Letter to the Editor

Developing Research Domain Criteria (RDoC) to improve diagnosis and treatment of social deficits in psychiatric disorders: The Mirror Neuron System as a model

Dear Editors,

The recent release of the new Diagnostic and Statistical Manual — 5 (DSM-5) (American Psychiatric Association et al., 2013) has sparked a rich debate about the validity of psychiatric diagnoses. In his Director’s blog, the head of the National Institutes of Mental Health, Dr. Thomas Insel, described the new DSM as a “dictionary” with good definitions and labels for disorders that make it reliable, but questioned its neurobiological validity (Director’s Blog: Transforming Diagnosis). Despite major advances in neuroscience in the last two decades, psychiatric diagnoses continue to be based on phenomenological descriptions, rather than being firmly planted in neurobiological mechanisms. To remedy this gap, he and others have suggested using the Research Domain Criteria (RDoCs) to develop a new system of diagnosis and treatment based on brain circuitry. The model assumes that mental disorders may be understood as neural circuit impairments, which lead to impairments at the level of cognition, emotions and behavior, and function. Five major RDoC domains have been identified — 1) Negative Valence Systems, 2) Positive Valence Systems, 3) Cognitive Systems, 4) Systems for Social Processes and 5) Arousal/Modulatory Systems, that cross current DSM diagnostic lines (Director’s Blog: Transforming Diagnosis).

For the domain of Systems for Social Processes, the Mirror Neuron System (MNS) provides one potential starting point. The concept of “mirror neurons” was first introduced by Pellegrino and colleagues to reconcile the finding that a subset of premotor neurons fires when a pri- mate either performs or observes an action (di Pellegrino et al., 1992). Their initial study was followed by a series of findings in primates, and subsequently in humans (Fabbri-Destro and Rizzolatti, 2008), that support the existence of a neural network in humans comprised of neurons with mirroring properties, i.e., the MNS. It was also thought that in humans, the MNS not only serves motor matching functions, but rather with its interconnections with limbic and cognitive areas provides the neural basis of social cognition (Muthukumaraswamy and Singh, 2008). Along these lines, key components of social cognition, including empathy (Decety and Jackson, 2004), theory of mind (Gallese and Goldman, 1998), affect recognition and attributional bias are associated with MNS function in healthy individuals (Sinigaglia and Sparaci, 2010). Using conventional neurobiological investigative tools, some studies in psychiatric disorders with prominent social deficits such as autism spectrum disorders and schizophrenia spectrum disorders have shown deficits in MNS functioning, or social cognition, an emergent property of the network (Fig. 1) (McCormick et al., 2012). Singh and colleagues have used scalp electrical recordings of mu oscillations over sensorimotor cortex to assess information processing within the MNS and found evidence of abnormal signals (mu suppression) that are associated with reduced social functioning in first episode psychosis patients (Singh et al., 2011), providing construct validity for this model.

Nonetheless, the results have not been consistent across studies, owing to the limitations of the particular tool employed (fMRI: low temporal resolution, EEG: lack of anatomic specificity) or the particular paradigm (variable emotion processing tasks, theory of mind tasks) used to study the MNS. Therefore, although evidence is converging to suggest that the MNS plays a critical role in social adaptation in healthy individuals, the details of how MNS dysfunction relates to psychopathology remain to be worked out. For instance, the relationship between information processing in the network and social cognition, behavioral and functional deficits and treatment in clinical populations remains largely unknown.

In this context, the authors are investigating the link between “mu suppression”, a marker of MNS function, and treatments thought to promote social adaptation in patients with schizophrenia. For instance, small-scale studies in normal adults have shown a link between the prosocial neurohormone oxytocin and mu oscillations (Perry et al., 2010). It remains to be seen whether mu rhythm abnormalities show improvement in patients with prosocial treatments, and if early neural response can predict later behavioral response. In addition, it is not known whether similar disturbances exist in other disorders with psychotic features. A similar approach can be used to interrogate other neurobiological circuits underlying negative and positive symptoms, cognitive systems and arousal/modulatory systems implicated in psychiatric disorders. As early methodological challenges are resolved, we hope that the MNS will provide a model system to implement RDoC recommendations by integrating 1) research findings from seemingly disparate fields, 2) using readily available low-cost tools, and 3) the MNS. Therefore, although evidence is converging to suggest that the MNS plays a critical role in social adaptation in healthy individuals, the details of how MNS dysfunction relates to psychopathology remain to be worked out. For instance, the relationship between information processing in the network and social cognition, behavioral and functional deficits and treatment in clinical populations remains largely unknown.

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combining them with pre-existing treatments. This is an exciting era in psychiatric research where developments in basic and cognitive neuroscience can finally be used to tackle some of the most significant clinical questions relevant to psychiatric disorders.

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Disclosures

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References


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