Connection Between the Amygdala and Autism: A Review of fMRI studies

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Autism spectrum disorder (ASD) is a highly complex neurodevelopmental disorder characterized by its heterogeneity because it has different causes, levels of severity, and interventions. Due to its heterogeneity, learning more about its underlying mechanism through functional magnetic resonance imaging (fMRI) can help researchers identify atypical neurobiological processes associated with it. Specifically, fMRI research can help determine etiologies of ASD, improve the scalability of research, and find targets for new areas of research. The amygdala is one area of focus as a deficit in the amygdala could help explain the socio-emotional behavior of ASD individuals. This article synthesizes fMRI studies findings and aims to assess fMRI studies of the amygdala in autistic individuals to identify differences in emotional, social, and cognitive behaviors and determine its implications for future research. Analysis of prior work reveals amygdala dysfunction, suggesting inhibition in the social function in ASD individuals. Outside of areas of localized dysfunction, the idea of alternate pathways and functional connectivity networks emerge as areas that need more study. Recommendations for future empirical work include more secondary analyses of existing data-sets with the concurrent inclusion of more under-represented groups and individuals with comorbidities.

Keywords:

Autism spectrum disorder, fMRI, amygdala, functional connectivity

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by deficits in communication, social functioning (e.g., how an individual interacts with their social environment through recognition, understanding, and behavior), and restrictive, repetitive behaviors or routines[1]. One of the earliest documented studies of autism was in 1943 by Leo Kanner when he observed communication, sensory, and repetitive behavioral issues in eleven cases of children.[2] Despite its relatively high prevalence in populations, ASD individuals still face difficulties in diagnosis, treatment, and prevention due to its heterogeneity in clinical presentation. This is shown in low- to high-functioning autism, comorbidity, and different clinical features such as delayed language or movement. Common comorbidities amongst ASD individuals include intellectual disability (ID), sleep dysfunction, and attention-deficit/hyperactivity disorder (ADHD).[3] However, it is also common for ASD individuals to present a wide variety of symptoms and behaviors without consistent symptom clusters.[4]

To gain a better understanding of the disease researchers have shifted from a diagnostic framework to a research framework which incorporates neuroscience to isolate biomarkers and neural correlates. Identifying biomarkers can be beneficial to better understanding the etiology, diagnostic signals, and treatment targets for autism.[5] Structural brain research has yielded particularly promising results. The amygdala has been suggested to be one of the major structures implicated in the underlying neurobiological basis of autism. Baron-Cohen et al. (2000) first proposed 'the amygdala theory of autism' based on existing evidence for the amygdala's role in social function. The amygdala is a complex organ that has functions related to value representation (encoding value into stimuli), decision-making, and is part of the limbic system that directs socio-emotional responses to stimuli after the amygdala receives somatosensory, olfactory, visual, and auditory information.[6,7] The 'amygdala theory' hypothesizes three main ideas: the amygdala has a social function, amygdala abnormality has a direct relationship with autism, and that ASD individuals have amygdala deficits. The researchers posited that since autistic individuals often demonstrated social deficits, and the amygdala has a social function, there could be amygdala abnormality in this population.[6]

The 'amygdala theory' suggests a functional abnormality for individuals with autism. As such, current research on the neurobiology of autistic individuals has moved towards fMRI studies which observe function and activity and away from

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MRI studies that observe structural differences. fMRI studies have shown a trend of abnormal activation when autistic individuals were treated with various social and emotional processing tasks. Unlike MRI studies, fMRI studies have researched various domains including reward circuitry and face processing. Due to a large amount of data on different tasks within fMRI studies, this literature review will synthesize existing fMRI data from 2010 to 2022. In this review, I will evaluate data on amygdala activation in order to assess how fMRIs of the amygdala of ASD individuals can be used to identify differences in ASD-related emotional, social, and cognitive behaviors. In doing so, I will relate neurobiological deficits in behavioral tasks to autism behavior deficits by outlining key trends across the data, gaps, and limitations. See Table - I (Appendix) summarizing key findings. Finally, I will discuss the implications as well as provide recommendations for future research.

Methods

Articles cited in this review were selected from an advanced Pubmed database search by keyword and date filters. Keywords were "autism", "amygdala", and "fMRI." Publications from 2010 to 2022 were included. Additional articles were collected from the citations within eligible Pubmed articles. Articles discussing or reviewing amygdala-specific results from the fMRI of autistic individuals were included. Exclusion criteria included articles discussing autism quotient in healthy humans and animals (e.g., primates and mice) because they did not involve the primary group of focus. Clinical trials were also not discussed due to the focus on individuals without medical interventions. Comorbidity and comparisons between disorders (e.g., anxiety, Klinefelter syndrome, Fragile X syndrome) were not included due to the extra comparisons between other disorders and ASD.

Literature Review

Historically, 'the amygdala theory of autism,' remained consistent with the idea of abnormal amygdala activity in fMRI studies. However, separate scholars proposed that the theory should be modified as they did not find a direct relationship between amygdala evaluations (e.g., volume) and autism behaviors (e.g., emotional understanding) in studied groups.[8] As a result, the trajectory of current research has shown a breakthrough from typical comparison of localized dysfunction. Following analysis of various studies relating tasks to levels of activation in the amygdala, two trends emerged: alternate pathways that compensate for the amygdala's deficits and functional connectivity deficits of the brain. These trends are key as they direct research into new deficit areas and away from the 'amygdala theory' to better explore ASD differences. As opposed to observing a localized region of connectivity, researching cross-brain pathways and the functional connectivity of these pathways can help better the understanding of the involved networks within the brain.

Atypical Activation of the Amygdala in ASD individuals

Over the past few decades, research on the specific functions of the amygdala has increased. One common experimental paradigm is the Mind in the Eye test, which typically involves asking participants to select which emotion out of four-option responses is the correct emotion that the eyes are displaying. Prior neuroimaging studies that used an emotion recognition task of neutral faces found amygdala hypoactivation,[9] while others found no significant differences with neutral faces.[10,11] Meta-analyses on emotion face processing for ASD individuals also have conflicting results. One 2015 meta-analysis found indications for hyperactivation in the bilateral thalamus, bilateral caudate, and right precuneus, suggesting issues with involuntary emotional face processing as the thalamus and caudate are involved in a subcortical route connected to "cortical regions for emotional-face processing without any conscious awareness of the stimulus".[12] Only the sub-analyses were able to reveal amygdala hypoactivation, suggesting a deficit in this center of emotional processing.[12] On the other hand, a 2021 meta-analysis was not able to identify any of the same patterns after excluding ROI studies, as those have been criticized for the potential inflation of results and the possibility of false positives.[13,14] This recent meta-analysis suggests that any studies using ROI analysis may be problematic for future use due to unreliability as seen in the difference between these two papers.

In addition to meta-analyses, empirical work assessing domains like aesthetic judgment, face anticipation in reward circuitry, sensory stimuli response, and gaze to the eye region have revealed hyperactivation. Autism is correlated with extraordinary art abilities. For example, Stephen Wiltshire was an English artist with ASD who was able to draw city landscapes after a single helicopter ride.[15] Additionally, one study tested the ability of autistic individuals to judge the aesthetics of landscapes and fractal images based on the degree of color in the images. Results indicated hyper-activation of the right amygdala when ASD individuals made judgments; additionally, the ASD group thought the fractal artwork was less beautiful than the control did. The paper hypothesized that due to the rareness of fractal images, the ASD group felt unused to them. This was shown functionally, as the amygdala was hyperactivated, suggesting the ASD group experienced "greater anxiety or surprise" to the fractal images and thus liked them less.[15] With face anticipation, one study further divided the reward process into anticipation and outcomes for

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monetary and social stimuli when obtaining fMRIs. The authors found heightened bilateral amygdala activity in response to face anticipation compared with controls which matched existing knowledge about the amygdala's social function.[16] Other studies tested sensory over-responsibility (SOR), that is, the negative response or avoidance of sensory stimuli in ASD individuals. Results found hyperactivation in auditory and joint auditory-visual conditions.[17] Another study also found hyperactivation of the amygdala and primary sensory cortices.[18] With gaze, instead of the Mind in the Eye task, the study conducted natural viewing of various faces and found hyperactivation in response to angry and neutral faces. Interestingly, gaze modulation during the experiment suggested that amygdala activity could be modified by gaze behavior.[19] There is still much to be done in this area of study as aesthetic judgment and SOR studies are the first of their kind, but this suggests that amygdala hyperactivation may be specific to certain tasks. Other domains have yielded hypoactivation including reward circuitry, voice perception, and moral reasoning. In testing reward circuitry, one paper used a go/no-go task with three incentives of non-reward, social reward, and monetary reward. They found hypoactivation in the amygdala, nucleus accumbens (NAcc), and prefrontal circuitry in response to social/monetary rewards.[20] This aligns with previous studies that found a diminished response from other brain areas like the NAcc and frontostriatal networks to social/monetary rewards.[21,22] Others have researched using human voice cues as motivating social stimuli to investigate why ASD individuals are indifferent to voice cues. A resting-state fMRI study found hypoactivation in reward circuitry and the voice-selective cortex.[23] More research is needed as a different voice-selective cortex study unrelated to reward circuitry found hyperactivation of the amygdala in response to unfamiliar voices.[24] This suggests that the dysfunction is reward domain specific. Another study found hypoactivation of the amygdala and superior temporal sulcus (STS) when participants were subject to different clips of neutral, basic (e.g., sad, happy), and complex (e.g., proud, confused, hurt) emotion tones.[25] With the amygdala, a study looked at ASD individuals' ability to conduct moral reasoning. The ASD and control group took tests on sixty social-ethical, collective gain vs. individual loss, and non-moral dilemma questions. Results showed a hypoactivation of the limbic region including the amygdala.[26] Another study specifically found reduced functional connectivity between bilateral amygdalae and the left inferior occipital cortex areas.[27] These studies give an understanding of amygdala functioning due to their intersection in abnormal activation in reward circuitry and social processing.

Domain Specific Dysfunction & Functional Connectivity

Experimental studies must consider the disadvantages of isolating the amygdala when considering amygdala abnormality as amygdala activation is often connected to other brain organs including the fusiform gyrus, NAcc, thalamus, and more.[12,20,28] Several studies note the possibility of domain-specific dysfunction to explain findings. For example, a 2015 study conducted on emotional face processing found that ASD individuals showed no significant difference in activity in the social brain area involving the fusiform gyrus, STS, and medial prefrontal cortex, suggesting that this was due to emotion face processing not involving activation of the social brain area.[12] Instead, amygdala abnormality was only found in subsequent sub-analyses which compared emotional face and non-face processing conditions.[12] Another study found that ASD individuals had no significant differences in brain activity when reading body emotions; ASD individuals were subjected to images of people with blurred faces in positions of fear and neutral expression.[29] This suggests that body-emotion perception, though a part of social functioning, is not activated in the same domain as facial emotion perception with the amygdala. Other examples include the abnormal activation of the amygdala with other areas including the subgenual ventromedial prefrontal cortex (vMPFC) and middle temporal gyrus in emotion face processing.[30] This is key as determining the amygdala's functional connections to other brain parts can help research separate ASD and control pathways rather than local dysfunctions for neurological processes.

Functional connectivity abnormalities could indicate alternate pathways for ASD individuals. For example, one meta-analysis found a common activity of amygdala hypoactivation paired with thalamus hyperactivation.[12] Another found a specific pattern of functional underconnectivity between the bilateral amygdalae and the left inferior occipital cortex with increased functional connectivity between the right amygdala and right sensorimotor cortex correlated to less severe ASD symptoms and better functioning[27] These examples suggest that these specific connectivity patterns may be a response to neural functioning deficits that arise from ASD. Specifically, there is a pattern where the upregulation of one brain structure is compensated by the downregulation of another. Both studies postulate that the hyperactivation of the structure or hyperconnectivity compensates for the hypoactivation or hypoconnectivity of another.

This idea of alternate pathways could also explain altered functional connectivity between the amygdala and other areas. One study found connectivity disruption in preschool-age children, with weak connectivity between the amygdala and bilateral medial prefrontal cortex, temporal lobes, and striatum, which have functions in communication and behavior.[31] Other studies have found similar functional underconnectiv-

ity in adults and young adults.[32,33] One 2013 study found that while control groups had increased connectivity in the left amygdala-vmPFC during face processing over time, the ASD group did not show the same progression, suggesting underconnectivity along this pathway.[34] Similarly, hypoconnectivity in the amygdala-ventrolateral prefrontal cortex (vlPFC) pathway may be associated with disruptive behaviors as a study noted that ASD individuals with disruptive behaviors had lower functional connectivity compared to ASD individuals without.[35] Interestingly, the previous case of hypoactivity of the bilateral amygdala/left inferior occipital cortex and hyperactivity of the right amygdala/right sensorimotor cortex[27] demonstrates how there can be both hyper- and hypoactivation within the amygdala. This indicates that the left and right amygdala regions can have different functions. Similarly, another paper found a difference in functional connectivity within the amygdala's nuclei for input and output.[36] This is important as it shows the basis for studying each of the nuclei in the amygdala and specifying left versus right amygdala concerning autism, to specify which areas activate during tasks. Though there has been a shift away from localized neuroimaging to functional connectivity due to increasing evidence about atypical large-scale neural connectivity[30] there is still a role in studying localized amygdala function.

Discussion

Atypical activation of the amygdala is associated with different tasks including reward circuitry, face processing, and gaze aversion. These differences in biological processes may then be tied back to ASD-related behaviors. It is clear that more work needs to be done on analyzing prior experimental findings and their implications for real-world actions. In addition, it should be considered that seemingly conflicting findings could actually be the result of the heterogeneous nature of ASD. Tying all behaviors to amygdala dysfunction may be helpful, but more data is needed due to current limitations which include small sample size and underrepresented ASD subgroups. Future work will need to focus on gaps in knowledge and representation in samples in order to obtain more generalizable evidence.

In reviewing existing work, it is clear that there is a shift from singular, localized patterns in the amygdala to the idea of domain-specific dysfunctions, dysfunctional connectivity, and alternate pathways to compensate for amygdala dysfunction. With domain-specific dysfunction, evidence suggests that it is not localized dysfunction in the amygdala, but localized dysfunction in the broader brain region that includes the amygdala. More research must be done as it is not necessarily clear which domain is involved with the implicit tasks in experimental studies. For example, by tying ASD behaviors to biological evidence, one study postulated that emotion face processing would involve the social brain areas (e.g,, fusiform gyrus, medial prefrontal cortex, etc), yet the main analysis involving comparison of activation region did not find any atypical activity.[12] This suggests a push for more connectivity studies and regional studies that focus on multiple brain regions. However, even with functional connectivity, centering the amygdala may still be important as evidence suggests specific atypicalities in the amygdala. It may be critical to study the functional connectivity of these pathways (i.e., upregulation of thalamus and downregulation of amygdala) to better understand the interplay between alternate pathways and their functional connectivity. It may also be beneficial to observe the strength of the connectivity in these abnormal pathways when compared with typically developing controls. Finally, the emerging area of research in functional connectivity and alternate pathways can facilitate a better understanding of the relationship between atypical observable behaviors and brain activity.

With Baron-Cohen's 'amygdala theory of autism,' it is clear that modifications should be made due to a shift in research. Mounting evidence indicates that the amygdala has a social function from its activation in social tasks like emotion processing and decision-making. With the current paradigm shift from localized dysfunctions to dysfunction in connectivity networks, the amygdala has a key role in social function for ASD individuals, but may not have a direct, causal role. Instead, multiple regions with varying activations are likely implicated in ASD cognition. Though autistic individuals have deficits, research suggests that the deficits are compensated for through alternate neural pathways.

Future Directions and Recommendations

Despite the current paradigm shift, there are several reasons to continue researching amygdala function in ASD individuals: (1) To confirm past studies by replicating them with increased sample sizes, (2) to expand samples by studying underrepresented ASD subgroups, (3) to evaluate the results of secondary analyses on existing fMRI images, and (4) to move towards multi-modal studies to strengthen research quality.

One possible direction for confirming past studies is utilizing existing databases of ASD neuroimages. This can increase the sample size and diversity of studies. An example is the Autism Brain Imaging Data Exchange (ABIDE), an anonymous, pooled database for ASD and control fMRIs that could potentially accelerate research and provide a place for data to be analyzed together. Today, the ABIDE II connectome has 1114 datasets that require additional analyses.[37]. A pooled database can allow for consistency in study methods and procedures for future studies to create more uniform quality data for secondary analyses. As this database grows, we can increase the number of studies using the same procedures and

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sample profiles, which may allow for a reduction in the number of conflicting studies. For example, one whole brain analysis was able to reconcile contradicting evidence about hyper- and hypo- connectivity within the brain through this type of pooled work; in a similar way, conflicting amygdala findings may be better addressed through more uniform and pooled analyses.[38]

It is also essential to increase the quality of studies by including more ASD subgroups. There is a lack of representation of comorbidity and levels of autism spectrum functioning in most studies due to eligibility and experimental design requirements. For studies with small sample size, matched pairs design is conducted, resulting in intelligence quotient (IQ) matching with typically developing groups, or ASD groups with only high-functioning individuals.[39–41] Comorbidity is also a variable that is removed from the majority of experiments to control for results that might be caused by an anxiety disorder or attention-deficit/hyperactivity disorder instead of ASD.[42–44]. One study found that there is a lack of neuroimaging studies on ASD with intellectual disabilities, minimal verbalism, and history of regression.[45]. One meta-analysis found that 19 out of 32 studies did not discuss this possible confound of ASD individuals being unable to stay still during fMRI.[46] Future researchers should take this into account as results can only be generalized to the population of ASD individuals without such criteria. One 2018 study was able to make methodological improvements, such as providing behavioral preparation for severe ASD individuals, providing flexible support, and improving scan/analysis techniques without sedating the children for fMRI.[47] It is clear that using such methods could improve the rate of studies on a larger variety of ASD individuals.

As the current field of neuro-imaging has increased, research should include more secondary analyses through meta-analyses and follow-up studies of existing data. In doing so, the secondary analysis would be able to elucidate patterns of activation in the thirteen nuclei of the amygdala, determine the regions of activation within the amygdala, and review newer patterns such as alternate pathways or functional connectivity in past studies with different focuses. One example is reviewing past studies' imaging datasets to look at the region of activation. This could help explain the mixed findings with localized amygdala hyper-, hypo-, and typically developing activations found in studies with social and emotional tasks as seen in a 2018 study that found increased functional connectivity involving the bilateral amygdala with decreased functional connectivity involving the right amygdala.[47] Further, future secondary analyses could benefit from databases with more accessible data that accounts for all categories of effect like sex, IQ, and comorbidities.

Finally, future research should also move towards multimodal studies, as all studies discussed in this review were singular in fMRI results. For example, one study which pushes for multi-modal studies discusses the possibility of imaging genetics to help explain and clarify ASD and intellectual disability pathology better. In this specific scenario, imaging genetics could help determine a gene that is potentially implicated in both ASD and ID, shared common biological pathways that have certain structures and functions. The study talks about the possibility of genotyping sample individuals to obtain more information compared with phenotypic characteristics such as IQ or age.[45]. Outside of atypical activations, functional connectivity studies have remained unimodal due to the difficulties of synthesizing across imaging modalities including electroencephalography (EEG) and magnetoencephalography (MEG). Integrating these models together could help accommodate for inconsistency in findings resulting from one model.[48] Research can be confirmed through different modes, thus helping with low sample size and representation issues if the trend holds in other circumstances. Through quality study, neuroimaging could have huge benefits on the objectivity of diagnosis and understanding of the etiology and pathophysiology of autism.

0.1 Strengths & Limitations

Strengths of this literature review include its diversity of sources and unique focus on amygdala dysfunction and autism. Sources included fMRI, MRI, longitudinal, literature reviews, and meta-analytic studies. The focus on amygdala activity from fMRI studies and future directions is beneficial in clarifying findings and trends, as well as proposing ways to modify existing theories. Limitations include the synthesis of many sources with different procedures, geographic limitations given that the majority of studies were US based, and the exclusion of various related studies including ASD individuals with comorbidities, animal trials, and clinical trials. Synthesizing across various studies could weaken an argument due to the possibility of error. The majority of studies were produced in the US, but several originated in Japan, Germany, and South Korea which may potentially yield different results due to cultural differences. Though the specific focus on the amygdala can be a strength, it also limits the scope of this literature review as the review did not synthesize across other types of studies like clinical trials or comparisons with comorbid disorders like anxiety disorder and ADHD.

Conclusion

Research indicates that 'the amygdala theory of autism' is consistent in atypical neural activity in the amygdala, but cannot explain such inconsistencies. There is a growing shift away from the idea of the amygdala having a direct role and towards the amygdala's role in connectivity networks with

other regions and structures of the brain. Evidence of domain-specific dysfunctions and alternate pathways emerge, including atypical connectivity in the amygdala in concordance with other structures and the upregulation and downregulation of two neural structures. This also exists with functional connectivity networks, where increased connectivity between two structures could substitute for decreased connectivity between two other structures. Looking at fMRIs of amygdala localizations is beneficial to other areas such as neural correlates of behavior and amygdala function in ASD individuals compared with typically developing groups. Future recommendations for research include more utilization of a neuroimaging database and increased representation in ASD subgroups. One last area of focus should be the integration of multiple neuroimaging methods of study. Though this review only focused on fMRI, MEG- and EEG-focused studies also have different results which could be improved via integration of the different neuroimaging methods. Doing so may yield increased convergent validity and can better support effective advances in research for targets of intervention and the understanding of autism.

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Author (s)	Title	Domain	Summarized Findings
Baron-Cohen S, Ring H, Bullmore E, Wheelwright S, Ashwin C, & Williams S.	The amygdala theory of autism.	Face Emotion Processing	fMRI study where adults with high-functioning autism participated in the "Mind in the Eye Test," judging emotions from faces. Results showed significant hypoactivation in the amygdala.
Hadjikhani N, Joseph R, Snyder J, & Tager-Flusberg H.	Abnormal activation of the social brain during face perception in autism.	Face Emotion Processing	fMRI study where adults with autism viewed non-emotional faces passively. Results showed hypoactivation in a network of brain areas including the right amygdala, inferior frontal cortex (IFC), superior temporal sulcus (STS), and face-related somatosensory and premotor cortex.
Tottenham N, Hertzig M, Gillespie-Lynch K, Gilhooly T, Millner A, & Casey B.	Elevated amygdala response to faces and gaze aversion in autism spectrum disorder.	Face Emotion Processing	fMRI study where individuals with autism participated in natural viewing and eye gaze manipulation conditions. Results showed hyperactivation of amygdala in response to natural viewing of faces and hyperactivation of amygdala in response to gaze manipulation of neutral, but not angry, faces.
Ibrahim K, Eilbott J, Ventola P, He G, Pelphrey K, McCarthy G, & Sukhodolsky D.	Reduced Amygdala-Prefrontal Functional Connectivity in Children with Autism Spectrum Disorder and Co-occurring Disruptive Behavior.	Face Emotion Processing, Functional Connectivity	fMRI study where children with autism participated in face emotion perception task of fearful and calm faces. Results showed hypoconnectivity between the amygdala and vlPFC connectivity in children with disruptive behavior compared to without.
Pierce K, Haist F, Sedaghat F, & Courchesne E. The brain response to personally familiar faces in autism: findings of fusiform activity and beyond.	The brain response to personally familiar faces in autism: findings of fusiform activity and beyond.	Face Recognition Processing	fMRI study where adults and adolescents with autism participated in pressing buttons in response to familiar and stranger faces. Results showed significantly more limited network response to familiar faces which included the amygdala and other limbic structures.
Hadjikhani N, Joseph R, Manoach D, Naik P, Snyder J, & Dominick K et al.	Body expressions of emotions do not trigger fear contagion in autism spectrum disorder.	Body Emotion Processing	fMRI study where adults with high functioning autism participated in a task on perceiving bodily expressed emotions (fear and neutral). Results showed no significant neural activation differences in response to fear and neutral conditions.
Green S, Rudie J, Colich N, Wood J, Shirinyan D, & Hernandez L. et al.	Overreactive Brain Responses to Sensory Stimuli in Youth with Autism Spectrum Disorders.	Sensory Stimuli Processing	fMRI study where children with autism participated in responding to mildly aversive auditory and visual stimuli. Results showed hyperactivation of the amygdala, primary sensory cortical areas, hippocampus, and orbital-frontal cortex.
Green S, Hernandez L, Tottenham N, Krasileva K, Bookheimer S, & Dapretto M.	Neurobiology of Sensory Overresponsivity in Youth with Autism Spectrum Disorders.	Sensory Stimuli Processing	fMRI study where children with autism participated in responding to mildly aversive auditory and tactile stimuli. Results showed hyperactivation of the amygdala and primary sensory cortical areas.
Rosenblau G, Kliemann D, Dziobek I, & Heekeren H.	Emotional prosody processing in Autism Spectrum Disorder.	Emotional Prosody Processing	fMRI study where adults with autism participated in a task involving emotional prosody processing, or judging emotion from the pitches/tones of

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			speech. Results showed hypoactivation of amygdala and hypoconnectivity between the amygdala and STS for complex compared to basic emotions.
Schneider K, Pauly K, Gossen A, Mevissen L, Michel T, & Gur R et al.	Neural correlates of moral reasoning in autism spectrum disorder.	Moral Reasoning	fMRI study where adults with autism judged textual dilemma situations followed by proposed solutions to which they could agree or disagree. Results showed hypoactivation of amygdala.
Park S, Son J, Chung S, Lee S, Ghim H, & Lee S. et al.	Autism and Beauty: Neural Correlates of Aesthetic Experiences in Autism Spectrum Disorder.	Aesthetic Judgment	fMRI study where adolescents with autism participated in aesthetic judgment tasks on two types of art: magnificent landscape images and fractal images. Results showed hyperactivation of the amygdala in response to fractal images.
Swartz J, Wiggins J, Carrasco M, Lord C, & Monk C.	Amygdala Habituation and Prefrontal Functional Connectivity in Youth with Autism Spectrum Disorders.	Habituation	fMRI study where children and adolescents with autism were observed for amygdala habituation individually and in connection to vmPFC while performing gender identification on a variety of faces. Results showed decreased habituation of amygdala in response to sad and neutral faces, and abnormal vmPFC-amygdala functional connectivity correlated with decreased habituation.
Abrams D, Padmanabhan A, Chen T, Odriozola P, Baker A, & Kochalka J et al.	Impaired voice processing in reward and salience circuits predicts social communication in children with autism.	Reward Circuitry	fMRI on voice-selective cortex study (activation in response to voice of mother and unfamiliar voice) only found hyperactivation of the amygdala in response to unfamiliar voices. suggests hypo is reward specific
Dichter G, Richey J, Rittenberg A, Sabatino A, & Bodfish J.	Reward Circuitry Function in Autism During Face Anticipation and Outcomes.	Reward Circuitry	fMRI study where adults with autism participated in reward anticipation and outcome for monetary and social rewards. Results showed hyperactivation of the amygdala in response to face anticipation (social award anticipation).
Abrams D, Lynch C, Cheng K, Phillips J, Supekar K, & Ryali S et al.	Underconnectivity between voice-selective cortex and reward circuitry in children with autism.	Reward Circuitry, Functional Connectivity	fMRI study where children with autism participated in resting state functional connectivity imaging. Results showed hypoconnectivity between the amygdala, right-hemisphere pSTS, and the orbitofrontal cortex.
Fishman I, Linke A, Hau J, Carper R, & Müller R.	Atypical Functional Connectivity of Amygdala Related to Reduced Symptom Severity in Children with Autism.	Functional Connectivity	fMRI study where children and adolescents with autism participated in resting state functional connectivity imaging. Results showed reduced functional connectivity between bilateral amygdalae and left inferior occipital cortex, with greater connectivity between right amygdala and right sensorimotor cortex.
Shen M, Li D, Keown C, Lee A, Johnson R, & Angkustsiri K et al.	Functional Connectivity of the Amygdala Is Disrupted in Preschool-Aged Children with Autism Spectrum Disorder.	Functional Connectivity	fMRI study where preschool aged children with autism participated in resting state functional connectivity imaging. Results showed hypoconnectivity between amygdala and several brain regions including bilateral medial prefrontal cortex, temporal lobes, and striatum.
von dem Hagen E,	Reduced functional connectivity	Functional	fMRI study where adults with autism participated

Stoyanova R, Baron-Cohen S, & Calder A.	within and between 'social' resting state networks in autism spectrum conditions.	Connectivity	in resting state functional connectivity imaging. Results showed hypoconnectivity between the amygdala, insula, and medial temporal lobe network.
Guo X, Duan X, Long Z, Chen H, Wang Y, & Zheng J et al.	Decreased amygdala functional connectivity in adolescents with autism: A resting-state fMRI study.	Functional Connectivity	fMRI study where adolescents with autism participated in resting state functional connectivity imaging. Results showed hypoconnectivity between the amygdala and subcortical regions including the bilateral thalamus and right putamen.
Rausch A, Zhang W, Haak K, Mennes M, Hermans E, & van Oort E et al.	Altered functional connectivity of the amygdaloid input nuclei in adolescents and young adults with autism spectrum disorder: a resting state fMRI study.	Functional Connectivity	fMRI study where adolescents and adults with autism participated in resting state functional connectivity imaging. Results showed hypoconnectivity between the amygdaloid sensory input channels, and difference in functional connectivity between amygdala nuclei for input and output.

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