Self-other control: a candidate mechanism for social cognitive function

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Despite ever-growing interest in the “social brain” and the search for the neural underpinnings of social cognition, we are yet to fully understand the basic neurocognitive mechanisms underlying complex social behaviors. One such candidate mechanism is the control of neural representations of the self and of other people (Brass et al., 2009; Spengler et al., 2009a), and it is likely that “common” disorders of social cognition such as autism and schizophrenia involve atypical modulation of self and other representations (Cook and Bird, 2012; Ferri et al., 2012). This opinion piece will first consider self-other control as a possible low-level neurocognitive mechanism for social functioning across many domains of social cognition. Neuroscientific evidence will be drawn upon and the potential for a better understanding and identification of neuropsychological markers for atypical social cognitive development, discussed.

A CANDIDATE MECHANISM

Humans are uniquely social beings and therefore identifying commonalities in the mechanisms recruited across various domains of social cognition is important, providing an understanding not only of typical social cognitive function but also what happens when this goes wrong. A candidate process which may be recruited across a range of socio-cognitive tasks is the ability to hold in mind and manage neural representations of both the self and of other people. Motor representations pertaining to the self and of the other are necessary in the case of imitation (di Pellegrino et al., 1992; Gallese et al., 1996), and self and other representations of mental and affective states are necessary for theory of mind and empathy, respectively (Decety and Grèzes, 2006; Brass and Spengler, 2008; Iacoboni, 2009). Within each of these domains of social cognition a form of “contagion” can be observed where information is shared between representations of the self and other. In the case of action observation, individuals automatically and often non-consciously imitate the actions of those with whom they interact (Chartrand and Bargh, 1999; Brass et al., 2000; Heyes, 2011).

Social interaction therefore appears to be facilitated by a shared representational system. However, social situations sometimes require an individual to distance themselves from other people and in other instances require one to engage more with representations of others. For example, when taking another’s perspective, engaging a successful theory of mind, or empathizing with others it is important to put aside or inhibit one’s own perspective, mental or affective state and enhance that of the interacting other. Conversely, in order to control the tendency to imitate others’ actions and generate our own independent actions, we must inhibit the motor representation pertaining to the interacting other and activate the motor representation for our own intended action. Differing requirements to inhibit or enhance representation of the self or the other for successful social interaction highlights the crucial role played by the ability to control or switch between neural representations attributed to the self and to other people, hereafter referred to as “self-other control” (Decety and Sommerville, 2003; Brass and Heyes, 2005; Spengler et al., 2009a).

A task now readily used as a behavioral index of self-other control is that of the control of imitation (Figure 1; Brass et al., 2001, 2005, 2009; Spengler et al., 2009a; Catmur and Heyes, 2011; Santiesteban et al., 2012a,b; Sowden and Catmur, 2013). The task requires participants to inhibit imitative response tendencies, and therefore provides an index of an individual’s ability to enhance the self-representation whilst inhibiting the other-representation. Additionally, Obbi and Hogeveen (2013) have proposed a complimentary task whereby performance under the opposite control requirements can be investigated; inhibiting the self-representation whilst exciting the other-representation. In combination, these tasks provide a neat index of control, the ability to supress not only representations of the other but also of the self.

Despite the very different higher-level cognitive processes involved in a wide range of social cognitive abilities, a series of behavioral findings in neurotypical adults support the existence of a common low-level mechanism of self-other control. Performance in one social domain such as the control of imitation correlates highly with performance in other social domains requiring self-other control. These include perspective-taking, theory of mind and empathy (Spengler et al., 2010a), and remain even when controlling for more general executive functioning processes (e.g., Spengler et al., 2010b). The link between performance on different tasks requiring self-other control is not merely correlational; training to inhibit
Imitative compatibility

Monitor display

Required response

Lift Index finger

Lift middle finger

FIGURE 1 | Example of stimuli presented and participant responses required in a task to measure the control of imitation. The task: Participants are instructed to make either an index or middle finger lifting action in response to a colored cue (orange or purple square) presented on the monitor display. The cue is also accompanied by a task-irrelevant hand performing an index or middle finger lifting action. Thus, task-irrelevant stimuli can be either imitatively compatible or incompatible with the required finger response. An index of self-other control is calculated by subtracting response times on compatible trials from those on incompatible trials. Imitative compatibility of the task-irrelevant stimuli with the required finger lift response is also indicated for trials in which an orange square indicates lift index and a purple square indicates lift purple.
of this region has been associated with impaired social cognitive performance, in particular theory of mind and emotion processing domains (Benedetti et al., 2009; Lee et al., 2011; Das et al., 2012).

More recently it has been suggested that the impairments seen in ASD and schizophrenia can be characterized as a failure of top-down modulation of social behavior (Southgate and Hamilton, 2008; Cook and Bird, 2012; Cook et al., 2012; Wang and Hamilton, 2012). Of particular note, Cook and Bird (2012) found that the modulatory effects of priming pro-social attitudes on self-other control observed in neurotypical adults were absent in individuals with ASD. In the same vein, reduced fronto-temporal functional connectivity is now a well-established feature of schizophrenia and has been linked to diminished top-down modulatory control over social behavior (Allen et al., 2008; Cook et al., 2012).

A NEUROCOGNITIVE MARKER FOR ATYPICAL SOCIAL COGNITION?

Although we know little about the precise developmental trajectories for the neurocognitive deficits discussed, by highlighting a mechanism with the potential to explain many facets of social cognitive function researchers may be better equipped to advise on neurocognitive markers and possible interventions for common disorders of social cognition. Self-other control emerges as such a candidate neurocognitive mechanism. Future assessment of disorders of social cognition can benefit from the now widely used task of imitative control (Figure 1) as a robust behavioral index of self-other control which includes the requirement for online modulation of both self- and other-representations in one task. Performance on imitative control tasks predicts performance across various domains of social cognition, and thus may provide a means to predict a pattern of atypical social developmental, in addition to measures of the structure and function of critical regions such as the rTPJ and mPFC. One may predict that individuals with autism or schizophrenia will be impaired at controlling imitative response tendencies, indicative of a deficit in self-other control.

This opinion piece has explored behavioral and neuroscientific evidence for self-other control as a candidate neurocognitive mechanism for social cognition. With advances in the field, a mechanism such as this may be crucial in identifying neurocognitive markers of atypical development and providing a therapeutic target to ameliorate the symptoms of atypical social development. Of particular promise from the application of such a mechanism is a unified account of the broad range of social functioning impairments associated with ASD and schizophrenia.

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