Page 188 to 192

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Feeling Different: A Mini Review of Emotional Facial Recognition and the Role of the Amygdala for Individuals with Autism Spectrum Disorder

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Abstract

Research in the field of Autism Spectrum Disorder is influenced by multiple fields. Neuroscience, specifically neuroimaging, has contributed to the increasing understanding of Autism Spectrum Disorder in a number of ways. One such contribution is the role that neuroimaging has played in deepening the basic understanding of how emotions may be processed differently in the brains of individuals with Autism Spectrum Disorder as compared to individuals without. This paper will focus on two strands within this research, emotional face processing and neural activation in the amygdala, in order to elucidate some of the emerging understanding of structural and functional brain differences amongst individuals with Autism Spectrum Disorder.

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Introduction

The field of research in Autism Spectrum Disorder (ASD) has grown exponentially over the last several decades, gaining attention by a number of different disciplines across the scientific landscape. Neuroscience is one such discipline whose researchers have taken a deep interest in better understanding ASD from a neurological perspective, and has been able to contribute to the breadth and depth of the knowledge base from diagnostic and phenotypical organization to interventional practices. One specific area of scholastic advancement to which neuroscience has specifically contributed is the discovery and delineation of variations in both structural and functional patterns in brain regions of individuals with ASD. These discoveries have allowed stakeholders in the field, from diagnosticians, parents and teachers, as well as individuals with ASD themselves to gain an understanding of ASD from a unique perspective; one that can potentially offer a more experiential picture than any other perspective that has come before it.

The differences in both the structure and function in the brain regions that are specifically associated with emotion and emotional processing has been elucidated widely by advances in neuroimaging. While practitioners, parents, and individuals with ASD have long anecdotally related differences in emotional responding among those with ASD, the field was largely limited to behavioral observations and cognitive speculation as to why these emotional differences existed and how they could be understood.

The existence of differences in emotional processing and expression is well-established in the literature. Hill, Berthoz, and Frith (2004) conducted a study with 27 young adults with ASD along with 40 of their relatives, as well as 35 adult controls, in order to investigate the role of cognitive processing and emotion. Using the Toronto Alexithymia Scale and the Beck Depression Inventory, the researchers concluded that the participants with ASD were significantly more impaired emotionally than their non-autistic counterparts [1].

Silani, Bird, Brindley., *et al.* (2007) contributed to further understanding the emotional situation of individuals with ASD and the brain's potential role in it by conducting a study using functional Magnetic Resonance Imaging (fMRI). Using 15 male participants with ASD as well as 15 control participants without ASD, researchers gathered data using pictures from the International Affective Picture System on two separate occasions, and compared the participants' answers with activity measured during the fMRI. Interestingly, results indicated that individuals with ASD differed in brain activity from control participants regardless of whether they self-identified difficulty processing emotions. These findings suggest that difficulties in emotional functioning and regulation may be likely due to gross brain function rather than cognitive or mentalizing functions, establishing the very real possibility of organic difficulties in processing emotions [2].

Mazefsky, Herrington, Siegel., *et al.* (2013) offered a theoretical perspective investigating amplified emotional responses and poor emotional control specifically, which was conceptualized as compromised emotional regulation. The authors suggest that the compromised emotional regulation among individuals with ASD is likely a multifactorial issue, with some aspects potentially acting in a negatively synergistic fashion. Therefore, better understanding individual contributors to compromised emotional regulation is likely to result in more effective individualized treatments [3].

While it is clear that there is evidence of a difference in the emotional responsiveness, processing, and social outcomes of individuals with ASD, there is also emerging evidence suggesting potential neurological sources for this difference. This paper will emphasize two main strands in the neurological research investigating the root of emotional differences in individuals with ASD: (1) atypical structure and processing in the amygdala; and (2) atypical processing of faces and facial expression.

Facial Processing for Individuals with ASD

There appears to be a clear connection between emotional recognition and processing and facial processing as evidenced by a number of studies in the extant literature. These reports typically suggest that an area of the brain known as the fusiform gyrus (FG), which lies on the basal surface of the temporal and occipital lobe and specifically the fusiform face area (FFA), which is on the lateral side of the FG on the temporal lobe, is directly involved, and appears to be atypical functionally for individuals with ASD.

Dawson, Webb, Carver., *et al*. (2004) used a process called event-related potentials (ERPs) to investigate potential neural activity differences between 29 3 to 4 year old children with ASD and 22 age-matched controls when processing neutral or fearful facial expressions. Results suggested that children with ASD showed little difference in the processing of a fearful face with a neutral face, while the control children did [4].

Corbett, Carmean, Ravizza., *et al.* (2009) investigated structural and functional MRIs for 12 children with ASD, 8-12 years old with 15 age-matched controls deemed neurotypical. Results indicated that children with ASD showed reduced activation in the FG area as compared to controls [5].

Speer, Cook, McMahon., *et al.* (2007) investigated whether facial processing differed for children with ASD in four specific types of situations: (a) social dynamic, or video clips of social interactions (b) social static, or pictures of social interactions, (c) isolated dynamic, or videos of alone individuals, and (d) isolated static, or pictures of alone individuals. Results indicated that, compared to controls, the participants with ASD differed in facial processing only for social dynamic scenes, in which they exhibited decreased fixation for eye

regions and increased fixation for body regions. These findings suggest that eye contact as a global issue may not be as significant as the role of eye contact in specific social situations [6].

The Role of the Amygdala in Emotions for Individuals with ASD

While the brain is undoubtedly a complex organ ever posing challenges to neuroscientists, there are some aspects of brain research that appear to be relatively consistent. One of these areas is the apparent role of the amygdala in emotional functioning for human beings. The amygdala is an almond-shaped structure that is considered to be part of the brain's limbic system. Geographically, it is located in the medial temporal lobe, specifically at the anterior end of the hippocampus. Generally speaking, the amygdala is associated with processing emotions, particularly fear and pleasure, and may be associated with emotional memory.

An early attempt at delineating an amygdala-based theory of ASD was proposed by Baron-Cohen, Ring, Bullmore., *et al*. (1999), which resulted from fMRI studies of individuals with ASD and their ability to judge emotion based on cues from other people's eyes. The imaging demonstrated that the participants did not show activation in the amygdala when making such judgments, while participants without ASD did, as was expected [7].

Bachavalier and Loveland (2005) contribute to this neurodevelopmental model for an amygdala-based theory of emotional processing for individuals with ASD; one which is directly connected with social cognition. The theory focuses primarily on the reciprocal neurological relationship between the orbitofrontal area of the cortex and the amygdala. Specifically, the orbitofrontal area of the cortex receives far more signals from the amygdala than other areas of the frontal cortex. Because these areas communicate closely, the connection between their various functions (social dynamics and emotional regulation) are likely interconnected. As suggested by the authors: …the anatomical organization and reciprocal relationship between the amygdala and the orbitofrontal cortex implies that these brain regions may share a close functional relationship within a system essential for the maintenance of intra-specific social bonding and the self-regulation of emotional states (p. 102).

This notion becomes important in the study of ASD in that the authors suggest that some of the emotional symptomatology of ASD could potentially be explained by issues with the medial temporal lobe, of which the amygdala is a part, thus affecting the functionality of the orbitofrontal-amygdala circuit [8].

Schumann, Hamstra, Goodlin-Jones., *et al*. (2004) used magnetic resonance imaging (MRI) to measure both the total cerebral volume and specific amygdala volume of individuals with ASD as compared to those without. Participants included individuals aged 7.5 to 18.5 years in four diagnostic groups: (a) autism with mental retardation (n = 19); (b) autism without mental retardation (n = 27; (c) Asperger syndrome (n = 25); and (d) age matched individuals without a diagnosis (n = 27). Results showed that children with ASD (both with and without MR) had amygdalae that were larger in volume (specifically between the ages of 7.5 and 12.5) than those without. These results suggest that both the occurrence of ASD as well as age may have an effect on the volume of the amygdala [9].

Kleinhans, Richards, Weaver., *et al*. (2010) conducted a study using fMRIs for 31 adults with ASD and 25 age, gender, and IQ-matched controls to investigate brain reactivity, including amygdala action in response to facial expression matching, specifically of fear or anger. The researchers also investigated the connection between brain activity and self-reported social anxiety measured by the Social Avoidance and Distress Scale (Watson & Friend, 1969). Among a number of differences in brain activation between controls and participants with ASD, individuals with ASD showed increased activation in the amygdala which also correlated with high levels of self-reported social anxiety [10].

Kliemann, Dziobek, Hatri., *et al.* (2012) investigated the connection between eye contact avoidance, a common trait in individuals with ASD, with amygdala activation. Participants included 17 males with ASD as well as 16 controls considered to be neurotypical.

Researchers showed an initial image of a fixation cross, toward which participants aimed their eyes, followed by a series of faces to participants and asked them to gauge emotion with either the mouth or the eyes occupying the area of the initial fixation cross. Using eye-gaze measurement technology as well as blood oxygen level-dependent (BOLD) signal in the amygdala, the researchers were able to detect differences in both amygdala activity and eye gaze. Unsurprisingly, individuals with ASD gazed more often away from than toward the eyes as compared with controls, as well as different BOLD activity between the two groups, suggesting both behavioral and neurological differences in people with ASD as it relates to facial expression processing and corresponding amygdala activity [11].

Swartz, Wiggins, Carrasco., *et al.* (2013) investigated the specific process of amygdala habituation, which is the neurological tendency for the amygdala to decrease in responsiveness when presented with repeated stimuli, and is thought to be indicative of typical brain functioning. This process is important in allowing individuals to regulate and maintain appropriate levels of arousal in predictable social stimuli. The lack of amygdala habituation is thought to be associated with higher levels of anxiety. Data collected from fMRIs of 32 children and adolescents and 56 typically developing control participants were analyzed to measure levels of amygdala habituation in response to pictures of faces showing the emotions of happy, sad, fearful, and neutral. In general, results indicated that the participants with ASD showed decreased amygdala habituation as compared to the typically developing controls. Similarly, amygdala habituation correlated with the individual's score on the Social Responsiveness Scale, with increased severity indicating decreased habituation. These results suggest that faces, specifically repeated faces, are processed differently amongst individuals with ASD, and that the amygdala is likely to play a role in this experiential difference [12].

Perhaps among the more intriguing studies involving amygdala function and people with ASD is that conducted by Guastella, Einfeld, Gray., *et al*. (2010). Building on the idea that oxytocin can enhance amygdala activity, potentially stimulating the ability to feel empathy and process emotion in a way more indicative of typical brains, the researchers sought to test its effect on individuals with ASD in a double-blind, randomized placebo clinical trial. Administering either oxytocin via intranasal spray or a placebo to 16 males aged 12 to 19 years old, the researchers found that individuals who received oxytocin as compared to the placebo showed an improvement in the Reading the Mind Through the Eyes Task. This study suggests that understanding the neurological makeup of individuals with ASD can enhance basic understanding of the condition, but can also inform potential treatment and intervention options as well [13].

Conclusion

While there are many avenues that are ripe for exploration in the area of using neuroscience to contribute to the general and interventional understanding of ASD, this paper reveals that a particularly rich area is that of facial processing and, specifically, the role of the amygdala in emotional recognition and processing. The role of neuroscience and its ever increasing effectiveness and sophistication of neuroimaging will continue to be an imperative contributor to better understanding how individuals with ASD experience the world and provide much needed insight to a field that is often limited to behavioral observations and clinical interpretations.

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