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The emerging impact of social neuroscience on neuropsychiatry and clinical neuroscience

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Social neuroscience has made great strides toward clarifying the neural basis of brain–behavior relationships. In the last 25 years, social neuroscience has made contributions to many fields, including cognitive neuroscience, social psychology, ethology, economics, and even philosophy. The field has spawned a highly productive collaboration between investigators in these areas, many of whom have profited from the great leap forward in functional neuroimaging.

It is now time for social neuroscience to make similar contributions to neuropsychiatry. Social behavior is integral to all brain–behavior disorders. Neuropsychiatry, and an understanding of the topics in this special issue, depend upon the basic neural correlates of social behavior. It begins with social perception, or the ability to detect the presence of another mental agent, including the critical roles of face recognition and the fusiform face area (Kanwisher & Yovel, 2006), and the superior temporal gyrus or sulcus (STG/STS) for observed biological motion. Mechanisms for social simulation of not only others' movements but also their intentions and emotions have emerged in conjunction with discoveries in a mirror-neuron system. Areas of socioemotional significance include the anterior insula and anterior cingulate cortex, the anterior temporal cortex, and, especially pertinent to the articles in this issue, the amygdala, which plays a role in emotional salience and significance. Our brains are primed for thinking about the minds of others. Making inferences about others' mental states, or theory of mind (ToM), produces increased activity in the medial prefrontal cortex (MPFC), temporoparietal junction (TPJ), and medial parietal cortex (Frith & Frith, 2006).

In this special edition, we view neuropsychiatry in the context of these advances in social neuroscience. Nearly every neuropsychiatric illness involves social behavioral disturbances (Adolphs, 2010), and much of the promise and justification for the investment in this burgeoning field is its possible clinical implications. To what extent do neuropsychiatric illnesses result from neural impairments in social perception, social simulation and the appreciation of social salience, mental state attribution, or disturbances in social regulation? Psychiatric illness may be a continuum with variability in the healthy population, as reflected in one article in this issue. Finally, understanding the social neurocognitive source of symptoms such as suspiciousness and apathy can be a step toward the development of therapies for these disorders.

CONTENTS OF THE SPECIAL ISSUE

The papers in this series study or review various clinical entities by varied methodology. Clinical

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entities include autism spectrum disorder (ASD) and its variant, pervasive developmental disorder (PPD); schizophrenia and schizotypal disorder; depression; attention-deficit hyperactivity disorder (ADHD); traumatic brain disease (TBI); psychopathy; frontotemporal dementia; and Parkinson's disease. The methodology includes behavioral assessments, eye-tracking, event-related potentials (ERP), and brain imaging, particularly functional magnetic resonance imaging (fMRI). Topics covered include social attention and perception, amygdalar reactivity, social attention, social and emotional recognition and competence, social approach and withdrawal, suspiciousness, immoral or corrupt behavior, and ToM.

SPECIFIC STUDIES

Three articles in this series start with an exploration of face perception in ASD, particularly as it relates to the amygdala. The amygdala is involved in guiding fixations on the eyes, a region of the face that is socially salient, and it plays a role in emotion and emotional inference from faces. Birmingham, Cerf, and Adolphs compare social attention in ASD to that in S.M., a patient with rare, bilateral, amygdalar lesions. As revealed by eye-tracking to complex social scenes that contain faces, S.M., but not ASD subjects, increased her gaze to the eyes when the task required social attention. The authors conclude that face-perception difficulty in ASD is not due to amygdalar dysfunction in directing gaze to the eyes but instead to insensitivity to socially relevant information. McPartland et al. compare ASD and non-ASD subjects on N170, an ERP marker of early face processing, and on behavior measures of face recognition. Individuals with ASD show decreased N170s, slowed face processing, and decreased sensitivity to face inversion. The authors postulate that decreased face perception in ASD is the consequence of a developmentally reduced social drive. Uono, Sato, and Toichi present a study of emotion-expression recognition in mild PPD, a variant of ASD, which reveals decreased performance on the Ekman face emotions, especially fearful ones. Fearful-expressions recognition does not improve with age, as in normally developing children, and is worse with the severity of PPD, suggesting an atypical development of facial expression recognition. In PPD, impaired recognition of fearful faces may result from abnormal amygdalar development for these socially important stimuli.

Three other studies investigate impairments in face-emotion recognition in various conditions. Among adults with ADHD, Ibáñez et al., in a similar study to McPartland et al., present faces and words to test the effects of stimulus type, valence, and face–word compatibility. The adult ADHD group shows deficits in N170 emotion discrimination, especially for positive face stimuli, in the absence of deficits in facial structural processing. In ADHD, the reduced N170 amplitude for positive stimuli suggests a specific right hemisphere impairment for the early processing of emotional faces. Among patients with depression, Derntl et al., using fMRI, investigate the neural correlates of social approach and withdrawal with facial emotional expressions. In addition to higher avoidance scores and stronger, wider activation during avoidance in depression, they report decreased amygdalar function during approach and during processing of happy faces in particular. This points to secondary effects of depression on amygdalar activity. Kumfor et al. report a study of face-emotion recognition in the three major variants of frontotemporal dementia. All patient groups have impaired overall facial emotion recognition, but particularly for negative emotions. They increase the intensity and perceptual salience of face emotions by digitally manipulating the images and show that all FTD subgroups, except semantic dementia (SD), improve with the increased emotional saliency. The authors conclude that, in SD, direct damage to the amygdala results in widespread emotion-recognition impairment, which cannot be overcome by increasing emotional salience.

A theme in several articles in this issue is mechanisms underlying schizophrenia, especially early emotion-processing deficits, inability to pick up and integrate body emotions, and susceptibility to suspicious thinking. Garrido-Vásquez, Jessen, and Kotz's review of the literature confirms the presence of emotion-perception deficits in psychiatric populations when patients are tested in dynamic and multimodal naturalistic settings and under different task (explicit and implicit task instructions) demands. The authors propose that patients with schizophrenia are impaired in a fast, pre-attentive system involving the amygdala and its surrounding network. Van den Stock et al. evaluate the perception of bodily expressions of emotions and their integration with voice in patients with schizophrenia. On static pictures of emotions, patients with schizophrenia are impaired in emotional recognition, and on videos of dynamic emotions they are significantly influenced by the auditory information. They conclude that patients with schizophrenia are impaired in recognizing whole-body expressions of emotions and have difficulty with multisensory integration of emotional information. Sasamoto et al. use the autism-spectrum quotient (ASQ) to explore social perception and the "autistic tendency" in comparison to gray

matter alterations on structural MRI among patients with schizophrenia. There are significant negative correlations between the total ASQ score and gray matter volume in the area of the left STS. This region reciprocally interacts with regions, such as the amygdala, involved in attaching emotional salience to sensory input.

Another study investigates the predisposition to psychotic traits in a nonclinical population. Among Chinese students, Li et al. compared those with and without schizotypal personality traits on ERP changes elicited by suspicious thoughts. They use a clever paradigm, a novel digit-guessing task, to induce the subject's "feeling of been seen through." A "friend" is said to guess the patient's choice of digits, and they calculate difference waves (correct guess wave minus incorrect guess wave) on P3 and P3 amplitude. The amplitude is smaller for the schizotypal group, suggesting that it might be inhibited in those with higher level of paranoid ideation; the schizotypal subjects are not surprised by correct guesses of friends because schizotypicals might have more suspicious or paranoid thoughts.

A number of studies explore mental state attribution in neuropsychiatric disease. Two fMRI studies indicate that, in schizophrenia and in TBI, there is disturbed ToM related to the MPFC, a central region for ToM, with additional involvement of the TPJ in schizophrenia and of surrounding white matter in TBI. Among schizophrenics, Lee et al. examine ToM, using a specific task, a false-belief attribution condition versus false photograph for general reasoning and simple reading ability. Schizophrenics show reduced task-related activations in the TPJ and the MPFC during the false-belief condition, but not during the false-photograph condition. Among adolescents with moderate-to-severe TBI, Scheibel et al. examine ToM using an animated social attribution task. Compared to normal subjects, the TBI subjects have more diffuse and intense activation with sparing of the MPFC. Further diffusion tensor imaging for functional anisotropy as a measure of white-matter integrity shows reductions in surrounding frontal areas, indicating that white-matter changes may cause reductions in connectivity among the components of a brain network that mediates social cognition.

Although chronic social and emotional deficits are common in moderate-to-severe TBI, current techniques of assessment with ToM tasks may be insufficient. Hynes, Stone, and Kelso review existing social/emotional measures and introduce four new tasks. The Global Interpersonal Skills Test is a questionnaire that allows patients and their caregivers to identify specific social traits and behaviors that may be a source of difficulty. The Assessment of Social Context Task is a dyadic video task that examines patients' comprehension of social contextual information, including emotions, intentions, and attitudes. The Awareness of Interoception Task measures patients' sensitivity to their own heartbeat, which may underlie awareness of their emotions. Their Social Interpretations Task, a social framing task based on social geometric animation, appears less useful than the other three tasks.

Of significance to neuropsychiatry is whether there are cultural effects on mental state attribution and other social cognitive processes. As there are cultural differences in mode and development of ToM, one might expect differential brain activation patterns. Koelkebeck et al. present a paper on transcultural differences in activation of the MPFC in Japanese versus Caucasians on a ToM task of moving geometric shapes in a social pattern. The Caucasians have greater activation, possibly from a greater need to constantly distinguish between selves, others, and surroundings. The Japanese have less activation, possibly because of greater efficiency from early-life learning to be in tune with unspoken, nonexplicit social signals (ToM may take longer to develop in Japanese). Interestingly, MPFC activation in the Japanese "catches up" when there are more autistic features, probably due to compensation for reduced mentalizing abilities.

Two papers in this series address the important problem of psychopathy. Rather than more traditional regions of interest, Sato et al. analyze the MRI images of subjects with antisocial personality disorder with novel pattern classification techniques of support vector machines and maximum uncertainty linear discrimination analysis. Among the subjects with high psychopathy scores, there is decreased gray matter concentration in the STG bilaterally, especially on the right. Sobhani and Bechara review the somatic marker hypothesis as a mechanism potentially disturbed in those who manifest immoral and corrupt behavior. Their studies with the Iowa Gambling Task show that damage to ventral MPFC leads to impaired judgment and decision-making as well as failure to learn from repeated mistakes. Their review of the literature describes studies of patients with ventral MPFC lesions who show impairments in victim-based moral judgments, or acceptance of increased attempted harm to others (e.g., Young & Saxe, 2009).

This special issue extends to defining the neural circuitry for neuropsychiatric symptoms. Lawrence, Goerendt, and Brooks investigate the neuroanatomy of apathy, using a reward paradigm and positron emission tomography in patients with Parkinson's disease.

By manipulating search outcome (money reward versus valueless token) while keeping the actions of the participants constant, they examine the influence of apathy on the neural coding of money reward cues. In this study, apathy is associated with a blunted response to money in the distributed neural circuit integral to the representation of stimulus reward. Apathy may be associated with diminished high-level value representations in ventral MPFC, emotional blunting in the amygdala, and decreased reward value in the striatum and in the dopamine-dependent midbrain region of the ventral tegmental area.

THEORETICAL ISSUES HIGHLIGHTED BY THE CONTRIBUTIONS

Several of the papers in this issue demonstrate impairments at the early-input stage of social cognition—i.e., at the level of facial perception. The papers in this series advance our understanding of disturbances in this area including ASD, ADHD, and dementia. Many of these studies focus on the changes in the amygdala, a structure known to play a role in attention to the eyes and in facial emotion appreciation. Several papers on ASD indicate that insensitivity to aspects of face processing is consequent to a more fundamental insensitivity to social phenomena, and McPartland et al. further suggest that the face-perception system does not develop normally for social stimuli in ASD. In other words, this "social insensitivity" early in life leads to developmental impairments in the ability of face-perception areas and face-emotion areas to respond to social stimuli.

Face emotion is the most important information for social behavior, and studies show difficulty in face-emotion recognition in ASD/PPD, adult ADHD, depression, and semantic dementia. These studies point to more than one mechanism. For example, in ASD, recognition of fearful faces may follow the abnormal amygdalar development of responsiveness to social stimuli; in ADHD, there are right hemisphere deficits in processing of facial emotions; in depression, there is a top-down late effect on the amygdala; and in SD, there is direct injury to the amygdala with increasing intensity.

Social cognition is a key determinant of poor functioning in schizophrenia, and several studies in this series indicate difficulties with the social perception of emotion, not only from faces but also from the body, as well as the ability to integrate body, face, and other socioemotional clues. In addition, Li et al.'s study of suspicious thinking among schizotypal subjects supports the concept of a continuum from suspicion in normal subjects to delusions of reference and persecutory delusions in schizophrenia. This study is significant for the view that psychotic traits are part of a continuum from normality to psychosis.

The disturbances in ToM found in neuropsychiatric illnesses is associated with corresponding disturbances in the circuitry for mentalization (Frith & Frith, 2006). In schizophrenia, studies show reduced activation in TPJ, as well as in MPFC, and gray matter reductions in the cortical areas surrounding the left STS. Scheibel et al.'s findings in TBI indicate that these alterations in mentalizing circuits are not restricted to areas that typically mediate social cognition, as the surrounding white-matter tracts can affect this mentalizing circuit.

A final theme involves the important neuropsychiatric issue of psychopathy and the possible neural correlates of immoral or corrupt behavior. A study using novel fMRI analysis of subjects with antisocial personality disorder shows changes corresponding to psychopathy in the right STG, an area important for social perception. Sobhani and Bechara further make the case for linking disturbed somatic markers, or re-enacted social and emotional representations that affect decision-making, to the commission of immoral or corrupt acts. Usually, the ventral MPFC reactivates somatic effectors in the insula and somatosensory and related structures, and this fails to occur in psychopaths.

FUTURE DIRECTIONS

Many challenges remain for social neuroscience including its more thorough application to neuropsychiatric illnesses (Adolphs, 2010; Cacioppo et al., 2007). The studies in this special edition of *Social Neuroscience* advance our understanding of the neural underpinnings of neuropsychiatric diseases, but there remains much work to be done. As Hynes, Stone, and Kelso point out for TBI, investigators need additional social/emotional measures for clinical as well as research assessment. As Koelkebeck et al. point out, investigators need to consider the effects of culture on their results. As Sato et al. illustrate, novel fMRI computational approaches have potential applications to neuropsychiatry. Social perception, social salience and simulation of mind, social cognition and social representations in the brain, and the extent of social regulation need further clarification of their neuropsychiatric implications. The specificity of a social system in the brain and the role of specific structures (Mitchell, 2009), the primacy of social behavior and its default state, and how much social mechanisms share with nonsocial cognitive processes are all issues pertinent to understanding neuropsychiatric diseases. In sum, social neuroscience holds great promise for clarifying many aspects of brain—behavior disorders.

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