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Visual Mismatch Negativity as an Indicator of Emotion Processing in Individuals with Autism Spectrum Personality Traits

A thesis submitted in partial fulfillment of the requirement for the degree of Bachelor of Science in Psychology from The College of William and Mary by Leigh Catherine Gayle

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Abstract

The primary purpose of this research was to determine how modulation of the visual mismatch negativity (vMMN) by emotion is related to autism spectrum personality traits using electroencephalography (EEG). The mismatch negativity is an ERP component that occurs in response to a deviant stimulus that interrupts a sequence of repeated, or standardized, stimuli. In the current experiment, emotionally neutral faces served as the standard stimuli and happy and sad expressions served as deviants. Additionally, a neutral expression with a green tint served as a control condition. Consistent with prior research, we anticipated that the amplitude of the MMN would be increased for emotionally salient stimuli. Extending this finding, we expected that this emotion-based amplitude sensitivity would be decreased in individuals with higher levels of autism spectrum personality traits. The results replicated earlier research and were consistent with this hypothesis. Higher levels of autistic personality traits as determined by the Adult Autism Spectrum Quotient (AQ) were associated with smaller amplitudes of the vMMN in response to happy emotional expressions. This effect was more pronounced over the right parieto-occipital cortex. Taken together this research suggests that vMMN elicited by emotional expressions can be used as an index of early emotion processing and may be related to social competency in autism.
Visual Mismatch Negativity as an Indicator of Emotion Processing in Individuals with Autism Spectrum Personality Traits

According to the National Institute of Mental Health, autism is a group of pervasive development disorders that chronically affect the brain’s normal development of social and communication skills that typically appear within the first three years of life. Analogous to Schizophrenia and a myriad of other psychological disorders, experts refer to autism as a spectrum disorder due to the varying degrees of similar symptoms that are present across individuals diagnosed with the disorder. The disorders included in the autism category are autistic disorder, Asperger’s disorder, pervasive development disorder not otherwise specified (PDD-NOS), Rett’s disorder and Childhood disintegrative disorder (CDD). Collectively, these disorders are referred to as Autism Spectrum Disorder (ASD); however, Rett’s disorder and CDD are less frequently discussed in the context of autism (NIMH, 2011). Symptoms of ASD include difficulties in pretend play, social interactions and verbal and nonverbal communication, as well as patterned or repetitive behaviors and actions such as twirling and banging of the head (“Clearing the fog,” 2011). ASD is also typically accompanied by speech and learning difficulties and rigid, inflexible routines. These social and communication deficits are most often measured by eye contact, facial expressions and body language, as well as an evaluation of the child’s relationships with peers and family members, (CDC, 2009).

Leo Kanner, an American psychiatrist, is credited to be the first to study autism after the publication of his research regarding early infantile autism titled "Autistic disturbances of affective contact," in 1943. Kanner described early infantile autism as characterized by severe social and communication deficits as well as extreme aversion to change. Hans Asperger, a German researcher, also contributed to the establishment of ASD through his work in 1944 on
“autistic psychopathy” or Asperger’s disorder (Wing & Potter, 2002). Autism has recently been a topic of much interest and study, however, from a historical perspective; autism has been present for at least a thousand years. Ancient European folklore describes “changeling children” who were socially and developmentally challenged, and thought to be non-human replacements of young children stolen from their parents. According to lore, these children were developmentally typical until the “switch”, at which point the changelings began to exhibit characteristics similar to those children diagnosed with contemporary autism. Due to the regressive qualities of ASD, the child could have appeared to have typical development and then rapidly lost their acquired social and communication skills around the age of two, giving the illusion on an entirely different, or changed, child (Wing & Potter, 2002; NIMH, 2011).

Additionally, there is evidence for the existence of ASD in late 19th century. Henry Maudsley, a British psychiatrist, chronicled what he called cases of “insanity in children,” some of which are similar to the diagnostic traits of autism (Wing & Potter, 2002). When it was first proposed, the diagnosis of autism was considered rare; however, the prevalence has been dramatically increasing to the present where six out of every thousand children have been diagnosed with some form of ASD (NIMH, 2011; “Clearing the fog,” 2011).

The epidemiology of ASD is currently unknown; however, it is most often linked to neurobiological, neurochemical and genetic abnormalities (CDC, 2009). In the 1950’s Kanner, the psychiatrist who originally described autism in the context of mental disorder, believed that it was a genetically based phenomenon. Prior to the mid-twentieth century, the principal belief, especially in the United States, was that autism was the result of defective parental behavior and a corrupted child-rearing environment. In fact, it was not until the 1960’s that professional organizations and associations regarding autism and its causes began to be formed such as the
National Autism Association and Autism Speaks. It was during this time that attention began to shift toward potential neurological causes. Out of the research done by these organizations came the currently recognized spectrum of disorders that share deficits in social interaction, communication skills, adaptive functioning and imagination, along with a tendency to perform a narrow range of repetitive activities (NIMH, 2011; Wing & Potter, 2002). Presently the development of ASD is credited to an interaction between genetic and environmental factors. Several genes have been identified as contributing to autism spectrum disorder, as have disruptions in typical neurodevelopment during the fetal period as evidenced by irregularities in cortical structures present in infancy (“Clearing the fog,” 2011). A factor to take into account when examining the epidemiology of ASD is that male children are four times more likely to have a form of ASD than females (“Clearing the fog,” 2011). The thread that ties most of the studies done on the etiology of autism together is that more research is necessary to identify the root of what causes this pervasive development disorder.

The diagnostic methods to identify ASD and the associated disorders are currently rooted in behavioral approaches. Diagnostic algorithms draw conclusions based on the observation of the child’s social and learning behaviors by both their parents, teachers and by psychiatrists (Lord & Risi, 1998). Standardized tests, such as the Autism Quotient (AQ), the Checklist for Autism in Toddlers (CHAT), Autism Spectrum Screening Questionnaire (ASSQ) have been developed for the explicit purpose of identifying autism, (Baren-Cohen et al., 2001; NIMH, 2011). These algorithmic methods draw strength from their ability to be applied to individuals at differing levels of cognitive and social development as well as to the international diagnostic criteria (Lord et al., 1997; Lord & Risi, 1998) but their weakness stems from their lack of tangible physiological and neurological markers that identify ASD and distinguish it from other
disorders. Although the behavioral methods for diagnosing autism can be successful in identifying children with ASD and with helping them to gain access to the therapy and assistance they require to develop more typically, these methods can inadvertently misdiagnose and mislabel socially eccentric children or teenagers as autistic, which is a dangerous problem. This has especially been an issue in cases of the milder form of autism, Asperger’s disorder.

Autism is currently a thoroughly discussed topic in the media because of the diagnostic methods, the proposed narrowing of the diagnosis and how these modifications will affect those currently defined as autistic. In 1958, James Anthony, acknowledged that there are not enough distinct symptoms to make up all of the separate diagnoses associated with autism (Wing & Potter, 2002). This sentiment is interesting in light of the combining and narrowing of the disorders associated with autism, (Wing & Potter, 2002). Some believe that narrowing the diagnosis will cause individuals who need therapy and support from their insurance companies to be excluded (“New autism diagnosis,” 2012; Carey, 2012), while others think that the dangers and consequences of an incorrect diagnosis can affect the way that people, especially children in their formative years, view themselves, which can cause permanent and unnecessary developmental scars (Nugent, 2012; Bahsoun, 2012). For example, Benjamin Nugent was incorrectly diagnosed with Asperger’s syndrome because he met the behavioral diagnostic criteria during adolescence (Nugent, 2012). A change in the way that autism is diagnosed could have precluded Benjamin and other socially awkward, but not deficient, young adults from being labeled as autistic during their most seminal years in terms of both academic and social intelligence. Due to the common thread of a deficit in social ability in all of the diagnoses included in ASD and a need for increased precision in the diagnostic process, a monumental step
forward for the diagnosis of autism would be identifying a physiological measure for social or emotional competency.

The social deficits associated with autism do not appear to be linked with fundamental defects in the visual system because, in contrast with the auditory processing deficits that have come to characterize ASD, individuals with autism demonstrate superior attention to visual detail, excel at pattern recognition, and have been shown to be visual learners and to use visual cues to understand instructions (Dunn, Smith Myles & Orr, 2002; Maekawa et al., 2011). Thus, one potentially useful procedure for investigating neural measures of social ability is the visual mismatch negativity paradigm (MMN) (Zhao & Li, 2006; Behrmann, Thomas & Humphreys, 2006). The mismatch negativity paradigm is a component of the event-related potential (ERP) that occurs in response to the presentation of a deviant stimulus in a sequence of repeated, or standardized, stimuli. It typically occurs 150-200 ms after a deviant stimulus is presented and lasts approximately 300 ms (Garrido, Kilner, Stephan & Friston, 2009; Näätänen, 2007). Due to the observation that this response appears despite a lack of conscious attention to the stimuli, the MMN is considered to be a pre-attentive reaction to change (Dunn et al., 2008), and additionally is indicative of the brain’s ability to automatically compare consecutive stimuli, as well as to provide electrophysiological evidence of sensory learning and perceptual accuracy (Garrido et al., 2009).

As evidenced by mismatch negativity studies that use distracter tasks in both the auditory and visual modalities, the MMN is characterized as an involuntary switching of attention. The benefit of looking at an ERP component that occurs as a result of a preconscious phenomenon is that it is uninhibited by cognition or capacity for attention. The idea that the MMN component is an unconscious, or preconscious, measure of change detection that is engrained in our brain is
evidenced by the discovery of a MMN ERP component in infants (Cheour et al., 2000) and even in comatose patients (Fischer et al., 2010; Holeckova et al., 2008). In the study of autism a preconscious paradigm is advantageous because the participant’s level of cognition or their developmental status will not affect the outcome of the experiment. This means that even if the participant has moderate to severe autism and is non-verbal or has a severe learning disability, the MMN ERP will still appear and can be used for comparisons between subjects.

Näätänen, Gaillard and Mäntysalo first discussed the mismatch negativity component in the context of the auditory modality in 1978. They described an ERP that occurred in the temporal region on the brain in response to a deviant auditory stimulus when it interrupted a long sequence of repeated stimuli. Further research into the mismatch negativity paradigm involved the possible existence of a MMN ERP in the visual modality (vMMN). Though its existence was originally questioned, there are now many studies confirming the mismatch negativity in the visual modality. Maekawa et al. (2005) performed a study using black and white windmill patterns to elicit a vMMN response in the parieto-occipital and infero-temporal cortices. Czigler, Weisz and Winkler (2006) used red and green checkerboards to examine how the brain processes violations of regulation, and found similarly that there was a vMMN ERP following a deviant stimulus occurring around 140ms at the occipital and parieto-temporal areas of the brain. As a follow up to that study, Czigler and Sulykos (2010) published a study that continued Czigler’s previous research on the visual mismatch negativity, but as an alternative to the checkerboard stimuli, they used bars presented in different spatial orientations. This research again supported the finding that there is a visual component to the mismatch negativity ERP (Czigler & Sulykos, 2010).
The visual mismatch negativity has been identified in response to deviances in color, luminance, image contrast, orientation, direction of motion, and spatial frequencies, (Stagg, Hindley, Tales & Butler, 2004; Li et al., 2012), as well as increasingly more complex visual stimuli such as emotional images or expressions. Variations of the vMMN task have been performed using pictures that elicit emotional responses. In these studies, a neutral picture, meaning one that elicits no emotional response, is the standard stimulus, and pleasant or unpleasant pictures that have previously been shown to induce either positive or negative emotions are the deviants. These experiments examine the participant’s unconscious, involuntary reaction to change in emotional valence (Delplanque et al., 2004; Delplanque et al., 2005; Kayser et al., 2000).

Zhao and Li (2006) demonstrated that the brain discriminates between emotional expressions independently from attention in participants with typical development. They created a mismatch negativity task using neutral faces as the standard, repeated, stimuli and faces depicting happy and sad expressions as the interrupting stimuli. The participants viewed trial blocks of these faces passively while performing an auditory distracter task where they focused on identifying and discriminating between tones. This distracter task was added to the experiment to ensure that the MMN they measured was attention-independent. By subtracting the amplitude of the ERP caused by the neutral faces from those caused by the happy and sad faces, they evaluated the differences in the event related potentials that occurred immediately after the deviant stimuli. They focused specifically on the N170 component, associated with facial recognition, and the P250 component, which is often referred to as P3 or P300 and is associated with attention switching. Their results showed that the deviant expressions elicited a larger, or more negative, N170 component and a smaller, or less positive, P250. They referred to
the two evoked visual mismatch negativities as “expressional mismatch negativity” or eMMN (Zhao & Li, 2006). In addition to the existence of the eMMN, Zhao and Li (2006) determined that a characteristic of the expression mismatch negativity is right hemisphere lateralization, which was more pronounced for the happy deviants than the sad deviants. Other studies have used facial expressions as the stimuli in mismatch negativity and oddball tasks and reported a similar right-lateralization of the eMMN to visual stimuli (Astikainen & Hietanen, 2009). The concept that the visual mismatch negativity can be used to measure an individual’s capacity to detect changes of the emotions expressed in a series of faces is particularly relevant to the study of autism because of the pervasive deficits in social cognition characteristic of each of the disorders considered to be part of the autism spectrum.

Thus, the primary purpose of this research was to determine how modulation of the vMMN by emotional expression is related to autism spectrum personality traits. VMMN amplitude was measured in response to faces depicting happy or sad emotional expressions amidst a standard train of neutral emotional expressions. Additionally, a neutral expression with a green tint served as a control condition. Consistent with prior research, this study anticipated that the amplitude of the mismatch negativity would be increased for emotionally salient stimuli. Extending this finding, we expected that this emotion-based amplitude sensitivity would be decreased in individuals with higher levels of autistic personality traits, reflective of a decreased sensitivity to affective expression.

**Method**

**Participants**

Forty-five participants (29 Male) from the College of William and Mary volunteered to participate in this research. The average age for the participants was 19.8 (SD = 1.67) years old.
Each participant provided informed consent and the study was performed in accordance to the rules and regulations of the College of William and Mary’s IRB. Seven participants were excluded because of excessive noise leading to poor EEG data quality, and one participant was determined to be an outlier during statistical analysis.

**Measures**

Prior to the commencement of the task, each participant was asked to read and sign an informed consent form approved by the IRB at the College of William and Mary. The participant then completed the Adult Autism Spectrum Quotient (AQ) behind a privacy screen. The AQ is used to identify autism spectrum personality traits in adults of average intelligence and consists of questions inquiring about social and communication skills, imagination, attention to detail and sensitivity to change. The participant’s level of autism spectrum personality traits was determined from their score (Baren-Cohen et al., 2001). The finished AQ questionnaires were filed according to the participant’s subject number and were never stored with the signed informed consent forms to ensure confidentiality.

**Procedure**

Participants were seated 37 inches from an LCD monitor inside an electronically shielded Faraday chamber and were fitted with a pair of Eartone 3a insert earphones. In order to distract attention from the faces, participants were asked to listen to an auditory track of short stories taken from Shel Silverstein’s *Where the Sidewalk Ends* and to count the number of words that began with the letters “T” and “K.” At the end of each trial block, or group of 115 trials, they were asked to report the number of words beginning with those letters. Participants were also instructed to fixate on the center of the screen. This was to ensure that the participants were passively attending to the faces.
Each block of trials consisted of 115 presentations of a single face displaying different emotions. The gender and race of the faces were counterbalanced across 12 blocks and included 6 males, 2 black, 2 white, and 2 Asian. Each image was on the monitor for 150ms. The sequence of images was pseudo-randomized such that image deviants were presented after six to ten repetitions of the standard image. In each trial block, three of each deviant were presented amidst the sequences of standard stimuli. The standard image for each block was a neutral, or non-expressive, face. Two of the image deviants were faces with "happy" and "sad" facial expressions. The third deviant image in each block was the same as the standard image, but with a green tint. The inter-stimulus-interval was randomized between 500 and 700ms. The inter-block interval was ten seconds. The twelve faces used for this research were taken from the NimStim database of standardized expressional faces (Tottenham, et al., 2009).

Data Acquisition/Analysis

Electrophysiological data were recorded continuously at 2000 samples per second using a DBPA-1 Sensorium bio-amplifier (Sensorium Inc., Charlotte, VT) with an analog high-pass filter of 0.01 Hz and a low-pass filter of 500 Hz. Recordings were made using a fabric cap bearing 72 Ag-AgCl sintered electrodes while participants were seated in an electrically shielded booth called a Faraday chamber. EEG recordings were made using a forehead ground and a reference at the tip of the nose. Vertical and horizontal eye movements were recorded from electrodes placed above and below the eyes and from electrodes placed at the lateral canthi respectively. All impedances were adjusted to within 0-20 kΩ at the start of the recording session.

EEG data were analyzed off-line using EEGLab for MatLab. Channels that contained excessive artifacts were interpolated using a spherical spline. Data were then corrected for both horizontal and vertical ocular artifacts using independent component analysis. Following the
removal of ocular artifacts, the data were segmented between -200ms and 800 ms with respect to stimulus onset. Following segmentation, data were baseline corrected and filtered using an IIR Butterworth filter with a high-pass frequency cutoff of .01Hz and a low-pass frequency cutoff of 20Hz. A simple voltage threshold artifact detection was run for each subject with the voltage limit set to -100 100µV. Participants with more than twenty-five percent of the trials rejected on this basis were excluded from further analysis (N=6). Segmented data were then averaged over trials for each of the standard and deviant stimulus presentations. A grand average ERP of all of the segmented bins for all of the included subjects was used to identify the time course and topographical distribution of the MMN.

Mean amplitude and peak latency of the MMN component were exported and used for the statistical analysis. Occipital and parieto-occipital electrodes (OZ, PZ, P03, P04, P07, P08) were used for the analysis based on methods used in prior research (Zhao & Li, 2006). A 3 x 2 x 2 ANOVA (emotion expression x hemisphere x cortical region) was used to determine the difference in amplitude of the MMN between hemispheres and electrodes. The Greenhouse-Geisser correction for violation of the sphericity assumption was used where appropriate. A Pearson Correlation Coefficient was used to determine the relationship between MMN amplitude and autistic personality traits.

Results

Grand averaged data revealed the visual mismatch negativity ERP component in the occipital and parieto-occipital areas of recording. As shown in the waveforms in Figure 1, the vMMN ERP can be seen over occipital recording sites.

Hemispheric Differences
An analysis of variance was performed to evaluate the variance in electrical activity across the scalp present in different locations and in response to different emotional expressions. Results from a 3 x 2 x 2 ANOVA (emotion expression x hemisphere x cortical region) identified a significant main effect in amplitudes of the visual mismatch negativity component between the right and left hemispheres at electrode sites P07 and P03 for the left hemisphere, and electrode sites P08 and P04 for the right hemispheres, $F(1, 36) = 16.410, p = .000$. The head plots and graphical representation in Figure 2 display that the stronger vMMN ERP component in the right hemisphere rather than the left. Additionally, the ANOVA indicated a significant main effect of Hemisphere with amplitudes being larger in the right than left hemisphere, $F(1, 36) = 8.804, p = .005$. This implies that the mismatch negativity is lateralized to the right hemisphere, and that the neurons in the areas of recording further away from the midline experience a stronger visual mismatch negativity response. Furthermore, there was a statistically significant interaction between the cortical region as determined by the electrode site and the cortical hemisphere, $F(1, 36) = 10.620, p = .002$, reinforcing the assumption that the vMMN component is stronger not only in the right hemisphere but also the more laterally that the measurement is taken (See Figure 3).

*Emotion Differences*

Results from the 3 x 2 x 2 ANOVA indicated a statistically significant main effect of Emotion, $F(2, 72) = 16.528, p = .000$ indicating a difference in how the emotions that were used in the task, happy and sad, are processed in a visual mismatch negativity paradigm. This result also shows the difference between the MMN ERP for the expressional faces and the neutral green face. Moreover, a significant interaction was also found between hemisphere and emotion, $F(1, 36) = 9.368, p = .004$, electrode and emotion, $F(2, 72) = 5.352, p = .027$, and hemisphere, electrode, and emotion, $F(2, 72) = 6.131, p = .018$. These results indicate that the expressions, or emotions,
assessed during the visual mismatch negativity task elicit a stronger MMN ERP in the lateral right hemisphere. Consequently, the next step for this research was to determine whether scores on the Autism Quotient were associated with the mismatch negativity ERP component in the right parieto-occipital cortex.

**Emotion and Autistic Personality Traits Relationship**

In order to determine how the MMN amplitude was correlated with autism spectrum personality traits, a Pearson correlation was used to evaluate the association between MMN amplitude at each electrode site and AQ score. Consistent with our expectation that the MMN may be an indicator of affective reactivity, there was a significant positive correlation between the mismatch negativity produced by the happy deviant facial expression and the score on the Autism Quotient at electrode site P08, r (37) = .343, p = .038. This is consistent with the analysis of variance, which suggests that the MMN is largest at the recording sites located on the right lateral parieto-occipital cortex. This is evidence for the hypothesis that higher levels of autistic personality traits are associated with higher MMN amplitude in response to emotional stimuli. This would imply that the automatic change detection processes of individuals with autism, or at least higher levels of autistic personality traits, do not register the change in emotion in the same way that individuals with lower levels of autistic personality traits do.

**Discussion**

The primary purpose of this research was to determine how modulation of the vMMN by emotion is related to autism spectrum personality traits. Electrophysiological data from the present study identify a positive correlation between level autistic personality traits and amplitude of the vMMN response in the parieto-occipital cortex in the right hemisphere. Right hemisphere lateralization of comprehension of emotional facial expressions is consistent with
previous research on emotion, (Schwartz, Davidson & Maer, 1975) and additionally with the results by Zhao & Li examining the expressional mismatch negativity, (Zhao & Li, 2006). This lateralization of emotion has been examined further through studies of patients with lesions to the right hemisphere. Patients in these studies have deficits in the comprehension of facial expressions (Blonder, Bowers & Heilman, 1991). These results confirm the hypothesis for this study that the amplitude of the visual mismatch negativity component positively correlates with level of autistic personality traits when the vMMN task uses facial expressions as the stimuli. Furthermore, these results suggest that the social deficits that accompany autism may be pre-conscious. Because the participants in this study were students, none of who were diagnosed with ASD, it could be assumed that this difference would be more pronounced in participants who have been diagnosed with ASD.

Evidence derived from the data also displayed a difference in how the faces with happy, sad and neutral expressions were processed irrespective of AQ score. There was a significant main effect for each of the MMN ERPs elicited by the different emotions, implying that the brain automatically makes a distinction between expressions when comprehending them. An interesting finding from this research was that the overall amplitude of the raw ERP that corresponded with the sad expression deviant was larger, however, the MMN that corresponded with the happy expression was the only expression with which there was a significant correlation with the level of autistic personality traits. This may be because while the sad expression produced a pronounced MMN, there was no significant difference between how deviant sad expressions are processed in neuro-typical and autistic brains. Assuming that the preconscious response to happy deviant expressions in individuals with higher levels of autistic personality traits is smaller, one could assume that the comprehension of expressions of positive affect is
impaired. Conflicting evidence appears in many studies concerning emotion processing and ASD, however, it is the general belief that emotion-processing is impaired, especially when comprehending emotional expressions with few physical differences, as is the case with the comparison between neutral faces and happy expressions where the mouth is closed (Blair, 2005).

Results from this study are significant to the study of autism because of the deficits in social comprehension that are included in the list of symptoms associated with ASD. Finding a tangible neurological difference between how the brains of participants with higher levels of autistic personality traits react to deviance in emotional expression could suggest that this difference exists in autistic individuals. Having a neurological marker for autism would be a colossal step forward for the diagnostic process due to the all too frequent mislabeling of socially awkward youth and adolescents. Because the mismatch negativity is an automatic reaction to change and can be assessed using the non-invasive technology of EEG, young children of any intelligence level can be evaluated. This could assist in the early identification of and therapy for autism. The combination of behavioral and electrophysiological techniques would make the diagnostic process from autism more valid and reliable. Early, and accurate, identification of autism can facilitate the spread of early intervention programs, giving children with autism the best chance for a typical development.

A limitation of the current study is its lack of trials using emotional expressions as the standardized stimuli. In other words, because there may be quantifiable differences between the happy and sad expressions on differences other than emotional valence, it is impossible to know whether the larger MMN to sad facial expressions is due to the nature of the emotional content or some other physical property of the image. Näätänen et al. (2004) described an “optimal
paradigm” in the auditory modality, which elicited five MMNs in each trial as opposed to one. Applied to this task, the “optimal paradigm” would contain trials where the each of the deviant emotional expressions act as the standard stimulus, and the deviants are either the other two deviants or the standard neutral expression. The use of the “optimal paradigm” described by Näätänen et al. (2004) in future research would allow for a more complete analysis of the effects of emotional expressions on the vMMN in participants with autism spectrum personality traits.

Concerning future research, it would be also be advantageous to examine the effect of gender on the vMMN paradigm. Investigating gender effects would be particularly relevant in the context of autism due to the gender differences in the diagnosis of autism. According to current statistics, males are four times more likely than females to be diagnosed with autism (“Clearing the fog,” 2011). A limitation of this study is that the population consisted of undergraduate students, all of average or above average intelligence. Out of the participants the mean score on the AQ was 15 with a standard deviation of 4.7. The highest score on the AQ was 26, which just meets the level of Asperger’s. Future research could replicate this study using participants who have been diagnosed with one of the disorders included in Autism Spectrum Disorder. Exploring this relationship would help to solidify the use of the vMMN ERP component as a diagnostic tool.

As is the case with much of the current research concerning autism, more research is required before any definitive conclusions can be drawn, however, the findings from this research will hopefully contribute to a greater understanding of this pervasive development disorder.
References


VMMN AS INDICATOR OF EMOTION PROCESSING IN AUTISM SPECTRUM PERSONALITY


Figure Caption

Figure 1: Comparison of the raw MMN ERP and the MMN difference waveforms at electrode sites FZ, CZ, PZ and OZ.
Figure 1

VMMN AS INDICATOR OF EMOTION PROCESSING IN AUTISM SPECTRUM PERSONALITY

**ERP**

- **FZ**
  - Time (s)
  - Amplitude (µV)

- **CZ**
  - Time (s)
  - Amplitude (µV)

- **PZ**
  - Time (s)
  - Amplitude (µV)

- **OZ**
  - Time (s)
  - Amplitude (µV)

**MMN Difference ERP**

- Happy Deviant
- Sad Deviant
- Gross Deviant
VMMN AS INDICATOR OF EMOTION PROCESSING IN AUTISM SPECTRUM PERSONALITY

Figure Caption

Figure 2: Graphical and topographic representation of the mismatch negativity in response to the happy deviant.
Figure 2

**Happy MMN at PO8**

![Graph showing MMN Amplitude (μV) over time with shaded area indicating significant activity.]

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![Image of head models showing brain activity with color scale from -1 to 1.]
Figure Caption

Figure 3: Comparison of the MMN amplitude in the right and left hemispheres at electrode sites PO4, PO8, PO3, and PO7.
Figure 3
Figure Caption

Figure 4: Graphical representation of the significant Pearson Correlation between amplitude of the MMN in response to the happy deviant and AQ score.
Correlation Between Happy MMN and AQ score