information about processing speed. Using EEG and ERPs it is possible to identify when dysfunctional or atypical brain processes are occurring, and sometimes it is even possible to explain why (Banaschewsk & Brandeis, 2007).

Sensory Gating Paradigm

While recording EEG, certain paradigms or tasks can be used to elicit ERPs related to sensory processing. One such paradigm is called sensory gating. Sensory gating is the process by which the brain suppresses neural responses to repeated, incoming stimuli (Boutros, Belger, Campbell, D'Souza, & Krystal, 1999; Jones, Hills, Dick, Jones, & Bright, 2016). In this study we are using an auditory sensory gating paradigm, the paired click paradigm, in which the participants hear repeated presentations of paired click stimuli. In this paradigm, sensory gating would be seen if the ERP amplitudes following the second click are smaller than the ERP amplitudes following the first click. The difference in amplitudes would show that the person has a decreased response to unnecessary and repetitive stimuli, which is a facet of sensory processing.

Within ERPs are individual components, or voltage deflections, that represent different neurological processes that occur within sensory gating (Boutros, Korzyukov, Jansen, Feingold, & Bell, 2004; Lijffijt et al., 2009; Rentzsch, Jockers-Scherübl, Boutros, & Gallinat, 2008). These components are defined by whether they are positive or negative peaks (P or N, respectively) and for the times at which they tend to occur following the stimulus (in milliseconds). Thus, the N1 component represents a negative peak that occurs approximately 100 milliseconds following the stimulus, the P2 component represents a positive peak that occurs approximately 200 milliseconds following the stimulus, and the N2 component represents a negative peak that occurs approximately 200 milliseconds following the stimulus. In this study we are focusing on the N1, P2, and N2 components. These components will be discussed in further detail in later sections. The P50 component will also be discussed, as the body of

research concerning this component related to sensory gating is larger and provides context for the processes of the later components on which this study is focused.

Using EEG to observe sensory gating in individuals with ASD is an emerging way of learning more about how they process sensory information. It can inform us of what specific deficits are occurring, and therefore guide interventions that target specific sensory, cognitive and behavioral deficits (Jeste & Nelson, 2009). An analytical review performed by Jeste & Nelson (2009) on the current state of ERP research in children with ASD found that overall the ERPs of children with ASD during auditory processing were abnormal compared to control groups. The abnormalities themselves were inconsistent though, with some studies finding longer latencies than the control group, others finding shorter latencies than the control group, and some studies finding smaller amplitudes in the ASD group. Overall, studies reporting on the ERPs of individuals with ASD in relation to sensory gating are infrequent and tend to be inconsistent. Looking at specific components involved in gating can further inform us about what is unique about gating in individuals with ASD, and show us where gaps in the literature exist.

P50 Component and Sensory Gating

Sensory gating at the P50 component has proven to be a reliable, significant neurological response by neurotypical people (Griefe, 2013; Rentzsch, Jockers-Scherübl, Boutros, & Gallinat, 2008). It has been associated with measures of both attention and anxiety (Madsen et al., 2015; Potter, Summerfelt, Gold, & Buchanan, 2006). Attention has been found to be one of the most significant correlating factors with the P50 component, with those who had more abnormal gating at P50 also doing worse on behavioral tests of attention (Potter et al., 2006). Differences have been found between groups of typically developing children and children with SPD at the

P50 component, with specific note that children with SPD did not have a significant relationship between their age and their sensory gating at P50, as the typically developing children did (Davies & Gavin, 2007).

While two studies found no difference in sensory gating at the P50 for individuals with ASD as compared to neurotypical individuals (i.e. typically developing children) (Kemner, Oranje, Verbaten & van Engeland, 2002; Orekhova et al., 2008), one study found the opposite (Madseon et al., 2015). In addition to differences in sensory gating, differences in latencies have also been found in children with ASD, with results of both abnormally short and abnormally long latencies being reported (Mei et al., 2014; Orekhova et al., 2008). Overall, the findings related to P50 gating in individuals with autism have been inconsistent in terms of both amplitude and latency.

N1 Component and Sensory Gating

Current research suggests that the N1 component relates to functional attention (Lijffijt et al., 2009; Phelan, 2012), sensitivity to touch (Griefe, 2013), learning (Lijffijt et al., 2009), and the function of sensory gating (Boutros, Korzyukov, Jansen, Feingold, & Bell, 2004; Griefe, 2013; Lijffijt et al., 2009; Rentzsch, Jockers-Scherübl, Boutros, & Gallinat, 2008). One study found the N1 component to be an even stronger indicator of sensory gating than the P50 component (Boutros, Belger, Campbell, D'Souza, & Krystal, 1999). In a dissertation by Crasta (2017) the results indicated that adults with ASD do not have significantly different sensory gating at the N1 component than their neurotypical peers.

Several studies examining the ERPs of individuals with ASD have focused on the N1 component, but the findings have been variable. One study with children participants found no significant difference in the N1 component of the ASD group compared to the control group

(Kemner, Verbaten, Cuperus, Camfferman, & Engeland, 1995). Two studies had the same findings in that children with ASD had both longer latencies and smaller amplitudes at the N1 compared to control groups (Bruneau, Roux, Adrien, & Barthélémy, 1999; Seri, Cerquiglini, Pisani, & Curatolo, 1999) and a third also found significantly longer latencies for adults with ASD (Crasta, 2017); while another study of children found that the ASD group had a significantly shorter latency period at the N1 (Ferri et al., 2003). Overall it appears that there are significant differences between individuals with ASD and control groups at the N1 component, but that these differences vary among lengths of latency and size of amplitude.

P2 Component

The P2 component has not been the focus of many studies as it has historically been considered a combined component with the N1 component (Crowley & Colrain, 2004). This viewpoint has changed over recent years, and research has shown that it is indeed an independent component with unique features related to sleep, attention, and enhancement across the adult life span (Crowley & Colrain, 2004). The P2 component has also been found to have connections with working memory, learning, and attention (Lijffijt et al., 2009), as well as multisensory processing (Balconi & Carrera, 2011). The P2 is thought to represent the first moment of the person being consciously aware of a stimulus (with previous processing components occurring subconsciously; Lijffijt et al., 2009) and is also representative of the brain's ability to rapidly access and code information in response to auditory stimulus (Tong, Melara, & Rao, 2009). The P2 component has not been explored in comparative groups of people with ASD and without, but it has been shown to have a significant role in sensory gating with strong test-retest reliability in neurotypical adults (Rentzsch, Jockers-Scherübl, Boutros, & Gallinat, 2008).

N2 Component

Currently there is not much known about the N2 component as it relates to functional behaviors and group differences in those with ASD, including any studies reporting on sensory gating at the N2 for individuals with autism. There are also no reports on latency at the N2 for individuals with autism, thus this review focuses on the research in regard to amplitudes at the N2. The strongest behavioral relationship found for N2 is when functional, sustained attention is required by the participant as they perform sensory processing, as it results in a larger amplitude at the N2 (Jodo & Kayama, 1992; Phelan, 2012; Righi, Mecacci, & Viggiano, 2009). The paradigm used to elicit this sustained attention while processing sensory stimuli is called an "active condition" and will be discussed further in the following section. In addition to associations with attention, the N2 amplitude has also been found to highly correlate with increased anxiety (Righi et al., 2009). It is worth noting that approximately 40% of children and teenagers with ASD also have a diagnosis of anxiety (Van Steensel, Bögels, & Perrin, 2011).

A study by Orekhova (2009) found that children with ASD had significantly smaller amplitudes at the N2 component when orienting to auditory input compared to a typically developing control group. They suggested that this deficit at the N2 component further impacted information processing at high-order cortical areas, and that it ultimately might be related to attention deficits observed through behavioral observations of children with ASD. Ultimately, the research in this area is limited, and conclusions about group differences cannot be drawn at this time.

Impact of Attention on Sensory Gating

During sensory processing the state of attention the person is in can impact their processing performance, especially in individuals with autism (Kemner et al., 1995; White &

Bishop, 2008). These attention states can be changed by what the person is doing, such as having to respond to a certain sound (active listening), or simply sitting and looking at a screen while a sound occurs in the background (passive listening). Research has shown that when people actively listen to a noise they tend to have larger amplitudes than when they are passively listen (Jodo & Kayama, 1992; Paz-Caballero & García-Austt, 1992). One study found that both environmental distractions and internal attention states have measurable impacts on both the amplitude and latency of ERPs during auditory processing (Crasta, 2017). In studying sensory gating these two states of attention (active and passive) can be used as conditions which can then be compared to one another. A passive condition is one in which the participant sits and passively listens to the presented stimulus, often while looking at an image on a screen. An active condition is one in which there is a task demand on the participant, such as counting the number of special tones they hear or hitting a button when they hear a special tone.

Purpose

This study aims to fill the gap in the research pertaining to individuals with ASD and their response to auditory input in terms of both sensory gating and latency. We also aim to add to the limited literature regarding how attention states impact latency and sensory gating for both individuals with ASD and neurotypical individuals. Overall, the literature has revealed inconsistent findings regarding amplitudes, latency, and sensory gating in ASD groups compared to neurotypical groups. This study will analyze measurements of both within group and between group differences to create broad, informative results regarding samples from both populations. By looking at attention states, sensory gating, and latency, this study will further define what differences, if any, exist between individuals with ASD and neurotypical individuals in terms of sensory processing as a neurological function. By furthering the understanding of these processes

and what deficits might be occurring, the results may guide recommendations for practitioners regarding what interventions are needed and how they might best be applied.

CHAPTER 2 – METHODS AND RESULTS

Autism and Sensory Processing

The Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) outlines the current criteria for the diagnosis of Autism Spectrum Disorder (ASD) as including deficits in two main areas: communication and relationships, and restricted and repetitive behaviors. (American Psychiatric Association, 2013). The second set of criteria, restricted and repetitive behaviors, includes reactions that are both excessive or limited in response to sensory input. Sensory Processing Disorders can occur in any individual, but are found to occur at a higher rate for people with autism (Davies & Tucker, 2010) and can further impact the person's ability to communicate and participate in their physical and social environments. Research has shown that those with a diagnosis of ASD tend to have dysfunction in at least one of their sensory processing systems (Minshew & Hobson, 2008; Su & Parham, 2014), and that for 77.6% of children with autism the auditory system is impacted (Tomchek & Dunn, 2007). Deficits processing auditory stimuli can result in observed behaviors of the child "tuning out" someone who is speaking, struggling to perform when there are auditory distractions or background noise, or being over-sensitive to certain sounds.

Impact of Attention on Sensory Gating

Attention states have been shown to affect sensory processing on a neural level, for both neurotypical individuals and people with ASD (Jodo & Kayama, 1992; Kemner et al., 1995; White & Bishop, 2008). The two attention states that this study used are called active and passive. An active attention state occurs when a person is required to provide a response to the stimulus that they are processing, such as when a person is playing a musical piece with a group

of people. A passive attention state occurs when the person is not providing a response to the stimulus they are experiencing, such as when a person is simply listening to music. Research has shown that active attention states tend to increase the amplitudes when compared to passive attention states in neurotypical individuals (Jodo & Kayama, 1992; Paz-Caballero & García-Austt, 1992).

Three studies have looked at the differences in ERP amplitudes of passive and active conditions for individuals with autism during auditory processing and found strikingly similar results. Kemner et al. (1995) found that children with autism had significantly larger amplitudes at the P3 component during active conditions compared to their own P3 amplitudes during the passive condition, while the typically developing control group did not have a significant difference in amplitudes between the active and passive condition. White and Bishop (2008) found that children with autism had reduced amplitudes at the P1 and N2 components during the passive condition compared to the typically developing control group, but when performing in an active condition did not have significantly different amplitudes from the control group at any components. Crasta (2017) found that in an active condition, amplitudes throughout the entire stream of auditory processing were increased compared to the amplitudes in the passive condition for adults in both the ASD and control group. This study also found fewer group differences between the ASD group and control group during the active condition than the passive condition (with the ASD group under-performing during the passive condition). These findings suggest that using active and passive conditions causes a significantly different outcome for individuals with autism during sensory processing, to the point that it might affect whether a significance difference occurs compared to the control group.

Purpose

The literature shows both a lack of research on sensory gating in individuals with autism at the N1, P2, and N2 components, and that when the studies on the topic do exist their findings are inconsistent. A possible reason for these inconsistences might be due in part to the fact that many of the studies used child participants. Research has shown that sensory processing is a developing system until sometime between 6 to 15 years of age with variation depending on the systems and the individual child (Cheung & Siu, 2009; Peterson, Christou, & Rosengren, 2006). These variations in sensory processing across development, may be contributing to inconsistent findings because studies use participants at different ages ranges. This study seeks to fill the gap in the literature by providing further observations on sensory gating in young adults with autism at the N1, P2, and N2 ERP components, as well as providing group comparisons with neurotypical adults. Young adults were used in this study as they should all have sensory systems that have completed development, thus limiting the possibility of development of sensory systems confounding results of the study. Because there have been inconsistent findings of differences in measurements of both amplitudes and latencies between ASD groups and neurotypical groups, this study analyzed both factors, with amplitudes used to measure sensory gating, and latencies to measure processing speed. This study used states of both active attention and passive attention in participants since these differences in attention have been shown to produce significantly different results in ASD groups, as well as in control groups. These differences require further exploration and comparisons both within groups and between groups.

By looking at factors of sensory gating (amplitudes), latency, and passive and active conditions this study hopes to further describe what differences, if any, exist in sensory processing at the neural level between individuals with ASD and neurotypical individuals.

Further understanding of these processes can inform how daily functioning is affected by sensory processing for individuals with ASD, and thus inform practitioners of how interventions might be most effectively directed. In addition, a search for ways to define and thus diagnose ASD through endophenotypes (physical, genetic markers that explain observable behaviors) is ongoing, and could include unique amplitude or latency differences at specific ERP components during sensory processing in individuals with ASD (Jeste & Nelson, 2009). The findings of this study will add to the existing literature to create a more consistent understanding of the amplitudes and latencies of the N1, P2, and N2 ERP components during a sensory gating paradigm in individuals with ASD.

Research Questions and Hypotheses

Research Question 1: Do adults with ASD exhibit slower processing speed as measured by longer latency of specific ERP components (i.e., N1, P2, and N2) than neurotypical peers as measured by the ERP components in both active and passive conditions?

Hypothesis 1: We hypothesize that adults with ASD will have significantly slower processing speed as measured by longer latency periods than neurotypical peers at each ERP component in both active and passive conditions.

Research Question 2: Do adults with ASD exhibit less robust sensory gating than neurotypical peers as measured by N1, P2, and N2 ERP components in a passive condition?

Hypothesis 2a: We hypothesize that adults with ASD will have significantly less robust gating than neurotypical peers at all three ERP components in a passive condition as measured by a difference score between click 1 and click 2.

Hypothesis 2b: We hypothesize that adults with ASD will have significantly less robust gating than neurotypical peers at all three ERP components in a passive condition as measured by a t/c ratio between click 1 and click 2.

Research Question 3: Do adults with ASD exhibit less robust sensory gating than neurotypical peers as measured by N1, P2, and N2 ERP components in an active condition?

Hypothesis 3a: We hypothesize that adults with ASD will have significantly less robust gating than neurotypical peers at all three ERP components in an active condition as measured by a difference score between click 1 and click 2.

Hypothesis 3b: We hypothesize that adults with ASD will have significantly less robust gating than neurotypical peers at all three ERP components in an active condition as measured by a t/c ratio between click 1 and click 2.

Research Question 4: Do adults with ASD and their neurotypical peers show less robust gating in the active versus passive listening conditions?

Hypothesis 4a: We hypothesize that for the neurotypical group the active condition will result in significantly less robust gating than the passive condition.

Hypothesis 4b: We hypothesize that in the ASD group the active condition will result in significantly less robust gating than the passive condition.

Research Question 5: Do adults with ASD experience greater differences in gating between the active and passive conditions than neurotypical adults do?

Hypothesis 5: We hypothesize that adults with ASD will have greater within-groups differences between the active and passive conditions during sensory gating than neurotypical adults do.

Methods

Participants

This study included 24 neurotypical participants, ages 18-29, (12 males, 12 females; M = 23.7 years, SD = 3.5) and 24 participants with ASD, ages 17-30 (17 males, 7 females; M = 23.3 years, SD = 3.8). There is no group difference in age, $t_{(46)} = .41$, $p = .69$. Participants were recruited using a convenience sample which included word-of-mouth, social media, and fliers posted throughout the university and local community. Participants were categorized into the neurotypical group by self-report of no major diagnoses or conditions and by filling out a screening questionnaire which ensured that they were free of neurological injuries, disabilities, and family histories of psychological disorders. Participants who reported a diagnosis of ASD were administered the Autism Diagnoses Observation Schedule (ADOS) by a graduate research student who is certified in the administration of the ADOS (Lord, Rutter, DiLavore, Risi, 1999). All participants that reported a diagnosis of ASD scored in the autism percentile on the ADOS and were thus included in the ASD group. One participant from the ASD group was excluded from the analyses and results as he was unable to finish the paradigm due to difficulties associated with his diagnosis. All the procedures performed in the research involving human participants were approved by Colorado State University IRB. All participants signed a written informed consent prior to the study.

Data Collection

Procedures. Upon arrival for the EEG visit, participants were given a verbal and visual explanation of the EEG process. Once the EEG cap and electrodes were applied, participants were shown their real-time brainwaves on the computer and given a brief training on strategies they could use to minimize eye blink and movement artifacts in the recordings. EEG was then

recorded to establish baseline metrics for each participant as they passively looked forward at a computer screen. Following this, EEG data were collected using the sensory gating paradigm. Participants also completed a second EEG paradigm, but these data are not reported here. Each paradigm lasted about 20 minutes, with breaks of one minute between each paradigm. The entire visit lasted about one hour.

EEG data acquisition. EEG data were collected in a sound attenuated and electrically shielded room which reduces environmental artifacts. Recordings were collected using a 64-channel BioSemi ActiveTwo -EEG/ERP Acquisition System (BioSemi, WG-Plein 129, 1054 SC Amsterdam, Netherlands. This system included 64 Ag/AgCl sintered scalp electrodes. The electrodes were located in accordance with the 10–20 system (American Electroencephalographic Society, 1994). EEG was recorded using the Common Mode Sense active electrode as the reference and the Driven Right Leg passive electrode as the ground (http://www.biosemi.com/faq/cms&drl.htm). Electrooculograms (EOGs) were recorded from individual electrodes placed on the left and right outer canthus for horizontal eye movements and on the left supraorbital and infraobital region for vertical eye movements. Two more individual electrodes were placed on the left and right earlobes to be used as the offline reference, an average of the two electrodes. The data were sampled at a rate of 2048 Hz with a bandwidth of 0 to 417 Hz. Of the 64 channels, the central site Cz was analyzed to maintain consistency with previous research.

Sensory gating paradigm. The sensory gating paradigm was presented using E-Prime software (Psychological Software Tools, Pittsburg, PA) running on a laptop computer. A modified sensory gating paradigm was used which consists of presentations of 80 pairs of click stimuli and 40 randomly interspersed single clicks. The click stimuli were binaurally presented

through the ER-3A inserted earphones (Etymotic Research). Each click had a 4 ms duration. The paired click stimuli were presented with a 500 ms stimulus onset asynchrony (SOA), and an 8 second inter-trial-interval between pairs. The clicks were played at 85 dB SPL (decibels sound pressure level) and a practice test of the paradigm was performed the ensure that the participant could hear the clicks adequately. Participants completed blocks in both the passive and active conditions. For each attention condition, the stimuli were presented in two blocks of 60 trials each. Each block was about 8 minutes with approximately 1 minute of break between blocks. The two conditions in this study were as follows:

The passive condition. In this condition, participants were asked to watch the computer screen with a fixation point while they were being presented with click sounds.

***The active condition*.** In this condition, participants were asked to selectively respond to single clicks with a button press and ignore the paired-clicks while watching the computer screen with a fixation point.

ERP Waveform and Component Analysis – Data Reduction Procedures.

Brain Vision Analyzer software (Brain Products GmbH, München, Germany) was used to conduct all offline EEG analyses. Baseline-to-peak measures for the N1, P2, and N2 components were determined using previously established methods (Davies et al., 2009; Boutros et al., 2004). Averaged ERPs were composed from the running EEG data. First, the four EOG channels were converted to a vertical and a horizontal bipolar EOG. For the N1, P2, and N2 components, data were filtered offline from the continuous EEG with a band pass setting of .1 to 30 Hz (24 dB/octave; Boutros et al., 2004; Lijffijt et al., 2009). Next, data were segmented time-locked to the stimulus onset into epochs representing either the conditioning or test click with a duration of 100 ms pre-stimulus onset to 500 ms post-stimulus onset. Baseline correction

relative to a baseline of -100 ms to 0 ms was performed. An eye regression technique designed to remove eye movement from trials was then performed (Segalowitz, 1996). Baseline correction was performed again relative to a baseline of -100 ms to 0 ms for the non-rejected segments. For ERP data reduction, segments with voltage deviations greater than ±100 microvolts (μV) on any of the EEG channels or the bipolar EOG channels was eliminated. Averages were then taken from the segments to create ERP waveforms for both click 1 and click 2 in order to measure the ERP components for each participant.

The N1 peak was scored between 70 and 140 ms after the stimulus onset, the P2 between 120 and 280 ms, and the N2 between 155 and 395 ms after stimulus onset. Test/conditioning (T/C) ratio and difference scores were computed to evaluate gating abilities. A T/C ratio near zero is considered very robust, while a T/C ratio near 1.0 demonstrates a lack of gating. A T/C ratio above 1.0 would indicate that the amplitude of click 2 was larger than the amplitude of click 1 (Cromwell, Mears, Wan, & Boutros, 2008). Difference scores were calculated by subtracting the amplitude of the second click from the amplitude of the first click. For the positive component, P2, a large positive difference score indicates better gating. For the negative components (N1 and N2), a larger negative difference indicates better gating.

Statistical Analyses

To test hypothesis 1, latency was measured in milliseconds from the onset of the stimulus to the highest peak in amplitude for the component being analyzed. Then, an ANOVA comparing (Group) x (Attention[Latency]) was conducted. An Independent T-Test was conducted to test hypotheses 2a, 2b, 3a, and 3b. A Paired Sample T-Test was used to test hypotheses 4a and 4b. Group differences between active and passive conditions were calculated, then an Independent T-Test was conducted to test hypothesis 5. The main effect of attention was used to determine

the impact of attention on gating, while the main effect of group was used to determine differences in gating between the ASD group and the neurotypical group (hypotheses 2a, 2b, 3a, 3b, 4a, 4b, 5). Effect sizes for η^2 were interpreted as follows, .04 – small, .25 – medium, and .64 – large (Ferguson, 2009). All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) for Windows software, 24.0 version.

Results

Latency of ERP Components- Processing Speed

The first research question addresses the differences between the groups in latency of ERP components. The means and standard deviation of the latencies of both groups in both conditions and at all three components are reported in Table 1. Overall, the ASD group tended to have longer mean latencies in both conditions at the N1, but the delays became less frequent at the P2, and even less so at the N2 (see Figures 1 and 2).

For the first research question addressing the differences in latency of ERP components or speed of processing between the groups a repeated measures ANOVA with 2 levels of group (ASD and NT) and 2 levels of attention (passive and active) was conducted. For latency at the N1 component, the main effect of attention was not significant indicating that there were no significant differences in latency between attention states across groups. The group main effect showed a significant group difference with $F_{(1, 46)} = 13.61$, $p = .001$, $\eta^2 = .23$. The ASD group had significantly longer latency times in both the active (M = 110.60) and passive conditions (M = 109.78) compared to the NT group (active condition M = 98.60; passive condition M = 99.81). There was no significant difference for the attention by group interaction.

For latency at the P2 component, the main effect of attention was significant, $F_{(1, 46)} = 8.23$, $p = .006$, $\eta^2 = .152$ showing a significant difference between the attention states with a

small effect size. The latency (for both groups combined) in the passive condition (M = 192.68) was significantly longer than in the active condition (M = 180.78). There was no significant main effect for groups or for the attention by group interaction.

For latency at the N2, the main effect of attention was $F_{(1, 44)} = 4.05$, $p = .050$, $\eta^2 = .084$. This shows a marginally significant difference between attention conditions, such that latency was significantly longer in the passive condition (M = 275.10) than in the active condition (M = 257.48) across groups. The main effect for group was not significant and there was no significant difference for the attention by group interaction.

Suppression of Click 2 in the Passive Condition

The second research question addresses group comparisons of gating in the passive condition through the use of Difference Scores and T/C Ratios (see Table 1, Figures 4 and 6). Independent T-Test was used to measure group differences. There were no significant differences found between the two groups such that at N1 $p = .64$, at P2 $p = .53$, and at N2 $p = .98$.

Gating at the N1 component in the passive condition was not significantly different between groups with $p = .97$. Neither was it significant at the P2 ($p = .89$) nor the N2 ($p = .54$) components in the passive condition.

Suppression of Click 2 in the Active Condition

The third research question addresses group comparisons of gating in the active condition (see Figures 3 and 5). Group differences were measured using and Independent T-Test. First, a difference score was used to measure gating by subtracting Click 2 from Click 1 (see Table 1). Gating at the N1 in the active condition showed no significant group differences between the

ASD and NT groups with $p = .10$. Gating at the P2 and N2 components also showed no significant group differences with $p = .16$ and $p = .77$.

Next, gating was measured using a T/C ratio of Click 2 divided by Click 1. Gating at the N1 component in the active condition was not significantly different between groups with $p = .20$. Neither was it significant at the P2 ($p = .25$) nor the N2 ($p = .56$) components in the active condition.

Effect of Attention on Gating

First, the fourth research question was addressed by looking at groups individually to test the impact of attention states on sensory gating. These findings were measured using a Paired-Sample T-Test. For the NT group (see Figures 5 and 6) at the N1 component, there was significantly more gating in the passive condition (difference score mean = -4.5) than the active condition (M = .3) with $t(23) = 5.85$, $p < .005$. For the NT group at the P2 component there is again significantly more gating in the passive condition (M = 4.3) than the active (M = .44) with $t(20) = -3.44$, $p = .003$. At the N2 component the difference between attention conditions is approaching significance, but does not meet statistical significance with $t(20) = -1.90$, $p = .073$ While the differences are not significant at the N2 component, it is observed that there is a larger difference score, thus more gating, in the active condition (M = -3.16) than the passive (M = -.34), which is the reverse of the observations made at the previous two components.

For the ASD group (see Figures 3 and 4) at the N1 component there is also a significantly more gating in the passive condition (M = -4.97) than the active (M = -1.24) with $t(23) = 4.34$, $p < .005$. At the P2 component there is significantly more gating in the passive condition (M = 5.19) than the active condition (M = 2.47) with $t(23) = -2.42$, $p = .024$ Lastly, the ASD group

has significant difference between conditions at the N2 component $t(19) = -2.36$, $p = .029$ and, similarly to the NT group, it is in the opposite direction of the previous components with more gating in the active condition (M = -2.71) than the passive (M = .26). Implications of these findings will be explored in the Discussion section.

The last analysis performed addresses research question five by measuring group differences between the NT and ASD groups in their differences in levels of gating between active and passive conditions (see Figures 3, 4, 5, and 6). An Independent T-Test showed that there are no significant differences between the groups and their differences in gating in the different attention states at the N1 component with $p = .37$. Similar results were found for the following two components, with no significant difference between groups at the P2 ($p = .48$) and N2 ($p = .94$).

Discussion

At all components analyzed (N1, P2, N2) latency was significantly delayed for the ASD group compared to the neurotypical group when having an orienting response at Click 1. This significant delay was also seen in both attention states at the N1 and P2 components, but only seen in the active condition at the N2. These results show that, overall, people with ASD have slower processing during an orienting response compared to their neurotypical peers. Previous research has reported on significantly longer (Bruneau, Roux, Adrien, & Barthélémy, 1999; Crasta, 2017; Seri, Cerquiglini, Pisani, & Curatolo, 1999) latencies at the N1 component for individuals with autism compared to neurotypical peers, and our results further corroborate the multiple studies showing delayed processing speed at this component. No other studies at this time have reported on latency periods for individuals with autism at the P2 and N2 components, thus the results of this study are the first to report that, while the ASD group had slower

processing speeds overall, they were not significantly different from the NT group at these later components. At the P2 and N2 components, latency was significantly slower in the passive condition than the active for the combined groups. These results suggest that by having attention focused on a stimulus, with the necessity of attention for directing a response, processing speeds for both adults with autism and neurotypical adults will increase.

Studies have reported on the possibility of delayed latency in response to an auditory stimulus having potential use as a biomarker for ASD (Gandal et al., 2010; Roberts et al., 2010). The results of our study add to the literature supporting delayed latency at the N1 component as a pattern of ASD response to auditory stimulus. Yet another study noted the difference in auditory processing performance for individuals with ASD using a functional assessment (the Sensory Profile [Dunn, 1999]) and suggested that this difference is so strong that the observation of deficits in responding to auditory stimulus should be included in the current diagnostic criteria (Dunn, Myles, & Orr, 2002). Studies that have used the Sensory Profile have shown that children with ASD tend to have the largest group difference with their neurotypical peers in the sensory system of auditory processing (Ashburner, Ziviani, & Rodger, 2008; Dunn, Myles, & Orr, 2002). As our results show no difference in gating between the groups, but do show significantly slower latency for individuals with autism at the N1 component, it is possible that it is latency delays that are causing the functional deficits, as opposed to deficits with gating of repeated stimulus. As Quill's (1997) review of the literature on educational instruction for autism showed that individuals with autism perform better on cognitive and instructional tasks when they are presented in a manner that uses stable, un-moving visual cues in place of auditory stimuli. Quill went on to theorize that the necessity of processing auditory information in an instantaneous manner is what caused the deficit in processing auditory information, in

comparison to static visual information, which can be examined as long as is necessary for processing.

Overall, no significant group differences were found in any analysis of gating. While the groups compared to one another were not significantly different, both groups were significantly impacted by attention conditions in the same direction. For the first two components analyzed, N1 and P2, both groups showed significantly more gating in the passive condition. This difference is expected, as the active condition required participants to listen for the second click because they were required to press a button for single click (and hit a button in response to it). This requires more processing of the conditioning click and thus increases the amplitude of that ERP component, thus reducing the difference in amplitudes between clicks. These results support other studies' findings of overall larger amplitudes resulting from an active attention state and that a response demand on the person mitigates the sensory gating effect of suppressing responses to repetitive, incoming stimuli. In functional interventions these findings can inform practitioners on how they can manipulate attention demands to either suppress or support a gating response. If seeking a higher processing response where it is not desired for the client to filter out noise, the practitioner can create a required response based on the stimulus being used to create an active attention state to mitigate sensory gating, or the filtering out of perceived irrelevant stimuli. To support a gating response a practitioner can instead create a passive attention state where the stimulus does not require any type of response and creates no attentional demands.

Interestingly, these findings at the N1 and P2 do not carry forward through the N2 response. In fact, a significant response in the opposite direction is seen: both the ASD and the NT group displayed significantly more gating in the active condition than the passive at the N2

component. It is not immediately clear why this response would be flipped at this later component. While the research on this component as a sensory gating function is limited, some of the literature does highlight the unique features of the N2 component. In a thesis by Phelan (2012) a similar result of unexpected, opposite outcomes at the N2 was found, wherein the N2 had larger amplitudes in the active condition at Click 1 than they did in the passive condition at Click 1. A review of the research shows that N2 has a relationship with inhibition, in quickly classifying and choosing to dismiss certain stimuli (Jodo & Kamaya, 1992; Phelan 2012; Righi, Mecacci, & Viggiano, 2009). The amplitude of the N2 tends to be larger when in the process of inhibiting a planned response, which would occur in active attention conditions when the participant is deciding not to respond to Click 1 in order to prepare for the possibility of a second click. It is possible that, because the participant is not required to respond to Click 2, there is a smaller amplitude from the N2 since it is not having to perform an inhibitory function. Thus, in an active condition a large difference in amplitude occurs between the response to Click 1 and Click 2 as the N2 component is performing higher processing through its inhibitory function at Click 1, and performing with less processing at Click 2 where there is no need for inhibition. Thus, this process results in an effect that is similar to gating, as there is a significant difference between Click 1 and Click 2. While this theory and review of the research provides an idea for why there is more gating in the active condition at N2, further research should be performed to better understand this neural process.

Limitations

Participants in this study were recruited using a convenience sample and most came from a local university campus which limits the ability to apply the findings to a greater population. In addition, with only 24 participants per group, there was limited power for some of the results.

Specifically, the findings of how attention impacted gating in the NT group which resulted in p = .073, and the finding that attention impacted latency at the N2 which resulted in p =.05 could have been strengthened and possibly resulted in increased significance with more participants. Overall, the majority of the analyses had ample power based on the sample size. The findings in this study indicate novel findings regarding the N2 which calls upon the need for replication and similar research studies to corroborate or further inform the knowledge on this topic. Future research can strengthen findings by controlling for other factors such as intelligence and functional attention abilities.

Conclusion

No group differences between the ASD and NT groups were observed for sensory gating measures. However, both groups were observed to have significantly more sensory gating in the passive condition at the N1 and P2 components, while having significantly more sensory gating in the active condition at the N2. This is possibly related to other research which indicates that the N2 is strongly associated with an inhibitory function that occurs in response to Click 1, but not Click 2, in the active condition. Latency at click 1 was significantly delayed for the ASD group at the N1 component, and overall was slower for the ASD group in all conditions at all components. These results indicate that differences in observed auditory processing for individuals with autism are possibly more related to delays in processing speeds, rather than deficits in sensory gating. Implications for practice support the recommendation of using more visual cues than auditory for individuals with autism, and also allowing more time for processing auditory information. Future research can add to these findings by further exploring stimulus responses at the N2 component in active and passive conditions, as well as testing the effects of interventions on latency speeds for individuals with autism.

Table 1: Amplitudes, Latencies, T/C Ratios, and Differences Scores for NT and ASD Groups

	Group							
	NT				ASD			
	Attention conditions				Attention Conditions			
	Passive		Active		Passive		Active	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
N1 Amplitude								
Click 1	-8.08	3.40	-6.83	3.33	-8.12	5.03	-7.69	4.57
Click 2	-3.58	2.21	-8.08	3.40	-3.15	2.84	-6.45	4.07
N1 Latency								
Click 1	99.81	10.32	98.69	10.88	109.78	11.85	110.60	11.90
Click 2	91.51	13.59	101.97	22.17	102.95	20.50	105.61	17.70
N1 Diff Score	-4.50	3.25	.30	2.31	-4.97	3.67	-1.24	3.85
N1 T/C Ratio	.46	.26	1.07	.40	.46	.37	.88	.57
P2 Amplitude								
Click 1	8.91	5.10	5.86	3.34	8.53	5.77	6.78	4.37
Click 2	4.66	2.54	5.85	3.89	3.34	2.55	4.31	5.02
P2 Latency								
Click 1	188.62	26.23	175.86	24.89	196.74	31.42	185.71	20.07
Click 2	189.43	33.87	206.45	28.60	182.47	37.74	193.46	37.30
P2 Diff Score	4.30	5.55	.44	4.51	5.19	4.87	2.47	4.88
P2 T/C Ratio	56	.28	1.23	1.66	.54	.45	.77	.66
N2 Amplitude								
Click 1	-.19	4.50	-.85	3.44	-.61	3.42	-2.01	3.23
Click 2	.05	2.43	2.70	4.11	-.34	2.34	.74	3.36
N2 Latency								
Click 1	275.29	49.28	251.38	37.15	274.9	55.06	264.14	35.74
Click 2	265.44	53.36	253.51	39.06	251.77	51.37	236.13	48.99
N2 Diff Score	-.34	4.14	-3.16	5.18	.26	3.41	-2.71	4.68
N2 T/C Ratio	.97	1.2	2.31	4.11	.79	.72	1.64	3.22

Key: NT = Neurotypical, ASD = Autism Spectrum Disorder, M = Mean, SD = Standard deviation, Diff Score = Difference score

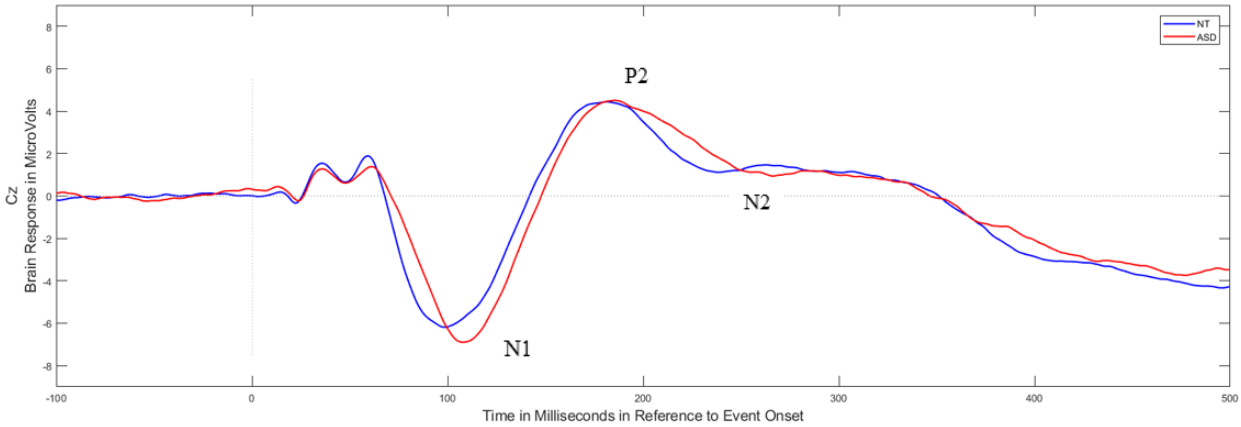

Figure 1: Latency Response to Click 1 in the Active Condition

Key: NT = Neurotypical, ASD = Autism Spectrum Disorder

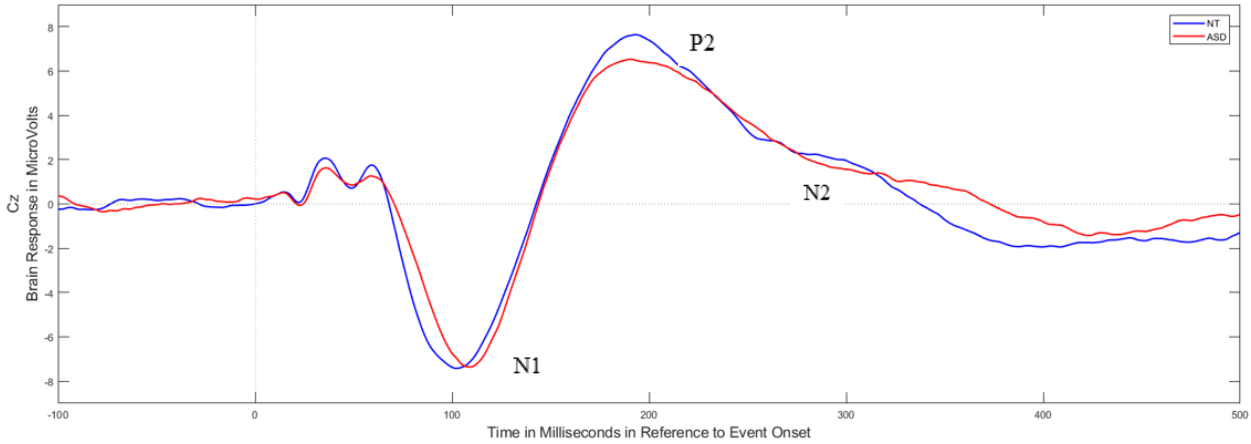

Figure 2: Latency Response to Click 1 in the Passive Condition

Key: NT = Neurotypical, ASD = Autism Spectrum Disorder

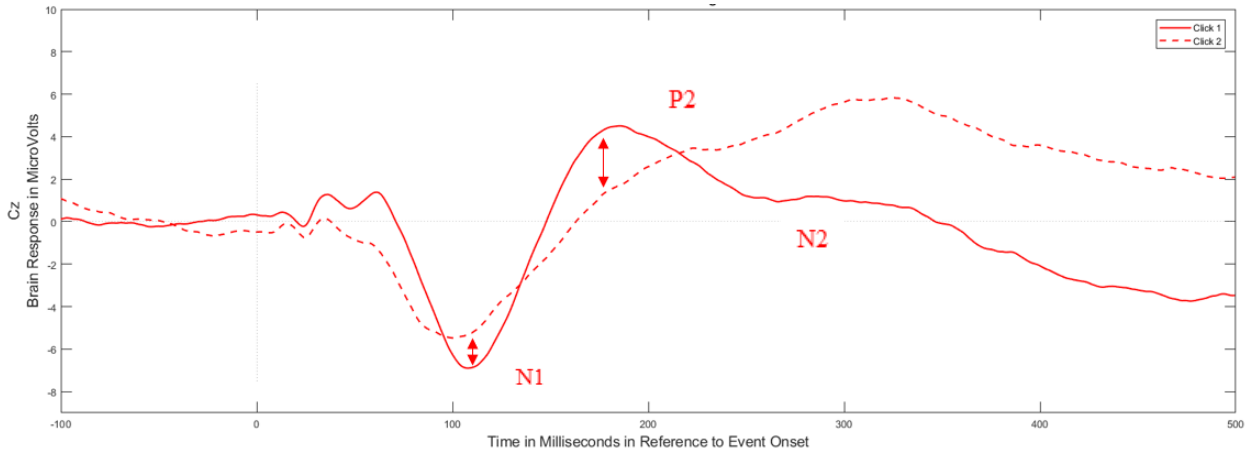

Figure 3: Autism Spectrum Disorder Group Sensory Gating in the Active Condition

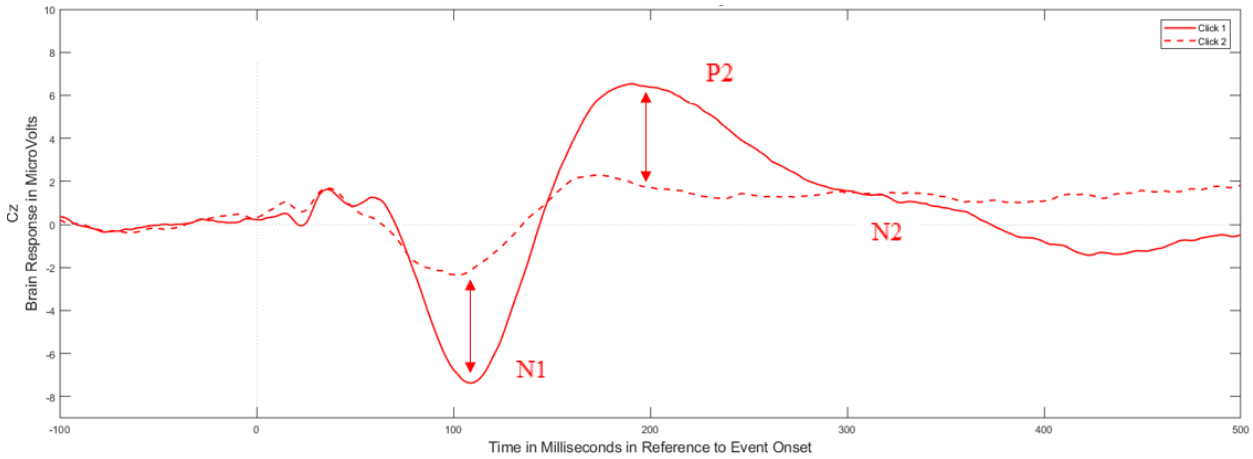

Figure 4: Autism Spectrum Disorder Group Sensory Gating in the Passive Condition

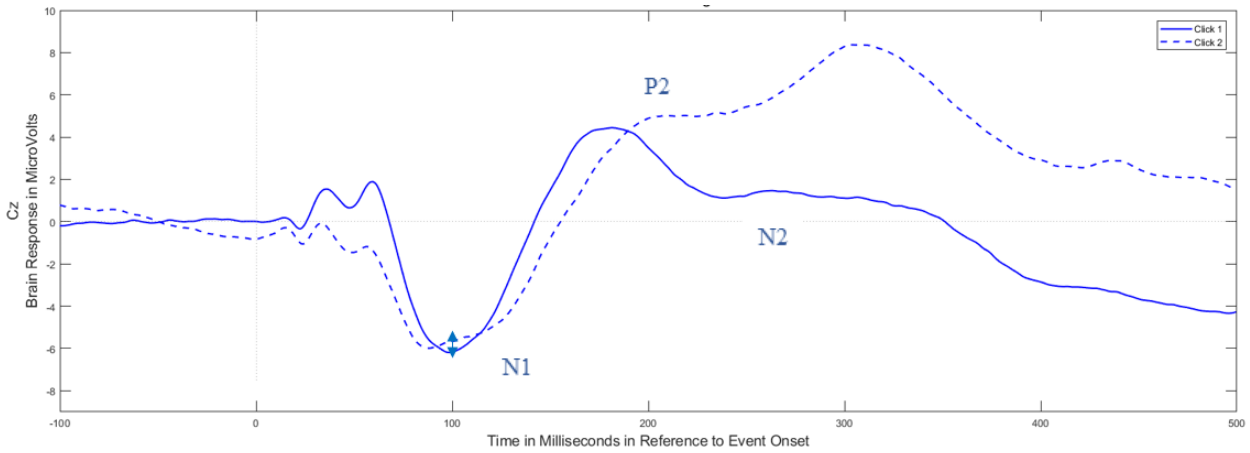

Figure 5: Neurotypical Group Sensory Gating in the Active Condition

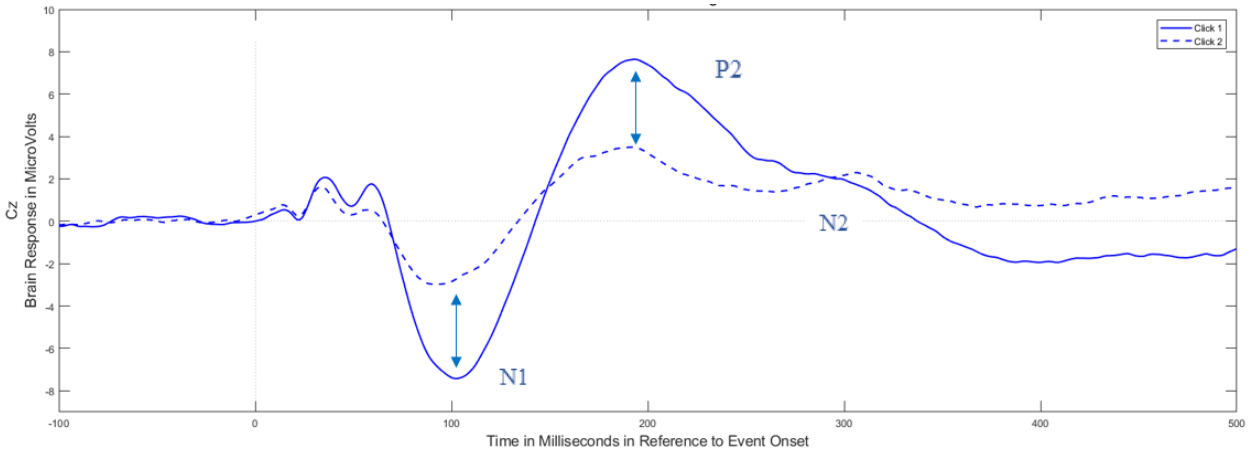

Figure 6: Neurotypical Group Sensory Gating in the Passive Condition

CHAPTER 3 – CONNECTION TO OCCUPATIONAL THERAPY

Person, Environment, Occupation Model

Occupational therapy is informed by many theories and ideas, but perhaps one of the most prominent is that of the Person, Environment, Occupation (PEO) Model by Law et al. (1996). This perspective of analyzing a situation is a piece of what makes occupational therapy a unique and distinct field. Sensory integration is another theory that is deeply rooted in occupational therapy, and one to which we can apply the PEO model. In fact, when an occupational therapist (OT) is working with an individual who is experiencing barriers to their participation and performance due to sensory processing deficits, changes in each element of the PEO are often considered. For example, if a child is struggling to focus in a classroom, a therapist might consider changing the occupation by training the teacher in providing the lessons in a different way; they might consider changing the environment by shutting the classroom door or providing headphones; or they might consider changing the person by providing sensory interventions that decreases the child's response to auditory stimuli. In many cases, an OT will use all three PEO elements to provide an intervention.

The research performed in this study further informs the decisions an occupational therapist might make when designing a sensory intervention. It provides information on the way that the occupation can be changed through use of attentional demands to create a response that either supports or hinders the filtering out of stimuli. For example, an OT can collaborate with a teacher on how to make their lessons interactive, requiring a response from the child throughout the information delineation, which will make the child less likely to "filter out" the teacher's voice. It also provides information on environmental changes that can be used by the findings

that processing speed of auditory information is slower for individuals with ASD, which adds to the body of literature on latency deficits within this population. Knowing that these deficits are neurological in nature supports compensatory strategies, such as slowing down the rate of auditory information being provided, and reducing extraneous auditory information, such as music or playground noise through open windows. The findings also support interventions that target change within the person. While future research is necessary to see if occupational therapy is able to change a person's ability to process sensory information on a neural level, the findings of this study support targeting the neurological system as a source of sensory processing deficits, and adapting both environments and occupations the further create change within the person and their real-time processing. In addition, the furthering of evidence that supports neural latency as a biomarker for autism provides the opportunity for future occupational therapy effectiveness studies to measure changes in neural processing speed as an outcome measure.

Jurisdiction and Evidence-Based Practice

The theory and practice of sensory integration and sensory processing has its roots deep within occupational therapy (Ayres, 1969; Schaaf, & Davies, 2010). Yet, as Abbot (1988) points out, professions must always strive to maintain jurisdiction over their field, and occupational therapy is no exception. Abbot goes on to provide the term "professional power", which is a profession's ability to maintain jurisdiction on a topic that other professions are attempting to influence, and provides thoughts on the factors that leave a field vulnerable to interprofessional competition for power. These methods of interprofessional competition include using abstract knowledge to claim jurisdiction, and taking over jurisdiction of a problem in which a profession is not providing sufficient measurable results of successful outcomes. As Abbot points out, most professions have a vulnerable field of expertise, an area in which they are more likely to

experience competition for jurisdiction. For occupational therapy, sensory processing is potentially one of our most vulnerable arenas. Yet, the American Occupational Therapy Association's Centennial Vision states a view for the future in which "occupational therapy is a powerful, widely recognized, science-driven, and evidence-based profession with a globally connected and diverse workforce meeting society's occupational needs" (American Occupational Therapy Association, 2007).

By being science-drive and evidence-based we further our jurisdictional claim on our areas of practice. The more evidence we can provide on a topic, the less we expose ourselves to interprofessional competition from those areas that may use abstraction to attempt jurisdictional claim. This is true for the study of sensory processing – while we began with an abstract theory from Ayres several decades ago, we are now able to provide evidence and science-driven methods for assessments, interventions, and successful results. This study furthers our understanding, and thus our professional power over the knowledge and clinical practice of sensory processing interventions. By analyzing sensory processing on a neural level and adding high-quality research to the existing literature in a way that creates more a more reliable and consistent understanding of sensory processing for individuals with autism, this study supports occupational therapy's ability to be successful in addressing a problem in which we currently hold jurisdiction. By owning the entire process of analyzing and intervening with sensory processing, from assessment of the deficits to intervention plans, through outcomes and understanding of the greater impact is has on individual lives and society, we maintain our position as the experts on this topic.

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