Mirror neuron system based therapy for emotional disorders

Ti-Fei Yuan a,*, Robert Hoff b,*

a Department of Anatomy, Li Kai Shing Faculty of Medicine, The University of Hong Kong, 21 Sassoon Road, Hong Kong
b Department of Psychology, Mercyhurst College, 501 East 38th Street, Erie, PA 16546, USA

Received 30 June 2008; accepted 2 July 2008

Summary Mirror neuron system (MNS) represents one of the most important discoveries in the area of neuropsychology of past decades. More than 500 papers have been published in this area (PubMed), and the major functions of MNS include action understanding, imitation, empathy, all of which are critical for an individual to be social. Recent studies suggested that MNS can modulate emotion states possibly through the empathy mechanism. Here we propose that MNS-based therapies provide a non-invasive approach in treatments to emotional disorders that were observed in autism patients, post-stroke patients with depression as well as other mood dysregulation conditions.

The mirror neuron system (MNS) has been regarded as one of the most important discoveries in cognitive neuroscience in recent years. The unique property of mirror neurons is that they discharge both during action execution and during observation of specific actions [1]. This property allows humans to understand the intentions of others through observation of their actions, and thereby plays a role in imitation-based learning and empathy. Importantly, it has been found that the adult MNS is still plastic [2,3] and that artificially induced MNS activation could provide a basis for brain rehabilitation in patients with post-stroke motor defects [4]. In this manuscript, we discuss the possibility and some pioneer practices of MNS activation as a treatment modality for emotional disorders.

MNS dysfunction as a risk factor for autism and deficits in understanding emotion

It has been suggested that developmental MNS dysfunction leads to disordered social cognition in humans, including autism spectrum disorders (ASD)
These disorders in theory are characterized by limitations of mind, imitation, and social communication, summarized by the phrase ‘seeing is not understanding’ [8]. A number of studies, including EEG [9–11] and brain imaging studies [12,13] have suggested the presence of MNS dysfunction in ASD patients. For example, brain imaging studies showed significant thinning of areas that belong to the MNS, such as inferior frontal cortex (IFC), the inferior parietal lobule (IPL), superior temporal sulcus (STS) as well as other areas that constitute the distributed neural circuitry for social cognition [13], in individuals with ASD. Additionally, it appears that the MNS undergoes abnormal activation during visual information processing [12,14]. Collectively these findings suggest that MNS dysfunction might be an important neurobiological risk factor for autism spectrum disorders.

Given the important contribution of face processing to emotion, it is likely that dysfunction of the MNS plays a role in the defective capacity of ASD patients to understand emotion [6,15–18]. With fMRI, it has been shown that there are reliable differences between normally developing children and children with ASD in the network comprising the frontal component of the MNS (namely, pars opercularis), the insula, and the amygdala, all of which enable emotion understanding via action representation [19,20]. Collectively, these data suggest that altered neural circuit activation in ASD patients includes emotion-processing brain areas. ASD patients was found to be correlated with mood disorders, both in children and adults [21–25], and the two diseases may share common genetic vulnerability, such as serotonin transporter genes that are one of the well-recognized family participating in anxiety and depression [26,27]. Consistently, patients with mood disorders show higher tendency to develop autism symptoms [28,29]. Thus the autism, possibly due to the mirror neuron dysfunction, can result in complex psychiatric conditions that include mood disorders.

Can autism treatments elevate mood states simultaneously? Antidepressants have been used in combined therapies to some autism patients [23,30,31], with proved efficiency. It is possible that the antidepressants can rescue the normal neurotransmission in brain areas that correlated to mirror neuron system, as well as their global neurotrophic effects. Psychologically, the important contributions of mirror neuron defects in autism patients to mood disorder symptoms permit improved integration ability, and restore the social network function [5,32,33]. The noninvasive manipulation of MNS activity may provide a novel approach to improving emotion modulation in ASD patients, and could possibly lead to overall clinical improvement in such patients.

Post-trauma emotional disorder: interpretation through the MNS

Focal brain trauma, such as stroke, can result in post-trauma emotional disorders [34–36]. These emotional disorders can be separated into several groups: dysfunction in emotion comprehension/understanding; dysfunction of those neurocognitive systems responsible for the experience of emotion; and dysfunction of emotion control and expression, which may be attributed to disrupted integration [37]. Given the putative role of MNS in emotion understanding, the impairment to MNS following stroke can lead to the first group of emotional disorders – the instrumental function of the MNS in the comprehension of emotion.

Brain imaging studies on imitation of facial emotional expressions suggest a critical role for the motor system in emotional behavior [37]. Those mood disorder conditions represent more specific complications of stroke than simply psychological responses to the motor disability [38]. Mirror neuron thus may mediate this lost link. Some studies have shown co-activation of mirror-system and limbic circuits through insula [19], and the focal stroke that interrupts the mirror neuron system may cause loss of modulatory amygdala affect and, finally, catastrophic reactions [35,39]. Additional complications include athymhormia, reflecting flat affect and a loss of motivation in the execution of motor actions [35]. Moreover, some other brain areas of motor significance, if damaged, can lead to emotional disorders [40–42].

Currently, the neuropsychiatric diseases resulted from motor cortex ischemia, such as post-stroke depression [43,44], could be treated with antidepressants and other general mood disorder therapies [45–47]. Here, the important role of MNS in post-brain trauma emotional disorders suggests a potential new avenue for treating stroke patients.

The MNS-based therapy: multi-functional and multi-modal

Mirror neurons fire to both observed and performed actions, hence there exists a possibility for observed-learning based brain activation or even rehabilitation [48–50]. Mounting evidence indicates that many forms of experience, including
physical exercise, sensory perception, and even direct brain stimulation or drug administration, may alter existing synaptic connectivity or induce synaptogenesis. The physiological bases of experience-dependent plasticity have been only partly elucidated, but may include upregulated endogenous neurogenesis [51–53], changed neurochemical transmission [54], induced expression of neurotrophins and neuropeptides [55] or improved body immune responses. Though we currently lack a clear understanding of the underlying mechanism for brain activity-related plasticity, activation of MNS has already been successfully used for post-stroke treatment and rehabilitation [4,56].

Considering the neurobiological role of MNS in emotional disorders inherent to both autism and post-stroke conditions, we believe that targeted MNS activation could be used to prevent and/or improve the experience of emotional disorders in such patients. First, the action-specific responses of mirror neurons provide a precise approach to activate related brain areas. Using computerized displays or virtual reality environments [57,58], it may be possible to design different scenes/actions in order to specifically modulate various brain regions. Observation therapy may therefore be useful in the prevention of emotional disorders with motor deficiency. Second, some mirror neurons may respond to sounds that are specific for actions—these have been termed “audio-visual” mirror neurons [59–61]. This suggests that combined therapies including both visual and auditory stimulations would maximize clinical efficacy. Furthermore, virtual reality may create such an environment. Recent neuroimaging studies indicate that music, like language, involves an intimate coupling between the perception and production of hierarchically organized sequential information, which links meaning to emotion via the mirror neuron system [62]. We believe that music could be a potent component in MNS-based therapies, as recent findings in the domain of stroke rehabilitation have shown [63].

Behavioral and psychiatric disturbances are frequent sequela to traumatic brain injury and major limitations on patient quality of life [64]. Pharmaceutical approaches that improve depression and other negative mood states have been the usual, if limited, therapeutic modality. Alternatively, MNS-based therapy provides the promise of a novel and non-invasive approach to improving the physiological and psychological condition of brain-injured patients. It is hoped that in the future MNS-targeted treatments can breathe fresh hopes for emotional disorders in autism children as well as post-brain trauma patients.

References

Mirror neuron system based therapy for emotional disorders


