Psychopathy and the mirror neuron system: Preliminary findings from a non-psychiatric sample

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Abstract

Recent advances in social neuroscience suggest a link between empathy and the mirror neuron system (MNS). Impaired empathy is one of the core diagnostic features of psychopathic personality disorder. In the present study, we investigated whether psychopathic personality traits in a non-psychiatric sample were related to MNS function. Healthy participants viewed short videos known to activate the sensorimotor MNS for pain (a needle penetrating a human hand) while transcranial magnetic stimulation (TMS)-induced motor evoked potentials (MEP) were recorded as a measure of motor cortex excitability. Individual psychopathic personality traits were assessed using the Psychopathic Personality Inventory (PPI) and correlated with the MEP findings. Consistent with previous data, observation of the painful stimulus was associated with a significant reduction in the amplitude of the TMS-induced MEP. Interestingly, the level of corticospinal excitability modulation was positively correlated with individual scores on the coldheartedness subscale of the PPI, such that individuals with the greatest MEP reduction were the ones scoring highest on the coldheartedness measure. These data suggest the existence of a functional link between 'motor empathy' and psychopathy.
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1. Introduction

The discovery of mirror neurons – cells that discharge when an individual executes, sees or hears a specific action or its corresponding action-related sound – in the macaque monkey and the subsequent description of a mirror neuron system (MNS) in humans (see Rizzolatti and Craighero, 2004) has led to unprecedented interest in

the neural basis of social cognition. According to the *embodied simulation* view (Gallese, 2003), the neural circuits that are activated during the observation of actions, emotions and sensations share critical, common substrates with those that are involved in the execution and experiencing of the same actions, emotions and sensations. If so, the mirror matching mechanisms that explain action (Fadiga et al., 1995; Hari et al., 1998; Iacoboni et al., 1999) and emotional (Carr et al., 2003; Leslie et al., 2004) recognition may also underlie key aspects of social behavior, such as mentalizing (the cognitive mechanism that allows one to ascribe goals and intentions to others;

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Iacoboni et al., 2005) and empathy (Gazzola et al., 2006). The term 'empathy' has been defined in a wide variety of ways in the scientific literature. Whereas some have argued that empathy is a unitary phenomenon, others have suggested that it encompasses distinct processes. For example, Blair (2005) describes three dissociable forms of empathy, each subtended by partially distinct neural mechanisms: cognitive, motor and emotional empathy. Here, we define empathy as the capacity to understand other's actions, sensations and emotions. MNS involvement in empathy has received support from functional magnetic resonance imaging (fMRI) studies. For example, Gazzola et al. (2006) showed that execution of hand and mouth actions and passive listening to their associated sounds produces activations in similar areas of the temporo–parieto–premotor cortex. Critically, activations in these mirror areas were positively correlated with scores on an empathy scale.

If the link between simulation mechanisms and the empathic response holds true, pathological conditions in which empathy is impaired may be associated with a dysfunctional MNS. Psychopathy is a good model for the study of empathy because of its prominent role in the symptomatology of the disorder (Blair, 2005), where autonomic responses to sad expressions, vicarious conditioning, and recognition of emotions in others are impaired ('affective empathy'; see Blair, 2005). Despite the wealth of information detailing the clinical aspects of psychopathy, the neuronal mechanisms underlying this disorder as they relate to empathy remain unclear. Of particular interest considering the nature of the disorder is how this population perceives – and understands – pain when it occurs in someone else. Numerous studies support the idea that MNS mechanisms are involved in understanding pain in others. Singer et al. (2004) used fMRI to assess brain activity while healthy participants were receiving painful stimulation or received a signal indicating that a loved one was receiving a painful stimulus. They found that the affective components of the pain matrix (the anterior insula and the anterior cingulate cortex) were activated in both conditions. Importantly, empathy scores were correlated with the level of activation in the insula and cingulate cortex. It was subsequently shown that the 'sensorimotor' aspects of empathy for pain were also subserved in part by a resonance mechanism, whereby sensorimotor components of the pain matrix are activated both when an individual *feels* pain or *observes* pain in others. Using TMS, Avenanti et al. (2005) showed that passive observation of a needle penetrating the hand of a human model produced a reduction of corticospinal excitability in the motor cortex representation of the specific muscle that was pricked, an effect closely resembling that which occurs during the sensation of pain. Importantly, scores on *sensory* empathy scales – contrary to *emotional* or *affective* empathy scales – were correlated with the degree of corticospinal inhibition occurring during pain observation. In other words, subjects that rated pain as more intense in the observed model were the ones showing the greater stimulus-induced modulation of motor cortex excitability (Avenanti et al., 2005). A subsequent study by the same group showed that shared representations of pain were also present in primary somatosensory cortex (Bufalari et al., 2007). The pattern of somatosensory-evoked potentials (SEPs) during observation of a painful stimulus applied to a model hand was found to be highly similar to that occurring during a painful experience. Furthermore, SEP amplitude was correlated with intensity ratings of the observed pain event. Taken together, these study clearly highlight the sensorimotor aspects of empathy for pain and argue in favor of an understanding of empathy that involves, at a basic level, the motor system and the classical MNS.

In the present study, we aimed at exploring the link between the MNS and psychopathy by relating activity within the sensorimotor MNS for pain to psychopathic personality traits. To this end, the Psychopathic Personality Inventory (PPI; Lilienfeld and Andrews, 1996) was administered to a group of healthy individuals from the general population following the evaluation of corticospinal excitability of the motor cortex during passive observation of a needle penetrating the hand of a human model. Excitability of the motor cortex was evaluated when the needle was halfway through the skin or before it made contact with the model hand. This was done to determine the temporal course of excitability changes related to pain observation as complex time interactions have been reported for action observation (Gangitano et al., 2001). It was hypothesized that *i*) as in a previous study (Avenanti et al., 2005), passive observation of a needle penetrating the hand of a human model would significantly reduce the amplitude of the TMS-induced motor-evoked potentials (MEP); *ii*) these changes in excitability would be modulated in time, such that effects would be maximum when the needle was seen penetrating the model's skin; and *iii*) such changes in motor cortex excitability would be correlated with psychopathic personality traits.

2. Methods

2.1. Subjects

Eighteen male, right-handed college students (mean age=23.8 years, ±3.7) participated in the study. All participants gave written informed consent and the study