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Controlling automatic imitative tendencies: Interactions between mirror neuron and cognitive control systems

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ABSTRACT

Humans have an automatic tendency to imitate others. Although several regions commonly observed in social tasks have been shown to be involved in imitation control, there is little work exploring how these regions interact with one another. We used fMRI and dynamic causal modeling to identify imitation-specific control mechanisms and examine functional interactions between regions. Participants performed a pre-specified action (lifting their index or middle finger) in response to videos depicting the same two actions (biological cues) or dots moving with similar trajectories (non-biological cues). On congruent trials, the stimulus and response were similar (e.g. index finger response to index finger or left side dot stimulus), while on incongruent trials the stimulus and response were dissimilar (e.g. index finger response to middle finger or right side dot stimulus). Reaction times were slower on incongruent compared to congruent trials for both biological and non-biological stimuli, replicating previous findings that suggest the automatic imitative or spatially compatible (congruent) response must be controlled on incongruent trials. Neural correlates of the congruency effects were different depending on the cue type. The medial prefrontal cortex, anterior cingulate, inferior frontal gyrus pars opercularis (IFGpo) and the left anterior insula were involved specifically in controlling imitation. In addition, the IFGpo was also more active for biological compared to non-biological stimuli, suggesting that the region represents the frontal node of the human mirror neuron system (MNS). Effective connectivity analysis exploring the interactions between these regions, suggests a role for the mPFC and ACC in imitative conflict detection and the anterior insula in conflict resolution processes, which may occur through interactions with the frontal node of the MNS. We suggest an extension of the previous models of imitation control involving interactions between imitation-specific and general cognitive control mechanisms.

Introduction

During social interactions humans tend to mimic the postures and gestures of others. This mimicry is automatic in that it occurs without will or awareness (Chartrand and Bargh, 1999; Niedenthal et al., 2005). It also seems to be beneficial, increasing positive feelings and successful communication between social counterparts (Chartrand and Bargh, 1999; Lakin et al., 2003). The prevailing neural explanation for automatic imitative tendencies is that observing actions activates the corresponding motor program through a direct matching mechanism (reviewed in Heyes, 2011). This direct matching between observed and performed actions is thought to be mediated by the mirror neuron system (MNS) (Ferrari et al., 2009; Heyes, 2011; Iacoboni et al., 1999), which responds to both the observation of specific actions and the execution of similar actions. The strongest support for this model of automatic imitation comes from single-pulse transcranial magnetic stimulation (TMS), a technique that can be used to measure the cortico-spinal excitability of specific response representations. Many studies have now demonstrated that passive action observation causes increased cortico-spinal excitability specific to the muscles involved in producing the observed action (Baldissera et al., 2001; Borroni et al., 2005; Clark et al., 2004; D'Ausilio et al., 2009; Fadiga et al., 1995; Gangitano et al., 2001, 2004; Montagna et al., 2005). In other words, observing actions causes sub-threshold activation of the imitative response. This so-called "motor resonance" is reduced after the ventral premotor cortex (a putative MNS region) is disrupted with repetitive TMS, providing evidence that the frontal node of the MNS plays a causal role in the effect (Avenanti et al., 2007). In addition, TMS disruption of the same premotor region also reduces automatic imitation (Catmur et al., 2009), and social priming manipulations that modulate automatic imitation also modulate motor resonance (Obhi et al., 2011). Thus, there is increasing evidence for a link between motor resonance, the MNS and automatic imitation.

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While the neural substrates leading to automatic imitation are relatively well-studied, it is less clear how these automatic tendencies are brought under intentional control. Action observation automatically activates the corresponding motor representation, yet under normal circumstances we do not overtly imitate all observed actions. This is likely due to an active control system that inhibits unwanted imitation; the observation of patients who imitate excessively after large lesions in the frontal lobe (De Renzi et al., 1996; Lhermitte et al., 1986) suggests a disruption of this active imitation control mechanism. If imitation is supported by a specialized action-observation matching system (Iacoboni et al., 1999), imitation control may rely on neural systems distinct from other commonly studied control mechanisms. Specifically, imitative control may be different from control employed in Stroop, flanker and spatial compatibility tasks, where automatic response tendencies are evoked by non-social, symbolic stimuli. This hypothesis has received some support from neuroimaging (Brass et al., 2005) and neuropsychological (Brass et al., 2003) studies demonstrating dissociations between control processes in imitation and Stroop tasks and has led to the "shared representations" theory of imitative control (Brass et al., 2009; Spengler et al., 2009).

The shared representations theory proposes that a central process in imitation control is distinguishing between motor activity generated by one's own intentions from motor activity generated by observing someone else perform an action. This is required because both perceived and internally planned actions are represented in the same neural system (the MNS; Rizzolatti and Craighero, 2004), yet the system itself does not distinguish between the source of the representations (i.e. whether activity is caused by one's own intentions or the observation of others' actions; Jeannerod, 1999). Therefore, when two different (conflicting) motor representations are simultaneously activated by intentions and action observation, an imperative first step to carrying out the intentional action (and avoiding imitation) is to attribute each motor representation to either self or other.

Early support for the shared representations hypothesis came from the observation that neural substrates of imitative control are similar to those observed in more complex social tasks that also require self–other distinctions and the representation of conflicting mental states (Brass et al., 2005, 2009; Spengler et al., 2009). Specifically, the medial prefrontal cortex (mPFC) and temporo-parietal junction (TPJ) were shown to be involved in imitation control across a variety of studies (Brass et al., 2001b, 2005, 2009; Spengler et al., 2009; Wang et al., 2011b) and these regions are also involved in mentalizing, self-referential processing and determining agency (Amodio and Frith, 2006; Farrer and Frith, 2002; Farrer et al., 2003; Nahab et al., 2011; Ruby and Decety, 2001). Subsequent behavioral (Spengler et al., 2010b), neuropsychological (Spengler et al., 2010a, 2010c) and neuroimaging (Brass et al., 2009; Spengler et al., 2009) research provided more direct links between higher social cognitive functions and imitative control. Based on this work, Brass and colleagues proposed that in the context of imitative control the TPJ distinguishes between self- and other-generated motor activity by signaling that the observed action is related to another agent (regardless of the presence of conflict), whereas the mPFC enforces the self-generated action when it conflicts with an externally-generated action representation (Brass et al., 2009).

While the shared representations theory has gained traction, it does not describe mechanisms of imitation control beyond the involvement of mPFC and TPJ. For example, it is not clear how the mPFC resolves conflict between observed and intended actions after self-other distinctions are made. Furthermore, the mPFC and TPJ are not the only regions associated with imitative control tasks. The frontal operculum (Bien et al., 2009; Wang et al., 2011b) and ventral premotor cortex (Brass et al., 2005; Spengler et al., 2009) have also been observed to be active during imitation control. The inferior frontal regions have been interpreted as the frontal node of the human mirror neuron system (MNS) (Spengler et al., 2009; Wang et al., 2011b), suggesting that imitation control involves modulation of the MNS. However, this hypothesis has only received indirect support.

To build on previous models of imitative control we used dynamic causal modeling (DCM) for fMRI to examine causal interactions between regions involved in imitative control and to test the hypothesis that resolving imitative conflict involves MNS modulation. In an imitation interference task, subjects performed a finger-lifting action while simultaneously watching a video clip depicting either the same action or a different action. Numerous studies have demonstrated that subjects are slower to respond on incongruent trials, when the observed and performed action differ, compared to congruent trials, when the observed and performed action are the same (Bertenthal et al., 2006; Bird et al., 2007; Brass et al., 2000, 2001a, 2001b; Catmur and Heyes, 2010; Gillmeister et al., 2008; Kilner et al., 2003; Longo et al., 2008; Press et al., 2008; Stürmer et al., 2000; Wang et al., 2011a). This slowing is attributed to the recruitment of imitative control processes on incongruent trials; since the imitative response is incorrect, it needs to be inhibited to allow execution of the correct non-imitative response. Therefore, regions more active during incongruent compared to congruent trials are likely involved in imitation control.

In addition to the imitation interference task, we included a spatial interference paradigm that was identical except the stimuli depicted moving dots instead of moving fingers. The rationale for including the spatial task was twofold. First, it allowed us to identify regions that are involved specifically when conflict arises from action observation, in line with an imitation control mechanism that is distinct from mechanisms for overcoming automatic responses evoked by non-social, symbolic stimuli. In addition, comparing the imitation and spatial compatibility tasks provided a way to localize regions activated selectively for action observation so that we could identify putative mirror neuron regions within the same paradigm and subjects (Friston et al., 2006).

With a standard activation analysis based on the General Linear Model (GLM), we initially identified a specific imitation control network that was consistent with previous studies and included the frontal node of the MNS. Following this, we used DCM, a method of modeling effective connectivity, to test a set of alternative hypotheses about causal interactions between imitation control regions. We tested a set of models aiming to determine (1) whether the mPFC detects imitative conflict, as proposed by the shared representations model; (2) whether coupling between prefrontal regions and the MNS is stronger when control is required, as would be expected if imitation control involves modulation of MNS activity; and (3) which prefrontal control region interacts with the MNS.

Methods

Participants

25 adult subjects (15 female; age 19–39) were recruited through advertisement in the university newspaper and free online bulletins. All subjects were right-handed, had normal or corrected-to-normal vision, no history of neurologic or psychiatric disorders and were not taking psychoactive medications. Subjects were compensated for their participation and the study was approved by the UCLA Institutional Review Board. One subject was excluded from analyses for a structural abnormality and four additional subjects were excluded based on quality control criteria: two reported falling asleep during scanning and failed to respond on more than 15% of trials in two or more runs and two had excessive head motion (more than 10% of volumes with motion artifacts detectable by visual inspection in 2 or more runs). The remaining 20 subjects were included in data analysis, with 17 subjects entering the DCM analysis (3 did not show reliable activation maxima in one or more of the 4 ROIs).