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Examining the Role of the Mirror Neuron System in Social Anxiety Disorder and Borderline Personality Disorder: Pathophysiological Insights and Treatment Perspectives

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Abstract

Social Anxiety Disorder (SAD) and Borderline Personality Disorder (BPD) both involve serious social and emotional impairments, though their origins and clinical features differ. SAD is characterized by a persistent fear of social judgment, while BPD stems from early relational trauma and is marked by emotional instability and impulsivity. Both disorders show disrupted social cognition and hypersensitivity to rejection. Emerging evidence points to dysfunction in the mirror neuron system (MNS), This network that we possess allows us to interpret the actions, intentions, and feelings of other people by reflecting them in our brain. Research shows people with SAD often have reduced activity in core MNS areas like the inferior frontal gyrus and inferior parietal lobule, especially during tasks involving emotional recognition or social observation. These deficits may explain common SAD symptoms such as the like for social withdrawal, fear of evaluation, and trouble learning from social interactions. In BPD, the picture is less clear. Some studies suggest disrupted MNS function, but inconsistent methods and limited comparative data make it hard to draw solid conclusions. Still, both disorders involve altered brain connectivity in regions tied to emotional regulation and social processing, like the amygdala and prefrontal cortex. That matters, especially during adolescence when the MNS is still developing and social vulnerability peaks. Promising treatments are now starting to emerge, like neurofeedback, transcranial magnetic stimulation, and action observation therapy all show early signs of boosting MNS activity and improving social functioning. These tools, combined with cognitive or dialectical behavior therapies, could lead to a more tailored, neuroscience-informed care. Even better, spotting MNS problems early might help prevent full-blown disorders later. And in short, the MNS may be a key player in both SAD and BPD. But to unlock its full potential, we need

better methods, more diverse samples, and long-term studies that track how these brain patterns change over time and how we can use them to heal.

Keywords: *Social Anxiety Disorder (SAD), Borderline Personality Disorder (BPD), Mirror Neuron System (MNS), Social Cognition, Neuroscience informed Interventions*

Introduction

Social Anxiety Disorder (SAD), or social phobia, is a severe and persistent mental health condition that affects millions of people worldwide. It involves an intense fear of social judgment, rejection, or embarrassment. This fear often causes individuals to avoid interactions where they may be scrutinized, such as conversations, group settings, or even simple acts like entering a room or eating in public. Physical symptoms trembling, sweating, blushing often accompany the anxiety, reinforcing avoidance and isolation (1).

Social anxiety disorder (SAD) is more than shyness since it ranks among the most common psychiatric conditions worldwide, with a lifetime prevalence near 12%, often beginning in adolescence. In countries like the U.S. and New Zealand, roughly 10% of adolescents are affected, and this early onset disrupts crucial developmental milestones including forming friendships, academic progress, and building self-confidence (1). Over time, persistent avoidance behaviors may lead to social isolation, academic underachievement, and symptoms of depression. While SAD presents distinct emotional and behavioral features, evidence suggests its development involves neurobiological factors, particularly disturbances in the mirror neuron system (MNS). The MNS, which activates both during personal actions and when observing others, involves brain regions such as the superior temporal sulcus, premotor cortex, and inferior parietal lobule (2,3), supporting basic social functions like empathy, imitation, and intention understanding. Recent research shows that individuals with SAD exhibit altered MNS functioning, demonstrating abnormal responses when observing others' behaviors or emotional expressions, which may reflect difficulty in mirroring others' internal states (4–6). These changes, though less consistent and specific than in disorders like autism, are thought to contribute to challenges in interpreting and responding to social cues, as part of a broader network affecting social perception.

Like SAD, people with Borderline Personality Disorder (BPD) struggle with processing social cues, which often leads to unstable or unpredictable behavior around others. Both disorders share a hypersensitivity to social rejection. People with either condition tend to see unclear social signals as negative, blame themselves when things go wrong, and feel out of control in social settings (7,8). These feelings usually lead to withdrawal, shame, or sadness, which just fuels a cycle of distress.

Both BPD and SAD involve problems with social cognition are basically, understanding what others feel or intend but this difficulty tends to be worse in BPD (9–11). People with BPD might take things way too far, sometimes seeing aggression where there's none or misreading even positive interactions.

When they feel excluded, they can react with aggression or emotional outbursts, which isn't usually seen in SAD (7,8). These differences seem to come from how each disorder handles emotions, and maybe how the mirror neuron system works differently in each.

The social problems in both SAD and BPD might share a common root in the brain's mirror neuron system (MNS). Herewith we shall consider how MNS dysfunction might explain both the shared and unique social challenges in these disorders, and whether it could be a common neurobiological factor.

Methodology

This narrative review synthesizes evidence from neurobiological, psychophysiological, and cognitive studies examining the role of the mirror neuron system (MNS), social cognition, and affective regulation in Borderline Personality Disorder (BPD) and Social Anxiety Disorder (SAD). It spans literature published between January 2013 and August 2025, reflecting over a decade of research into the neural mechanisms underlying social and emotional dysfunction in these disorders. Relevant literature was identified through comprehensive searches of PubMed, Scopus, PsycINFO, and Google Scholar. Keywords included: *"mirror neuron system," "MNS," "social cognition," "mu suppression," "emotion regulation," "amygdala," "prefrontal cortex," "BPD," "borderline personality disorder," "SAD," and "social anxiety disorder".* We used Boolean operators and MeSH terms to guide and refine database searches. Additional sources were located by manually screening the reference lists of key publications, review articles, and meta-analyses. The review included peer-reviewed original research articles, neuroimaging studies (fMRI, EEG, TMS, fNIRS), meta-analyses, and systematic reviews that investigated social cognitive processes, MNS functioning, or neural circuitry relevant to BPD and SAD. Only studies involving human participants diagnosed with BPD and/or SAD according to DSM-IV or DSM-5 criteria were considered. Articles had to report on mechanisms of empathy, imitation, theory of mind, emotion processing, or neural connectivity involving the MNS or related networks (e.g., amygdala-prefrontal circuits, salience network). To ensure scientific relevance and quality, inclusion was limited to studies published in English and indexed in major databases. Studies focusing exclusively on pharmacological interventions, single case reports, animal research, non-peer-reviewed material, and conference abstracts were excluded. Key data extracted from the included studies comprised: sample characteristics, diagnostic criteria, imaging or electrophysiological methods, primary outcomes related to neural activation or connectivity, and interpretations relevant to the social-affective features of BPD and SAD. Findings were synthesized thematically, focusing on shared and distinct neurobiological patterns and their implications for understanding transdiagnostic processes. Given the methodological heterogeneity, no meta-analysis was conducted; instead, the review followed a qualitative integrative approach to highlight converging trends and gaps in current knowledge.

Discussion

The Mirror Neuron System

Mirror neurons were first discovered in the premotor cortex of macaque monkeys. These neurons lit up not only when the monkeys performed an action themselves, but also when they simply watched another monkey doing the same thing. This mechanism, often described as perceptionaction coupling, laid the groundwork for a broader theory of social cognition. Essentially, it helps explain how primates and possibly humans can grasp other people's actions, intentions, and emotional states just by watching. Since then, the mirror neuron system (MNS) has become central to neuroscience models of imitation, empathy, and learning through observation (3).

Apart from primates, mirror neuron systems also appear in songbirds, which use them for learning songs, and bottlenose dolphins, which show comparable skills in gesture communication and possibly self-recognition (3). While animal studies reveal evolutionary foundations of mirroring mechanisms, human MNS research faces methodological constraints. Research on humans has identified reliable neural markers of MNS activity, such as mu rhythm suppression in EEG, occurring during both action execution and observation, confirming evidence of a human mirror system (12).

Further functions of the MNS which have been identified so far relate not only to imitation (of actions, facial expressions, etc.), but also empathy and Theory of Mind (ToM) the ability to infer another person's mental state, beliefs, and desires from their behaviour as well as emotion recognition, and possibly language (3).

This was demonstrated by Schmidt et al., who analysed fMRI activation in 80 participants in 3 tasks, relating each to imitation, empathy, and ToM. As expected, the findings for the 'Imitation' task showed strong activation in areas such as inferior parietal lobule (IPL), premotor cortex, inferior frontal gyrus (BA44), and visual cortex, consistent with mirror neuron system (MNS) involvement. Three 'Empathy' conditions were measured: Affective (a state of empathy produced affectively, through the assumption of the other's affective state, while the observer is aware of the process), Cognitive (where empathy is evoked from cognition of the other's mental state, rather than affectively), and Distress (the level of distress felt by the participant personally) (13).

Each condition activated areas of the brain which have been implicated in the Mirror Neuron System, namely the posterior Superior Temporal Sulcus (pSTS), IPL, inferior frontal gyrus (BA44), amygdala, and visual cortex. The strongest activation was produced by distress, with particular presentation in the bilateral Temporoparietal Junction (TPJ), precuneus, fusiform gyrus, and pSTS. While the TPJ is not traditionally a part of the MNS itself, it is a region prominent for higher-order social cognition, and often interacts with MNS regions to support mentalization and perspective-taking. With regards to tasks invoking a ToM process, there was high activity in pSTS, BA44, insula, premotor cortex, and visual regions (13).

A more refined comparison (ToM vs. Neutral, Imitation vs. Observation) confirmed shared activation in bilateral pSTS, IPL, BA44, and premotor cortex. These findings support the Embodied Simulation hypothesis, reinforce the relevance of a human MNS, and suggest a common neural architecture for distinct social abilities, providing a neural explanation for how people achieve social understanding (13).

Sadeghi et al. further advanced the understanding of the MNS with the novel use of Dynamic Causal Modeling (DCM) for mapping MNS connectivity. This approach refined the model: as well as validating the core architecture of the MNS (that is, forward flow of sensory or social information from the STS to motor regions, supporting inverse models), it additionally revealed task-specific variations in connectivity (14).

Whereas for 'Empathy' and 'ToM', flow was unidirectional from STS to IPL or STS to BA44, 'Imitation' produced a bidirectional sensorimotor loop between STS and BA44, and BA44 and IPL. Thus, not only can perception of an action guide internal motor simulation, but one's own motor experience can also fine-tune one's interpretation of the actions of others (14). Moreover, imitation is distinguished this way from other social tasks in that the mutual reciprocation in the networks indicates active, embodied matching of others' movements, explaining why imitation engages motor systems more directly, and why it is foundational in early (particularly infant) social development .

Neurobiological Profiles of SAD and BPD

With regards to the neurobiological profile of SAD, structural and functional deficits have been investigated using high-resolution structural MRI and resting-state functional MRI (rs-fMRI). In one recent study, when contrasted with healthy controls, SAD patients were found to have reduced grey matter volume (GMV) in a number of areas, namely the bilateral putamen, right thalamus, and left parahippocampus. Widespread functional disconnectivity was reported in the cortico-striato-thalamo-cerebellar network, which is instrumental in emotional regulation, motor planning, and cognitive control. The volume loss found in the putamen and thalamus was linked to altered connectivity between both the putamen and cerebellum, and between the thalamus and middle temporal gyrus and supplementary motor area. The association between the structural deficiencies and the diagnosis of SAD is partially mediated by the alterations in connectivity, indicating that functional disruptions could be a mechanism by which these structural abnormalities lead to the illness (15).

It has long been documented that SAD patients show hyperactivation of the fear circuit (amygdala, insula, anterior cingulate cortex [ACC], and prefrontal cortex [PFC]), increased responsiveness to social threat cues in parietal and occipital areas, and hypoconnectivity in the default mode and dorsal attention networks (which are linked to increased arousal and threat vigilance). It has more recently been demonstrated that SAD involves not just local hyperactivity in the amygdala, but further altered connectivity with regards to emotion discrimination, Affective Stroop (the experience of emotional

words interfering with cognitive control), and fearful face perception. There are also task-specific connectivity changes in the broader, extended amygdala network, and the default mode network. Interestingly, studies have found that measuring connectivity alone can correctly identify SAD patients (16).

While both genetic causes and adverse childhood experiences are considered to play a role in the formulation of BPD, the specific neurobiology producing the disorder has yet to be identified (17).

Mitolo et al. reported structural alterations in the amygdala and insula in BPD patients, including reduced left insular cortical thickness, particulary posterior long gyrus, along with smaller right amygdala volume in females (p-value : 0.037). Functionally, rsfMRI showed weaker amygdala connectivity with the frontal pole, precuneus, and temporal pole, implicating disrupted executive, emotional, and social processing. However, notably, females exhibited heightened anterior insula connectivity with several other regions, suggesting altered self awareness and increased emotional sensitivity". This study also found an association between amygdala alterations and symptoms such as impulsivity and anger rumination. Reduced insula thickness was especially linked to anger rumination, suggesting that both limbic regions are involved in this particular propensity. This neural evidence supports the emotional cascades model (ECM), which proposes a feedback loop between emotional dysregulation and rumination leading to emotional outbursts (18).

A separate study that asked patients to moderate their emotional management compounds this. The results imply that it is more difficult for BPD patients to activate the prefrontal areas normally involved in emotion regulation, suggesting disturbances in direct regulatory pathways and deficiencies in cognitive control mechanisms like emotional reappraisal.

Additionally, avoidance linked to social-emotional distress may be reflected in the decreased engagement of self-referential processing areas (19).

According to another study, major executive control areas such as the orbitofrontal cortex, dorsal ACC, dorsolateral PFC, parietal cortex, and basal ganglia are hypermodulated, and so overruled, as it were, by the amygdala in BPD. This heightened emotional arousal disrupts the brain's ability to regulate behavior under stress, as these regions are heavily involved in the functions of impulse control, attention, and decision-making. Interestingly, using an affective Go/No-Go paradigm the study demonstrated that the disturbances in connection found manifest with greater strength during emotionally-charged demands which require strong self-control. This roots impulsivity and instability in BPD in flaws in neural connectivity, rather than personality per se (20).

The application of psychophysiological interaction (PPI) analysis adds depth, demonstrating how the amygdala influences other regions differently depending on the emotional context. The results also point to a lateralised setback in BPD processing of emotional and cognitive regulation, suggesting a right-hemisphere predominance in the dysregulated network (20).

MNS Dysfunction in SAD

The mirror neuron system shows altered function in individuals with SAD, which likely contributes to their difficulties in social perception and emotion processing. EEG studies reveal that people with anxiety symptoms, including SAD, have atypical MNS activation patterns during social tasks like action observation and emotion recognition. They often recruit additional brain regions, such as frontal and occipital cortices, suggesting compensatory mechanisms to offset reduced MNS efficiency (4,5).

Moreover, MNS activity in SAD is less sensitive to emotional facial expressions, especially negative emotions like fear, which are highly salient to individuals with the disorder. This reduced sensitivity may impair the accurate simulation and recognition of others' emotions, further hindering social interaction (5,6).

Importantly, the MNS functions within a larger network supporting social cognition, including empathy and Theory of Mind. In SAD, abnormalities are also evident in interconnected brain networks such as the default mode and sensorimotor systems, which interact with the MNS and likely contribute to the disorder's symptoms (15,21). However, unlike other conditions such as autism, where MNS dysfunction is more clearly established, evidence in SAD remains emerging and less definitive (22,23).

rsfMRI and neurochemical studies reinforce these findings, showing that frontal regions integral to the MNS exhibit altered integrity and connectivity in SAD, further linking MNS dysfunction to the disorder's pathophysiology (21,24).

MNS Dysfunction in BPD

One study found, however, that although BPD patients showed increased amygdala activity when viewing scenes which were melancholy but tranquil, and therefore low-arousal, such as mourning, the difference was no longer significant when high levels of depressiveness and neuroticism in patients were statistically controlled, suggesting that it is these factors which heightened the amygdala's response. However, when viewing these images of the mourning scenes, BPD patients activated the somatosensory and premotor cortices more than the controls did, a finding that was not associated with their levels of depression or neuroticism. The sensorimotor areas, part of the MNS, in which the BPD patients show increased activation are associated with the phenomenon of emotional contagion, that is, automatically feeling what others feel. It suggests that BPD individuals feel a strong emotional resonance, possibly over-identifying, with the pain of others. In contrast however, areas such as the inferior frontal gyrus, also a part of the MNS, which is necessary for higher-order social reasoning, such as understanding others' intentions and states of mind were less active. Therefore, in individuals with BPD, the MNS may be overactive, resulting in strong, automatic empathy, but without being balanced by higher-level reasoning and more complex interpretation, social cues may be misinterpreted and personalised, causing emotional and interpersonal instability (25).

Additional evidence from TMS-EEG studies targeting the primary somatosensory cortex (S1), a region which is implicated in the tactile mirror system (TaMS), reveals altered connectivity and disrupted sensory integration in these patients both during real, and only observed touch (26). Moreover, these alterations were not specific to the observed touch being directed at a person, or an object, indicating that the disruption may be a general one rather than a failure of empathic mirroring, and suggesting that BPD affects different channels of the MNS in varying ways (26).

Developmental and Sociocultural Influences

Adolescence:

Adolescence is the period in which the areas of a person's brain involved in social thinking, for example, the dorsomedial PFC and TPJ, become more responsive and flexible. This allows the adolescent to form an identity and learn how to interact with others, but also makes them much more vulnerable, especially when going through a negative experience in a social setting (27). If the mirror neuron system (MNS) does not develop as usual, it can affect both how emotions are processed, and how the person perceives themselves and others. increasing the risk of mental health issues like-BPD and SAD (5,25). Mood swings, impulsive actions, and low self-esteem are common in teenagers, but if these patterns persist and worsen leading to self-harm or withdrawal from others as they may signal early warning signs of borderline personality disorder (BPD)(28). These problems often indicate deeper issues in how the person is connecting with others or experiencing empathy, which may involve dysfunction in the social brain, especially the MNS (29).

Culture influence:

Cultural norms surrounding a person's emotional expression and societal expectations influence an individual's way of understanding, their experience and how their brain processes information on emotions. In here, the MNS plays an important role as suggested by many research where, for example the people of America who's culture values emotional expression shows a strong relationship with their own emotion and the activity of the brain region know as visceral somatosensory cortex (dAI), not only that this particular area is also involved in the physical recognition and also helps people experience emotions (30). This suggests that culture affects the brain encoding of emotional states, which is a basic function of MNS-related networks, in addition to how emotions are behaviorally manifested.

Furthermore, studies on the detection of emotions in diverse cultural contexts reveal that immigrant populations react to novel emotional expressions from a range of ethnic origins by exhibiting increased amygdala activity. This supports the idea that cultural familiarity and social significance affect how the brain reacts to emotional cues. This effect may also hold true for the mirror neuron system (MNS), which forms social connections by responding to familiarity and context (31). Additionally, display

norms have an impact on cognitive-emotional frameworks, as demonstrated by cultural disparities in how people perceive emotions, such as Americans' ability to differentiate facial expressions more clearly than Japanese or Russians (32). These frameworks may explain cross-cultural differences in mental health symptoms like social anxiety disorder and borderline personality disorder by influencing how the mirror neuron system (MNS) interprets the emotions of others.

Human Studies:

Nonetheless, applying these results to human psychological disorders such as SAD and BPD has clear limitations. Human brains exhibit significant individual differences in the functioning and organization of social regions. This makes it challenging to identify consistent patterns. In contrast to non-human primate (NHP) research, which can utilize invasive techniques to establish cause-and-effect links in the brain, studies involving humans must depend on non-invasive methods like fMRI that primarily reveal correlations. Additionally, due to the difference of structure and function between NHP and human, there is no standardized tool for direct comparison. These obstacles hinder the direct application of NHP research findings to clinical investigations of SAD and BPD (33).

Despite the limitations of animal studies, they have contributed in improving our knowledge about MNS and its influence on social behavior. However, it is crucial to have human studies to explore further the relationship between MNS and its effect on psychiatric disorders such as SAD and BPD. Studies have shown that the MNS is less active in SAD patients when they are doing imitation and facial mimicry tasks, suggesting that their capacity for embodied simulation in social situations is impaired (5). There is conflicting evidence on BPD. Some research shows increased emotional and sensorimotor mirroring, while others show decreased responses. V This difference can reflect how emotional and sensitive the people are during social situations or while performing any task (25,26).

Specific Studies Linking MNS to SAD and BPD

Most studies focus on schizophrenia and autism. Interest is growing in MNS functioning in Borderline Personality Disorder (BPD) and Social Anxiety Disorder (SAD). Early behavioral and brain imaging studies show changes in empathy and social cue processing. This section presents key studies and findings on mirror neuron system involvement in Borderline Personality Disorder and Social Anxiety Disorder, as detailed in Table 1.1.

Transdiagnostic and Theoretical Implications

The following Table 1.2 outlines key studies linking mirror neuron system (MNS) dysfunction to shared social and emotional deficits in SAD and BPD, providing a clear view of the overlapping neural mechanisms.

Treatment Implications

Indirect MNS Engagement

In social anxiety disorder (SAD), cognitive-behavioral therapy (CBT) remains the primary intervention for enhancing social functioning. Research suggests CBT may also influence the mirror neuron system (MNS) indirectly through modulation of emotion-processing networks. Neuroimaging data show increased activation within dorsolateral and dorsomedial prefrontal cortices regions essential for cognitive control and reappraisal of negative perceptions (35,36). These changes coincide with reduced activation in the insula and medial orbitofrontal cortex, indicating a recalibration between regulatory and affective circuits (37).

For borderline personality disorder (BPD), interventions specifically address emotional dysregulation and unstable interpersonal patterns. Dialectical behavior therapy (DBT) extends CBT principles by integrating mindfulness practice, affect regulation strategies, and validation methods, adapted to the clinical characteristics of BPD. Mentalization-based treatment (MBT) adopts a psychodynamic framework, strengthening the capacity for mentalization the ability to interpret one's own and others' mental states during social interaction (38).

Potential MNS-Targeted Therapies

Several experimental approaches directly target MNS activity. Neurofeedback provides real-time brain-activity metrics, enabling self-regulation of prefrontal and amygdalar circuits implicated in both MNS and emotional control. Connectivity-based EEG–fMRI neurofeedback has shown preliminary benefits for social-cognitive enhancement in SAD and BPD (39).

Virtual reality exposure therapy (VRET) delivers immersive, structured social scenarios, offering graded exposure for SAD. When integrated with CBT, VRET improves engagement and provides repeated opportunities for practicing social interaction, potentially refining MNS-related processes such as emotion recognition and action mirroring (40–42).

Transcranial magnetic stimulation (TMS) applies magnetic pulses to the motor cortex, measuring motor-evoked potentials in response to observed actions as an index of mirror neuron responsiveness. Initially developed for motor rehabilitation, TMS is now under investigation for enhancing both social and motor outcomes in psychiatric populations (43,44).

Overall, these strategies signal a shift toward linking neural-level processes with observable social behavior, offering avenues for addressing core social-cognitive deficits across psychiatric disorders.

Conclusion

The MNS plays an important role in social cognition, but it is more pronounced in promoting empathy, imitation, and the understanding of others' intention (2). Research suggests that during social-emotional tasks, individuals with SAD show hypoactivation in core MNS areas, such as the inferior parietal lobule and inferior frontal gyrus (25,45). This dysfunction may be responsible for general SAD symptoms like social withdrawal, fear of evaluation, and deficits in social learning (46,47). Some agreement supports MNS involvement in SAD, but not enough evidence exists to draw conclusions concerning BPD. While some reports suggest overlapping MNS disruptions in BPD, methodological inconsistencies and the scarcity of direct comparative studies hinder any clear interpretation (19,48). However, the present literature is restricted by several limitations, including most studies being cross-sectional, a lack of longitudinal studies, limited inclusion of culturally diverse populations, and variability in neuroimaging and behavioral assessment techniques (2,26,49). Nonetheless, therapeutic innovation is progressing. For instance, neurofeedback, action observation therapy, and transcranial magnetic stimulation have shown initial evidence for improving social functioning via MNS modulation (26,48). These techniques, in combination, indicate future directions for psychotherapy, particularly CBT, especially concerning the integration of MNS-based training and the application of biomarker-guided therapy tailored to individualized treatment (15,50). Importantly, given that SAD frequently develops in childhood and adolescence, early detection of MNS anomalies might also have implications for prevention during critical maturational stages (51,52).

In conclusion, the MNS holds promising potential as a neural substrate for understanding and treating SAD. The development of this field will depend on refining methodologies, broader and more diverse sampling strategies, and longitudinal assessment of the developmental trajectory of MNS-related impairments and their clinical implications.

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Tables/Figures

Table 1.1 - Overview of Key Studies on Mirror Neuron System Dysfunction in BPD and SAD

Topic	Details
BPD Findings (IFG Activity)	Mixed results overall. Noted drop in activity in the inferior frontal gyrus (IFG), which manages complex social skills like understanding context and mental states. BPD patients may feel emotions intensely but struggle to interpret them clearly (25).
Tactile Mirroring Study	Investigated tactile mirroring and responses to observed or felt touch in BPD. Found abnormal connectivity in primary somatosensory cortex (S1) and some unusual behaviors. Did not clearly confirm disruption of tactile mirror system (TaMS) (26).
TMS-EEG Study	Used combined TMS-EEG to directly assess TaMS circuits in BPD. Study is ongoing; early results suggest TMS-evoked potentials (TEPs) may offer a direct measure of MNS activity in clinical groups (34).
Challenges in MNS Research	EEG offers fast and easy measurement but has poor spatial resolution, making it difficult to identify specific brain areas (5). Mu rhythm suppression overlaps with alpha waves, which relate to visual attention, not just MNS (3).
Methodological Issues	Small sample sizes and inconsistent task designs limit firm conclusions. Newer methods like TMS-EEG may provide clearer insights into MNS function in disorders like BPD and SAD.

Table 1.2 - Shared Mirror Neuron System (MNS) Dysfunction in Social Anxiety Disorder (SAD) and Borderline Personality Disorder (BPD)

Feature	Social Anxiety Disorder (SAD)	Borderline Personality Disorder (BPD)	Citation
Shared Neural Disruption	MNS abnormalities; altered activity in salience and cognitive control networks (anterior insula, dorsal ACC).	Same MNS abnormalities and network disruptions (anterior insula, dorsal ACC).	(3)
Behavioral Manifestations	Interpersonal sensitivity, emotional dysregulation, impaired perspective-taking.	Same behavioral traits: sensitivity, dysregulation, poor perspective-taking.	(3)
Transdiagnostic View	Social cognition deficits stem from disruptions in shared sensorimotor and cortico-limbic networks.	Same network-level disruptions underlie social cognitive impairments.	(6)

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general knowledge of this area.

Conflict of Interest

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