Commentary

The Evolving Landscape of Social Neuroscience and Its Implications for Psychiatry

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Historically, the individual has logically been the focus of psychiatry, and by extension, the focus of basic neuroscience aimed at understanding disease etiologies and therapeutic interventions. However, the social nature of psychiatric co-morbidity has garnered increased attention in the past 20 years, as our therapeutic magnifying glass has expanded to examine individuals in the context of their social environments—in dyads, family units, communities, and even societies. Cross-diagnostically, patients with serious mental illness (SMI) often exhibit social dysfunction so severe that indispensible supports are torn away, leading to a decreased quality of life and life-threatening psychological and physical sequelae. Unfortunately, the vicious cycle that connects worsening psychiatric morbidity and social dysfunction becomes nearly impossible for individuals to break and poses a significant challenge for mental health professionals.

In response, we have begun uncovering the “social brain”—the neurobiological constructs underlying social behavior and cognition—and identifying governing brain regions such as the medial prefrontal cortex, anterior cingulate cortex, amygdala, anterior insula, and temporoparietal junction. This network allows individuals to perceive, interpret, and respond to social information and underlies empathy, theory of mind, and social decision making (1), while network disruptions have been linked to several forms of SMI, including autism spectrum disorder, schizophrenia, and social anxiety (2). Molecularly, the social brain uses neuropeptides such as oxytocin, which modulates physical contact, bonding, and reproductive behavior (3). However, preclinical findings have struggled to translate this knowledge into efficacious psychiatric care, as treatments have instead used pharmacotherapies aimed at managing symptoms of psychosocial disease states rather than targeting core pathophysiological mechanisms. Here, we outline a conceptual basis for how social neuroscience and psychiatry can interact to better understand complex behavior and pathophysiology with the goal of developing novel therapeutics that may modulate the social brain, starting with a historical contextualization of our broader field.

Social neuroscience is not restricted to humans, with the origins of our field bastioned by the work of ethologists and primatologists in the early to middle 20th century (Figure 1, left). Harry Harlow’s work on social attachment (4) challenged the prevailing notions of behaviorism, which emphasized how external stimuli and reinforcement shape behavior, neglecting the significance of social relationships and emotional bonds. These studies illustrated the interplay between social needs and environmental influences on behavior, emphasizing the importance of relationships in promoting psychological and emotional development. In parallel, the field of neuro-ethology—emerging from the observational work of Tinbergen and Lorenz—highlighted the adaptive significance of social hierarchy, mate selection, empathy, and cooperation. This framework provided compelling accounts for the neurobiological bases of primate cognition and provided insightful ways to compare neural substrates of social behaviors across different species.

The field then transitioned from conducting neuro-ethological studies to using easily replicable, though more restrictive, paradigms. This reductionism occurred alongside the emergence of behavioral pharmacology and genetics, which permitted causal claims about brain function via intracranial manipulations. We thus dissected complex social behavior into component parts, generally by presenting animals with a decision regarding a constrained social stimulus, thereby assessing “sociability” or “social discrimination.” These investigations enabled broad replication with multiple cohorts and laid the foundation for mapping the mammalian social brain (5). However, given the dynamic nature of interactions between multiple agents, the validity of these tasks is often debated. This led to the development of more ecologically valid assays that incorporated naturalistic social contexts and the consideration of individual differences.

In the past decade, we have progressed beyond reductionist measures of social interactions in constrained experimental designs to studying freely behaving and interacting animals while simultaneously assessing or biasing the social brain in real time (6). Computer vision techniques have been developed to capture the dynamics of previously overlooked social interactions with a fine lens, enabling the development of sophisticated behavioral paradigms and the identification of nuanced behavioral patterns and motifs. Likewise, bioengineering innovations have allowed us to manipulate and monitor neural activity in animal models, enabling unprecedented spatial and temporal resolution. Specifically, optogenetic and cell type–specific approaches permit the selective activation or inhibition of neural circuits involved in social behavior with high resolution. Miniaturized telemetric neuronal recording devices have enabled us to monitor neural populations once thought inaccessible, allowing for a much-needed return to naturalism in social neuroscience (7). Thus, behavioral paradigms historically deemed too complex have been reintroduced to understand the high dimensional nature of social behavior. However, there must be caution in extrapolating these findings from animals to humans, as the nature of social dysfunction poses challenges in developing behavioral models in nonhuman animals that capture the heterogeneity...
and complexity of human experiences. Although the field has seen an evolution in recent years, there remains a tenuous gap between preclinical models and clinical applications within psychiatric practice.

Psychiatry has evolved alongside social neuroscience over the course of the past half-century, though not at the same pace (Figure 1, right). With the advent of psychotropic medications, mental health providers transitioned from a psychoanalytic approach toward a biological diagnostic schema that emphasized neurochemical imbalances (i.e., the monoamine hypothesis) to explain and treat SMI. As such, our tools have largely been distilled to agents that target neurotransmitters and their receptors. Notably, this biological reductionism has neglected the important social and psychological dimensions of mental health care. Many have argued for a shift back toward a multidimensional understanding of mental illness that reintegrates the social, psychological, and biological factors in the assessment and treatment of SMI. Even still, the biological aspects dominate formulations, with social factors being considered with only moderate opportunities for targeted intervention.

Given the preponderance of social comorbidity in most, if not all, psychiatric conditions, it has become clear that the social world is not a backdrop for those with serious mental illness. Given the preponderance of social comorbidities in most, if not all, psychiatric conditions, it has become clear that the social world is not a backdrop in the individual experience of SMI, but rather a part of its genesis, maintenance, and treatment. For example, social amotivation is now recognized as a predictor of deterioration in schizophrenia and autism spectrum disorder. In parallel, group therapeutic approaches, ranging from dialectical behavioral therapy to multisystem family therapy, have gained popularity, with the goal of improving social functioning and interpersonal relationships for individuals with SMI. However, our understanding of how these interventions work at a molecular or neural circuit level remains elusive. Furthermore, the excitement surrounding psychedelics and entactogens such as MDMA for psychiatric treatment highlights the potential of targeting social phenomena. Particularly, these compounds have been found to enhance affiliation and prosocial states (8), and MDMA has shown significant clinical promise as a treatment for posttraumatic stress disorder (9), with evidence that its therapeutic effect is facilitated by the social context of therapy—though its evidence is scant and the psychobiological mechanisms remain unresolved.

Figure 1. The convergence between social neuroscience and psychiatry. (Left) Social neuroscience has evolved over the course of the last century, from primate neuroethology to pioneering behavioral work by Harry Harlow and colleagues. Further biological reductionism resulted in complex behavior being dissected into component parts, laying the foundation for the mapping of the “social brain.” Recent nonhuman animal investigations have used viral vector strategies to alter site-specific gene expression and manipulate neural activity with high temporal resolution, combined with the use of behavioral tracking software to capture unprecedented nuance of social interactions. (Right) Psychiatry has also evolved, similarly experiencing a reductionism from a psychoanalytic approach to the use of psychotropic medications targeting neurochemical systems such as serotonin and dopamine. Interventional approaches such as transcranial magnetic stimulation and MDMA-assisted psychotherapy are gaining traction as new modalities of psychiatric treatment. As such, there has been an increased emphasis on the social world as not merely a backdrop for those with serious mental illness. (Bottom) The future of social neuroscience is bright, with technological advances permitting circuit-level analyses in both rodent and nonhuman primate models with unprecedented temporal and spatial resolution. These studies will be important to validate using existing clinical research techniques in human populations to ensure efficacious translational interventions. Similarly, we endorse a “bedside to bench” approach, whereby clinical observations lead to hypotheses that can be tested in controlled laboratory settings. As we have a richer understanding of the social implications of serious mental illnesses, with new agents that appear to target social-specific symptoms, we are perfectly poised for a re-expansion of social neuroscience and psychiatry, converging to treat vicious cycles of social dysfunction and psychiatric morbidity.
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unclear. Thus, before all resources begin to pour back into pharmaceuticals, we must first answer the fundamental question: Will these therapeutics exert their effects in isolation from social stimuli, or are social interactions indispensable for their efficacy?

We propose a multidimensional solution to answer these questions, combining basic science and current psychiatric practice. While social neuroscience has thus far largely failed to influence psychiatric practice, perhaps due to limited communication between the two fields, the integration of social neuroscience with psychiatry heralds a new era of understanding and treating psychiatric illness (Figure 1, bottom). Preclinically, there is a growing emphasis on translating our mechanistic understanding of the social brain into novel therapeutics—combining pharmaceutics, neuroimodulation, and psychosocial interventions. This includes the use of advanced neuroimaging techniques, computational modeling, and naturalistic behavioral paradigms to elucidate the interplay between social behavioral and neural processes. Clinically, the emphasis on the social world has led to a renewed focus on social interventions in the diagnosis and treatment of SMI, alongside an increased focus on clinical trials and preclinical studies using various entactogens coupled with psychotherapy in the form of “integration sessions” targeting social-specific modalities. Likewise, psychiatry is now embracing computational tools developed in the lab aimed at individualized diagnoses and care via digital phenotyping (10). This underused “bedside to bench” approach will lead to novel therapeutic targets and personalized interventions if psychiatrists can better understand how and for whom social behavioral therapy is most effective.

To accomplish these goals, we must embrace the diversity of social experiences, reviving the culture of comparative, cross-species approaches used in neuroethology, instead of shying away from its challenges. Rather than relying solely on controlled laboratory settings or structured clinical environments, we must study social behavior in naturalistic, real-world contexts, just as our predecessors once did. By unraveling the interplay between the social brain and psychiatric disease, social neuroscience is poised to revolutionize psychiatric care. Through the recapitulation of key features of human social dysfunction in ecologically valid settings and contexts, animal models will be valuable in testing novel interventions and identifying potential biomarkers. As we capitalize on this diversity and harness the power of social neuroscience, psychiatry will inch closer to a future where mental illness is ultimately met with empathy, understanding, and individualized treatment strategies.

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Article Information

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