Social Dysfunction in Autism Spectrum Disorder: Tactile Processing and Oxytocin

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Date: 2022-12-28T00:00:00+00:00

Abstract

Autism spectrum disorder (ASD) is characterized by persistent social dysfunction as one of its core symptoms. In recent years, numerous studies have demonstrated that tactile input can influence social function by modulating the oxytocin system. Meanwhile, the social salience theory of oxytocin, proposed in recent years, posits that oxytocin can enhance the salience of social information by increasing activation of corresponding brain regions. Within this theoretical framework, when social interaction occurs, tactile input enhances oxytocin synthesis and release, while simultaneously oxytocin increases the salience of tactile information, thereby further promoting social interaction. Based on the social salience theory of oxytocin, abnormal tactile sensitivity and oxytocin system dysfunction in individuals with ASD may disrupt the bidirectional regulatory mechanism between tactile input and the oxytocin system, which may constitute one of the reasons for their social functional deficits. Investigating the relationship among tactile input, the oxytocin system, and social function can help us understand the pathogenesis of social dysfunction and provide new insights for future prevention and intervention strategies.

Full Text

Introduction

Autism spectrum disorder (ASD) is a lifelong neurodevelopmental condition characterized by persistent social dysfunction, repetitive behaviors, and atypical sensory responses. Symptom severity varies widely among individuals, with many displaying comorbid conditions such as depression, anxiety, sleep disturbances, and ADHD. Approximately 30% of individuals with ASD require psychological and psychiatric interventions, including pharmacological treatments for behavioral problems. However, no medications currently target the core symptoms of ASD, making it crucial to investigate the underlying mechanisms

of these symptoms to develop effective interventions. While the precise pathophysiology remains unclear, social dysfunction represents a key observable outcome. Normal social development requires appropriate sensory stimulation during early life; from a psychological perspective, without sensory input, higher cognitive abilities cannot emerge.

Tactile sensation has garnered increasing attention in recent years. From a developmental standpoint, touch is unique among sensory modalities in its relatively early maturation. Social touch holds profound significance throughout the lifespan. Fetuses in late gestation respond to maternal abdominal touch by actively exploring the uterine wall, and researchers suggest that these fetal movements, mediated by amniotic fluid, facilitate vestibular development and provide comfort through rhythmic motion. Disruption of tactile input during infancy severely impairs social functioning in adulthood, while infants deprived of caregiver touch exhibit abnormal tactile processing, such as heightened tactile sensitivity. For children and adults, social touch involves more complex physical contact with deeper social meaning. From an evolutionary perspective, the ubiquity of tactile contact among primates underscores its importance for social cohesion, with grooming and embracing strengthening intragroup bonds and maintaining social relationships.

Recent research has extensively examined the relationship between tactile processing and social function in ASD. Individuals with ASD demonstrate atypical tactile processing mechanisms, including both hyper- and hyposensitivity, compared to typically developing individuals. These abnormal tactile responses may cause individuals with ASD to either avoid touch due to oversensitivity or ignore it due to undersensitivity. Avoidance of social touch during infancy predicts later ASD symptoms, suggesting that atypical tactile responses may constitute a predisposing factor for social dysfunction. The oxytocin system represents another widely studied mechanism in ASD social impairment. Oxytocin is essential for prosocial behaviors, including maternal-infant attachment and adult social interactions. Research indicates that tactile input can regulate social behavior through the oxytocin system; for instance, skin-to-skin contact between parents and children significantly increases plasma oxytocin concentrations, facilitating bonding. Massage and stroking activate oxytocinergic neurons, leading to elevated plasma oxytocin levels.

The oxytocin system may thus serve as a critical link between tactile sensation and social function. Previous work has distinguished between discriminative touch, which identifies tactile stimulus properties, and affective touch, which conveys emotional information. Affective touch, carrying social significance, is more likely to influence social function via oxytocin system modulation. Investigating the interplay between touch and oxytocin in social function may illuminate the developmental mechanisms underlying ASD social dysfunction and inform future intervention strategies. This review synthesizes recent findings and, based on the social salience hypothesis of oxytocin, analyzes how potential dysregulation between tactile and oxytocin systems may contribute to social

dysfunction in ASD.

2 Atypical Tactile Responses and Processing Patterns in ASD

Atypical sensory responses, particularly to tactile stimuli, are common in ASD and have been incorporated into diagnostic criteria. These abnormal responses are characterized as either over-responsiveness (hypersensitivity) or under-responsiveness (hyposensitivity). Research reveals that 12-month-old infants with ASD may exhibit both tactile hyposensitivity and hypersensitivity simultaneously, while children aged 6-13 years show increased sensitivity to tactile and painful stimuli. These atypical patterns can be understood at both peripheral and central nervous system levels, manifesting as abnormal tactile thresholds peripherally and atypical activation in somatosensory and insular cortices centrally.

2.1 Abnormal Affective Touch Thresholds in ASD

Individuals with ASD show differential responses to distinct tactile modalities. Affective touch, encoded by unmyelinated C-fibers, transmits social-emotional information through gentle stroking at optimal velocities (1-10 cm/s), which maximally activates these fibers and correlates with subjective pleasantness ratings. Discriminative touch, conversely, is mediated by myelinated A-fibers. The affective touch hypothesis posits that C-fibers constitute a specialized channel for socially meaningful gentle touch. Studies demonstrate that children with ASD exhibit lower peripheral tactile thresholds for affective touch, showing reduced response thresholds to facial and dorsal hand stimulation—areas processed by C-fibers—alongside higher temporal discrimination thresholds and reduced sensory seeking behaviors. Notably, while typically developing children show differential thresholds between palm and dorsal hand, children with ASD do not, suggesting modality-specific atypical sensitivities. These threshold abnormalities may underlie specific behaviors, such as avoidance of affective touch due to low thresholds.

2.2 Atypical Tactile Processing Patterns in ASD

Following peripheral transmission, individuals with ASD display abnormal activation in social brain networks, including regions processing affective touch. C-fiber signals project to the insular cortex, where emotional significance is extracted. After entering the spinal cord via dorsal root ganglia, these signals ascend through the spinothalamic tract to the thalamus and sensory cortex, with the ventromedial posterior nucleus relaying information to the dorsal posterior insular cortex. The insula integrates affective inputs from multiple modalities, while additional regions including ventral striatum, posterior superior temporal sulcus, medial prefrontal cortex, dorsal anterior cingulate cortex, and orbitofrontal cortex also process C-fiber input. Neuroimaging studies reveal

that children and adolescents with ASD show reduced activation in social brain networks—including bilateral insula, right posterior superior temporal sulcus, bilateral temporoparietal junction, right fusiform gyrus, right amygdala, and bilateral ventrolateral prefrontal cortex—when processing C-fiber-mediated touch, while showing enhanced sensory cortex responses to discriminative palm touch. This pattern of reduced affective and enhanced non-affective cortical processing suggests that lower activation in social brain regions may contribute to atypical sensory sensitivity.

Atypical sensory sensitivity is hypothesized to result from altered excitation-inhibition (E/I) ratios in cortical activity, related to neurotransmitter systems such as GABAergic and glutamatergic signaling. In ASD children, feedforward inhibition correlates negatively with symptom severity, and this inhibition is associated with GABAergic function. Magnetic resonance spectroscopy has revealed elevated glutamate and glutamine levels in primary sensorimotor cortex of children with ASD, correlating with parental reports of sensory hypersensitivity and hyposensitivity, while GABA levels remain unchanged. Conversely, adult ASD patients show reduced sensorimotor cortex GABA concentrations that correlate positively with self-reported tactile hypersensitivity. These inconsistent GABA findings may reflect compensatory mechanisms such as reduced GABA receptor expression or contributions from other neurotransmitters like glutamate. Animal models support these observations, with Cntnap2 knockout mice showing enhanced local connectivity in primary somatosensory cortex and altered object discrimination, while Shank3 and Cntnap2 models exhibit reduced parvalbumin-expressing basket cells in somatosensory cortex layer 2/3, leading to decreased inhibitory input and tactile hypersensitivity. These findings collectively suggest that abnormal neurotransmitter activity in somatosensory cortex underlies atypical sensory sensitivity in ASD, though future research must examine E/I alterations in insular cortex, ventral striatum, and other social brain regions to fully explain atypical affective touch experiences.

3 Social Function Regulation Through Tactile-Oxytocin Interactions

Tactile input activates the oxytocin system, which in turn regulates social function and promotes prosocial behavior. The social salience hypothesis of oxytocin, proposed in recent years, explains oxytocin' s prosocial effects by suggesting it enhances attention to social cues by increasing activation in relevant brain regions. This framework provides a new theoretical perspective for understanding how oxytocin influences tactile processing. Atypical tactile responses in ASD may disrupt the regulatory mechanism linking touch, oxytocin, and tactile information processing, thereby impairing social function.

3.1 Tactile Input Regulates the Oxytocin System to Enhance Social Function

Gentle skin contact induces oxytocin release, reducing stress, enhancing well-being and social motivation, and facilitating social bonding. During mother-infant bonding, gentle touch from infants to maternal breasts increases maternal oxytocin release, while 10 minutes of skin contact between partners significantly elevates plasma oxytocin concentrations. In rodents, five minutes of gentle back stroking increases ultrasonic vocalizations indicative of positive affect and activates hypothalamic paraventricular nucleus oxytocin neurons. This oxytocin release mechanism appears mediated by C-fiber-transmitted affective touch, which selectively responds to gentle, slow stroking. The resulting reduction in stress and enhancement of social behavior closely mirror oxytocin' s effects, suggesting C-fibers influence social function via oxytocin system modulation.

Affective touch enhances social preference through oxytocinergic mechanisms. In healthy adults, salivary oxytocin increases correlate with subjective pleasantness ratings of C-fiber-activating touch. Animal studies demonstrate that tactile stimulation of juvenile mice at 3 cm/s activates a tachykinin 1-oxytocin neuron projection from the ventrolateral periaqueductal gray to the paraventricular nucleus, increasing adult social behavior and conditioned preference for cotton. Similar findings show that gentle stroking induces paraventricular oxytocin neuron activation and conditioned preference for the experimenter' s hand in rats, while in female mice, C-fiber input activates parvocellular oxytocin neurons to promote social interaction. These results collectively indicate that affective touch triggers oxytocin synthesis and release, thereby enhancing social preference.

Regarding social motivation, tactile input carries inherent social meaning beyond simple sensory stimulation. Touch behaviors such as stroking and hugging convey complex messages like "I want to connect," "I like you," and "I care," serving as crucial social bonding agents. Normal physical contact in social contexts provides feedback signals that help maintain social motivation. The social motivation theory posits that oxytocin and dopamine are key neuroendocrine hormones sustaining the reward value of social behavior. ASD social motivation deficits may stem from reduced levels of these neurotransmitters, potentially exacerbated by decreased physical interaction due to atypical tactile sensitivity, which fails to properly activate the oxytocin system.

In social cognition, ASD primarily manifests as Theory of Mind deficits—the ability to infer others' mental states and intentions. Early social interaction is critical for Theory of Mind development, enabling emotional sharing, imitation, and social preference learning. Avoidance of social touch in ASD infants compromises interaction quality and impedes this developmental trajectory. Additionally, atypical tactile processing may impair emotion recognition. In one study, ASD patients showed stronger N2 responses (related to emotional arousal) in anterior cingulate and anterior insular cortices when evaluating pain in others, but

reduced medial prefrontal activation, indicating heightened emotional arousal alongside diminished emotional understanding.

3.2 Oxytocin Enhances Social Information Salience by Regulating Tactile Processing

If tactile stimulation regulates the oxytocin system, does oxytocin conversely influence tactile information processing? The social salience hypothesis suggests oxytocin modulates attentional orientation toward social cues, enhancing cooperativeness in social contexts while potentially increasing competitiveness in competitive settings, with effects varying by sex and personality. Intranasal oxytocin administration enhances responses to personally relevant stimuli regardless of their social nature or emotional valence. In 4-month-old infants, C-fiber-activating touch enhances social information salience, paralleling oxytocin' s effects. Both exogenous oxytocin and touch-induced endogenous oxytocin release appear to amplify social information salience.

The neurobiological assumption is that oxytocin selectively enhances brain activity in response to social stimuli. Animal studies demonstrate that endogenous oxytocin release in the anterior olfactory nucleus increases neuronal excitability and projections to olfactory bulb granule cells, heightening responses to social odors. In pain processing, the paraventricular nucleus directly projects oxytocinergic fibers to the rostral agranular insular cortex. While direct evidence for oxytocin' s effects on sensory cortex projections remains limited, rodent studies show oxytocin modulates excitatory synaptic transmission in sensory cortex, with oxytocin synthesis decreasing under sensory deprivation.

Clinical research reveals that intranasal oxytocin enhances attention to eye regions in ASD patients during naturalistic interactions and increases gaze duration toward eyes in control subjects. Oxytocin also increases attentional resources allocated to both social and non-social stimuli, suggesting a general enhancement of attentional salience rather than selective social attention. This effect occurs even without conscious awareness, indicating oxytocin modulates attentional bias. Notably, the salience network shows hyperactivation in ASD children and infants, which may limit social interaction by flooding the system with unfiltered sensory information. Oxytocin administration can reduce salience network connectivity in older adults, suggesting a top-down regulatory role that helps filter irrelevant information and direct attention toward meaningful social cues.

Human studies also demonstrate oxytocin's modulation of tactile processing. Intranasal oxytocin increases the likelihood of categorizing photos of inanimate objects touching as affective images, consistent with the social salience hypothesis. Oxytocin also enhances tactile sensitivity, reducing peripheral mechanical detection thresholds, and increases pleasantness of affective touch while boosting orbitofrontal cortex activation. This enhancement occurs regardless of emotional valence, suggesting oxytocin amplifies tactile salience by increasing pleasantness

across stimulus types. Furthermore, hand massage elicits stronger endogenous oxytocin release than machine massage, with activation in affective touch processing regions like posterior superior temporal sulcus and orbitofrontal cortex, but not in primary somatosensory cortex. This indicates stimulus-dependent modulation, with oxytocin potentially projecting to both somatosensory and insular cortices.

In summary, affective touch increases oxytocin release, which may enhance attentional bias toward tactile information by modulating cortical responses, thereby increasing tactile salience. Reduced attention to social information contributes to ASD social dysfunction, and oxytocin may counteract this by enhancing insular activation and pleasantness of affective touch experiences.

4 How Tactile and Oxytocin System Abnormalities Lead to ASD Social Dysfunction

Research examining how atypical tactile responses affect social function in ASD has yielded mixed results. Some studies in adults indicate tactile hypersensitivity primarily mediates the relationship between ASD and social dysfunction, while research in children suggests early sensory hypersensitivity positively predicts later social development. Two-year-old children with ASD who displayed high sensory sensitivity showed stronger social function at age four, associated with enhanced attention to faces. This suggests that sensory hypersensitivity may help children with ASD notice social information, potentially facilitating social development. However, these discrepancies likely arise from failure to distinguish between affective and discriminative touch, which have independent processing pathways and distinct roles in social function. Parental reports of sensory sensitivity also encompass multiple sensory modalities beyond touch.

According to the social salience hypothesis, oxytocin neurons project to various cortical regions, including somatosensory cortex for discriminative touch and insular cortex, ventral striatum, posterior superior temporal sulcus, medial prefrontal cortex, and dorsal anterior cingulate cortex for affective touch. Hypersensitivity to affective touch may lead to tactile avoidance, preventing normal oxytocin system activation during social interactions and resulting in reduced cortical activation. Infant ASD patients who avoid tactile stimulation show predictive patterns for later symptom severity. Reduced tactile input may functionally resemble early tactile deprivation, disrupting the development of neural circuits underlying social function.

Tactile input may also influence social function by modulating oxytocin and other neuroendocrine systems. Oxytocin may reduce cortical activation by affecting neurotransmitter systems including GABA, serotonin, and glutamate. Abnormal tactile responses correlate with altered cortical inhibition, and rodent studies show oxytocin increases extracellular GABA concentrations in medial prefrontal cortex and dorsal hippocampus. Oxytocin also modulates serotonin systems and enhances frontostriatal dopaminergic connectivity in ASD. Tactile

input itself affects reward function through mesolimbic dopamine pathways—maternal licking in rodents increases dopaminergic neurons in ventral tegmental area and dopamine receptor mRNA in nucleus accumbens, while massage promotes dopamine release in humans. Oxytocin interacts with dopamine to influence reward, and socially meaningful touch behaviors like hugging and stroking have inherent reward value. Through classical conditioning, social partners associate touch with the pleasure of oxytocin and dopamine release, gradually building social relationships. Additionally, tactile input may reduce social anxiety by inhibiting amygdala activity and lowering corticosterone levels, thereby enhancing trust and reducing bias during social interactions.

Oxytocin system dysfunction itself may contribute to ASD social impairment. Children with ASD exhibit lower plasma oxytocin levels than typically developing children, without the normal age-related increase. Higher plasma oxytocin correlates with better verbal communication in ASD. Genetic studies link ASD symptoms to single nucleotide polymorphisms (SNPs) in the oxytocin receptor gene. However, these oxytocin system alterations are not unique to ASD, suggesting a general role in social function. Lower plasma oxytocin correlates with Theory of Mind deficits in ASD. A 2014 review proposed that oxytocin dysfunction may cause ASD symptoms by disrupting interoception—the sense of internal bodily states. Oxytocin normally attenuates interoceptive signals and enhances external social information salience. Early pathophysiological changes in the oxytocin system may disrupt integration of interoceptive and exteroceptive signals, impairing self-concept formation and attention to social stimuli. This hinders observational learning through imitation, preventing adequate development of language, social interaction, and Theory of Mind. As a form of interoception with affective and motivational components, tactile sensation conveys both physical properties and emotional meaning from early development, as evidenced by fetal responses to maternal touch.

5 Intervention Strategies for ASD Social Deficits Through Touch and Oxytocin

Intervening in the oxytocin system may represent a crucial approach for improving social dysfunction in ASD. Exogenous oxytocin administration shows promise but remains controversial due to small sample sizes, limited replicability, artificial experimental settings, and side effects such as nasal irritation and headaches. Tactile input offers an alternative intervention pathway. Before tactile information reaches the thalamus and sensory cortex via the spinothalamic tract, it can access downstream brain regions including the hypothalamus to trigger oxytocin release. In non-human primates, C-fiber activation accelerates attention to experimental images regardless of social content. Tactile stimulation also facilitates cross-modal emotional processing of vocal and visual information, similar to oxytocin' s attention-enhancing effects. Neuroimaging studies show that intranasal oxytocin alters activation in amygdala, medial prefrontal cortex, and anterior cingulate cortex during emotion recognition tasks.

While intranasal administration increases cerebrospinal fluid oxytocin levels in both humans and primates, no studies have directly compared cerebrospinal fluid concentrations between ASD and typically developing individuals, though plasma oxytocin is consistently lower in ASD. Intravenous oxytocin injection enhances social cognition and reduces repetitive behaviors in ASD, but primate studies show it alters plasma without affecting cerebrospinal fluid oxytocin, suggesting peripheral mechanisms may mediate its effects. Future interventions should consider both central and peripheral oxytocin actions.

Since gentle tactile input promotes oxytocin release and oxytocin enhances touch pleasantness and sensitivity, combining exogenous oxytocin administration with tactile training may prove more effective for improving social function. Early intervention is critical—animal studies demonstrate that neonatal oxytocin treatment rescues thermosensory dysfunction and restores hippocampal function and social memory in Magel2 knockout mice. Early-life oxytocin administration also reverses social deficits induced by whisker trimming (a form of tactile deprivation) in mice, whereas adult intervention produces only temporary effects. This suggests a critical period around 3-6 years of age when oxytocin intervention might improve social dysfunction, though further validation is needed. Parent-child interaction is bidirectional—early atypical tactile responses in ASD children may reduce interaction quality and tactile input, functionally resembling tactile deprivation and leading to adult social dysfunction. Since early sensory input lays the foundation for higher cognitive functions, early tactile stimulation is essential for developing neural circuits underlying social behavior.

6 Summary and Outlook

This review has discussed how tactile input regulates the oxytocin system and, based on the social salience hypothesis, proposed pathways through which oxytocin influences tactile processing. In typical development, tactile input enhances oxytocin synthesis and release, which increases tactile information salience and promotes social interaction. In ASD, oxytocin system deficits reduce tactile salience, decreasing attentional resources for social interaction and affecting emotional touch experiences. Simultaneously, oxytocin dysfunction impairs Theory of Mind and emotion perception. Atypical tactile sensitivity leads to social avoidance, reducing oxytocin release during social contact, which diminishes social motivation and preference, ultimately causing social dysfunction (Figure 1 [Figure 1: see original paper]).

Understanding the connections among tactile stimulation, the oxytocin system, and social function can elucidate how ASD social deficits emerge developmentally from early life. Future research should address several key questions:

6.1 Investigating Oxytocinergic Circuits in Somatosensory and Insular Cortices

The social salience hypothesis proposes that oxytocin enhances social information by increasing activity in corresponding brain regions. Tactile stimulation may induce oxytocin release to enhance activation in somatosensory and insular cortices, potentially through modulation of different neurotransmitter systems. Future studies should use neurobiological techniques in animal models to confirm the specific circuits and biochemical mechanisms through which oxytocin regulates tactile processing.

6.2 Distinguishing Effects of Affective Versus Discriminative Touch on Social Development

While tactile input' s role in prosocial behavior is well-established, maternal touch during mother-infant interaction is difficult to categorize and confounds multiple variables. Future research should systematically examine how different tactile stimulus types influence social development across the lifespan. Due to ethical constraints and long developmental timelines in humans, animal models could simulate specific tactile inputs during early development to test adult social function and investigate underlying neural circuit changes.

6.3 Examining Multisensory Integration in ASD

Beyond tactile abnormalities, individuals with ASD show similar hyper- and hyposensitivity in other sensory modalities. Brain regions showing reduced responses to affective touch also exhibit deficits in processing social-emotional information from visual and auditory stimuli, suggesting shared processing mechanisms across modalities. Since adequate social function requires accurate perception and timely response to social cues from multiple senses, ASD social dysfunction likely results from abnormalities across sensory channels. Future research must integrate findings across modalities and examine interactions between sensory systems to provide a comprehensive understanding of social dysfunction in ASD.

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