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Cognitive underpinnings of social interaction

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EPS Prize Lecture
Cognitive underpinnings of social interaction

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Human social interaction is part of what defines us. Here I present an overview of recent studies of imitation, a subdomain of social interaction that can be dissected and examined in a scientific fashion. I use these studies to test two core claims: (a) that there is more than one copying mechanism in the human brain and (b) that mimicry (a form of copying) is particularly relevant for understanding social behaviour. Evidence in favour of the first claim comes from neuroimaging studies that show distinct brain systems for understanding action kinematics, action goals, and irrational actions. Further studies of participants with autism show abnormal copying of irrational actions. Evidence in favour of the second claim comes from behavioural studies of the social cues that prime mimicry and from neuroimaging studies of the pathways involved in this priming. These studies suggest that medial prefrontal cortex has a core role in controlling mimicry responses and support the STORM (social top-down response modulation) model. Future work should determine what organizing principles govern the control of social responses and how these critical mechanisms for interpersonal connection differ in autism.

Keywords: Social interaction; Imitation; Autism; Mimicry.

The complexities of human social interaction have fascinated writers and artists for centuries. The scientific study of social interaction is more recent, and this huge field remains only weakly characterized. The present paper focuses on copying behaviour as an exemplar of social interaction that can be measured and studied in controlled settings. Here I set out some possible mechanisms underlying human copying behaviours, in the hope that these may contribute to the broader problem of understanding the brain and cognitive systems underlying social interaction.

Definitions

First, it is helpful to delineate the types of behaviour that can be labelled as "copying". Debate over what

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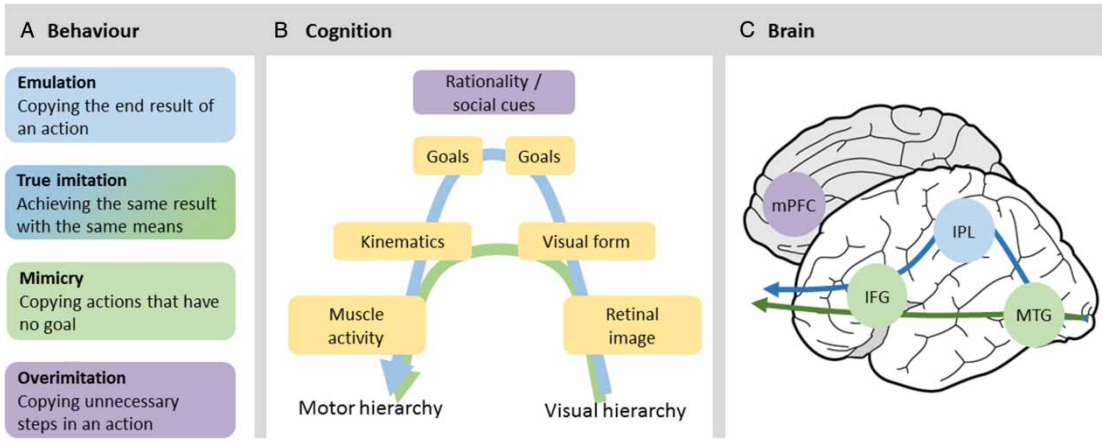

Figure 1. *Behaviour, cognition, and brain systems for copying. A. Four different types of imitation behaviour can be defined: Emulation, true imitation, mimicry, and overimitation. B. Cognitive processes involved in imitation. Both visual and motor actions can be represented in a hierarchical fashion, and matching can occur at multiple levels of the hierarchy. The green arrow indicates visual-form → kinematic matching, supporting mimicry. The blue arrow indicates goal-level matching supporting emulation. C. Brain systems for imitation: The emulation route and mimicry route can be mapped to different brain systems, with emulation (blue) from MTG → IPL → IFG and mimicry (green) from MTG directly to IPL. MTG = middle temporal gyrus; IPL = inferior parietal lobule; IFG = inferior frontal gyrus. To view this figure in colour, please visit the online version of this Journal.*

behaviours count as imitation and what imitation means has been active since before 1900 (Zental & Galef, 1988). Following this long tradition, studies of copying behaviour in nonhuman primates and other animals provide a careful classification of different forms of imitation. For example, Whiten, Horner, Litchfield, and Marshall-Pescini (2004) set out a taxonomy of social learning behaviours, including terms such as imitation, object movement reenactment, emulation, affordance learning, and stimulus enhancement. I borrow these terms and expand on them, to distinguish four types of copying present in human behaviour (Figure 1A). First, *true imitation* involves copying the result of a goal-directed action and the means by which that action is performed. For example, a new student watches someone press a sequence of buttons on a coffee machine and later presses the same buttons to obtain a coffee. Identifying true imitation has often been the goal of studies of imitation in other species (Whiten et al., 2004). Second, *emulation* involves copying only the goal or outcome of an action, but not necessarily using the same means (Tomasello, 1990). For example, a child sees the adult lift a book with one hand, but lifts it herself with her two hands. Third, *mimicry* involves copying the means or low-level features of an action without attending to a goal. For example, one student crosses her legs, and her friend does the same without either realizing (Chartrand & Bargh, 1999). Finally, *overimitation* involves copying features of an action even if those are not helpful to the action goal (Horner & Whiten, 2005). For example, a trainee sees a chef tap the knife on the edge of the board before rapidly chopping onions, and carefully does the same action.

Defining behaviours is a useful starting point, but a cognitive neuroscientist also seeks to understand the information-processing mechanisms that support the behaviour and how those might be implemented in the brain. In the past, I have made two claims concerning the mechanisms of imitation. First, I suggested that there are multiple routes for copying different actions (Hamilton, 2008) and that these routes contribute differently to the various copying behaviours that we observe. Second, I suggested that one of these routes, which I term the mimicry route, is specifically social and closely modulated by other social brain

systems (Wang & Hamilton, 2012). In the present paper, I reexamine these two claims and consider new evidence concerning imitation behaviour and its neurocognitive basis. I use this new evidence as a way to consider where our theories of social interaction can go next.

CLAIM 1: MULTIPLE ROUTES FOR COPYING

My first claim concerns the basic cognitive mechanisms of copying behaviour. There are many different models that attempt to describe how imitation might work. For example, in the active intermodal mapping (AIM) model (Meltzoff & Moore, 1997), visual images of an action and motor commands are both mapped to a supramodal representation, while proprioception provides a critical feedback system. In the direct mapping model (Rizzolatti & Sinigaglia, 2010), mirror neurons allow for a mapping between visual and motor systems. These two models both imply that imitation capacities are innate and likely to be present from birth (Ferrari et al., 2006; Meltzoff & Moore, 1977). In contrast, models like associative sequence learning (Heyes, 2001) suggest that imitation skills are learnt by general associative mechanisms and need not be innately specified. There has been substantial debate over these approaches (Heyes, 2009), and there is increasing evidence that learning has a major role in shaping imitation behaviour (Catmur, Walsh, & Heyes, 2007). However, all these models share the assumption that a single basic mechanism can account for many different types of copying, and they debate only the origins of this mechanism.

An alternative possibility is that different mechanisms are engaged for different types of imitation behaviour. Mechanisms might vary according to the effector used in a task (hand or face) or according to the different types of copying behaviour listed above. A number of different models of this type have been proposed. Studies of patients with brain damage have led to a dual-route model of imitation (Rothi, Ochipa, Heilman, & Gonzalez Rothi, 1991) in which meaningful actions are processed in a different way to meaningless actions. Further evidence in favour of this model (Rumiati, Papeo, & Corradi-Dell'Acqua, 2010) or even one with three routes (Buxbaum & Kalénine, 2010) has recently been found. In all these models, a core distinction is between copying of familiar, meaningful actions, which often involve objects (true imitation), and copying of novel, meaningless actions, which often have no goal or object (mimicry). A different argument for multiple imitation mechanisms comes from comparative studies of macaques and children (Subiaul, 2010) and suggests distinguishing between imitation of familiar and novel actions, as well as distinguishing between different effectors (hand or face). Finally, the EP-M model (Emulation & Planning–Mimicry), which I proposed (Hamilton, 2008), also suggests that different imitation behaviours (emulation vs. mimicry) might engage different neurocognitive pathways. Here I present an updated assessment of why multiroute models hold more promise for understanding imitation than single-route models.

Why multiple routes?

The first reason for suggesting that there might be multiple imitation routes is on a purely theoretical basis. We know that any action can be represented on multiple levels—her hand grasps the apple (kinematic level)—she wants to pick the apple (goal level)—she believes the apple is tasty (belief level; Grafton & Hamilton, 2007; Figure 1B). These multiple levels can apply to both performed actions and observed actions. We also know that imitation of an action requires a solution to a very complex correspondence problem—the retinal image of an action must be translated into the muscle activations required to perform the same action (Brass & Heyes, 2005). This must involve some level of abstraction away from those raw retinal/muscle representations, into a common space. Single-route models make different claims about the most appropriate level of abstraction to use. The associative sequence learning model, for example, focuses mainly on the kinematic level and suggests that visual feature of an action can

be mapped directly to motor features (Heyes, 2001). In contrast, the direct-matching model focuses on the goal level of representation and suggests that mirror neurons represent observed goals and allow implementation of the same action goals (Rizzolatti & Sinigaglia, 2010).

Though these models focus on matching at a single level, it seems more likely that the brain has the flexibility to use more than one level depending on the task and context (Figure 1B). Some types of copying (e.g., mimicry) might demand matching at the kinematic level, while other types (e.g., emulation) might require matching at the goal level. To test this hypothesis, it is useful to demonstrate two things—first, that different levels of action representation can be distinguished in the brain and, second, that goal-directed copying (emulation) and copying of kinematics (mimicry) can be differentially impacted in psychopathology. Here I review some recent evidence relevant to each of these.

Multiple routes in the brain

Within the human brain, a core visual–motor pathway provides the most likely substrate for the observation, copying, and implementation of visually guided hand actions. This includes higher order visual regions (extrastriate body area, EBA; middle temporal gyrus, MTG; superior temporal gyrus, STS), inferior parts of parietal cortex (inferior parietal lobule, IPL; anterior interparietal sulcus, aIPS), and inferior parts of premotor cortex (inferior frontal gyrus, IFG). Figure 1C illustrates some of these areas.

Previous work suggests that the inferior frontal region (IFG) is particularly important for representing the kinematic features of an action (Hamilton & Grafton, 2007; Pobric & Hamilton, 2006). This is coherent with single-cell studies showing sensitivity to different grasp types in this region (Brochier, Spinks, Umilta, Kirkwood, & Lemon, 2001; Kraskov, Dancause, Quallo, Shepherd, & Lemon, 2009). A study that used a repetition suppression functional magnetic resonance imaging (fMRI) method in humans showed cross-modal suppression (a signature of likely mirror neuron populations) in the IFG when performed and observed actions differ in basic kinematic features (Kilner, Neal, Weiskopf, Friston, & Frith, 2009). In contrast, the parietal components of this network (IPL and aIPS) seem more sensitive to action goals (Hamilton & Grafton, 2006) and even respond to goal-directed actions implemented by animated triangles that have no human kinematics (Ramsey & Hamilton, 2010). The aIPS also shows cross-model sensitivity to action goals in a multivoxel pattern analysis (Oosterhof, Wiggett, Diedrichsen, Tipper, & Downing, 2010). It is also possible to contrast coding of effectors (hand/foot) with coding of motor acts (push/pull); a study using this method found that IPL is sensitive to motor acts while IFG is sensitive to effectors (Jastorff, Begliomini, Fabbri-Destro, Rizzolatti, & Orban, 2010). Similarly, single-cell data show greater responses to more distant goals in parietal regions (Bonini et al., 2011; Fogassi et al., 2005). Together, these data suggest that parietal cortex may be more specialized for action goals, and inferior frontal cortex for action kinematics, though there is likely to be substantial overlap in these functions. The results also are consistent with the claim that the correspondence problem can be solved at multiple levels—matching of performed and observed actions can involve both kinematic and goal representations. This implies that there may be more than one route by which information can flow through the IPL–IFG visuomotor stream during copying behaviours.

Few studies have directly examined neural connectivity during imitation, but one has found interesting results. Sasaki and colleagues asked participants to execute or observe hand actions while watching hands moving two balls or two animated balls moving on their own (Sasaki, Kochiyama, Sugiura, Tanabe, & Sadato, 2012). They used dynamic causal modelling to contrast different models of brain connectivity in the fronto-parietal network during this task and found evidence for a model with multiple connections, including a direct link between posterior STS and ventral premotor cortex, and a second route from STS to IPL to premotor cortex. This possibility of two routes for visuomotor processes was explored in a 2008 paper (Hamilton, 2008), which suggested

that the MTG → IPL → IFG route is useful for representing goal-directed actions and supports emulation behaviour, while the MTG → IFG route is useful for representing kinematic features of actions and supports mimicry behaviour. These relationships are illustrated in Figure 1C. Future data may yet lead to specification of more than two routes (e.g., Buxbaum & Kalénine, 2010).

Beyond the visuomotor stream, there is also new evidence for another level of action representation in brain regions traditionally associated with theory of mind (Frith & Frith, 1999) including medial prefrontal cortex (mPFC) and temporoparietal junction (TPJ). For example, asking participants to consider action intentions when they view a picture of a person acting leads to engagement of mPFC and TPJ (de Lange, Spronk, Willems, Toni, & Bekkering, 2008; Spunt, Falk, & Lieberman, 2010). Stimuli that implicitly demand consideration of action intentions also seem to engage these brain systems. When participants observe irrational actions such as an actor turning on a light switch with her knee when her hands are free (rather than hands occupied), then mPFC and TPJ are activated (Brass, Schmitt, Spengler, & Gergely, 2007). In a related study, participants observed an actor moving a ball along a curved trajectory rated as "irrational", in contrast to the same curved trajectory to avoid an obstacle (rated as rational), a deactivation of mPFC was observed for the irrational actions (Marsh, Mullett, Ropar, & Hamilton, 2014). In both these cases, mPFC distinguishes between rational and irrational actions, and it is possible that this arises because observing irrational actions leads participants to mentalize about why the other person would do something silly. As I set out below, copying of irrational action (overimitation) is also an interesting behaviour, though the brain systems underlying it are not yet known.

Multiple routes in autism

A second way to test the claim that there are multiple routes for imitation is to show that different types of imitation are differentially affected in psychiatric or neurological disorders. Studies of neuropsychological patients detail how brain damage can impact on different types of imitation in line with a multiple-route model (Buxbaum & Kalénine, 2010; Rothi et al., 1991; Rumiati et al., 2010). There is also important evidence for multiple types of imitation from studies of developmental disorders—in particular, autism. Autism is a neurodevelopmental disorder impacting on social skills, and many studies have examined imitation in autism. If some types of imitation and some imitation-related brain systems are affected in autism more than others, this would support the multiple-routes hypothesis.

There are only a few brain imaging studies of imitation and action understanding in autism. It seems that brain activation in autism is abnormal when stimuli are emotional faces or actions, for both imitation (Dapretto et al., 2006) and observation (Grèzes, Wicker, Berthoz, & de Gelder, 2009). However, activations during observation of non-emotional faces (Grèzes et al., 2009) or hand actions (Dinstein et al., 2010; Marsh & Hamilton, 2011) are normal. In a recent review, I concluded that the mirror neuron system is not "broken" in autism (Hamilton, 2013). One limitation is that these neuroimaging studies did not specifically distinguish different levels of action representation, so it is also helpful to consider behavioural studies.

There are a very large number of studies of imitation behaviour in individuals with autism spectrum condition (Williams, Whiten, & Singh, 2004), and most do not discriminate between different types of imitation such as emulation or mimicry as classified in Figure 1A. This means it has often been hard to determine what is different about imitation in autism. By focusing on studies that use clear definitions of emulation or mimicry, we can gain much greater insights. An early study provided the key insight that emulation and mimicry differ in autism (Hobson & Lee, 1999). Participants saw an adult perform novel actions on objects (e.g., hit a box as if it were a violin) in different styles (e.g., gentle or harsh). Typical children emulated the action and mimicked the style, but children with autism only emulated and failed to mimic the action style. A number of other studies report intact imitation of goal-directed

actions (emulation) in participants with autism spectrum condition (Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001). In contrast, spontaneous or socially driven mimicry of actions is often abnormal in autism (Ingersoll, 2008; Ingersoll, Schreibman, & Tran, 2003). Based on these findings, I suggested that emulation might be intact in autism, while mimicry might be impaired (Hamilton, 2008). A new study directly tested this using an end-state comfort effect and found that adolescents with autism were able to emulate goal-directed actions but showed a reduced tendency to mimic inefficient actions (Jiménez, Lorda, & Méndez, 2013). Finally, a recent meta-analysis of studies of imitation in autism reinforces this claim, finding a reliable impairment in high-fidelity imitation across 27 studies, but no impairment in emulation across seven studies (Edwards, 2014).

The difference between responses to rational actions and irrational actions provides an alternative means to partition the space of possible types of imitation and thus understand different routes for imitation in autism. In typical participants, observing irrational actions leads to engagement of "mentalizing" regions of the brain (mPFC and TPJ; Brass et al., 2007). We recently used irrational action stimuli to examine responses in mirror systems and mentalizing systems in participants with autism (Marsh & Hamilton, 2011). Nineteen typical adults and 18 matched adults with autism spectrum condition (ASC) watched video clips of goal-directed hand actions and irrational actions during fMRI. Both groups of participants engaged left aIPS when watching goal-directed actions and right aIPS when watching irrational actions. This demonstrates that parietal components of the mirror neuron system are functioning normally in ASC. In contrast, group differences emerged in mPFC when participants observed irrational actions—typical participants showed a deactivation of this region only when irrational actions were observed, whereas ASC participants did not. These results suggest that distinct brain systems are involved in responding to rational and irrational actions, and that only the latter functions abnormally in ASC. This further implies that behavioural differences may be found when participants with and without ASC are given the opportunity to imitate irrational actions.

A small number of studies have examined behavioural responses to irrational actions in participants with ASC. Wild et al. conducted an imitation task in which participants were asked to watch a video of a hand making pointing movements and then to perform the same sequence of actions (Wild, Poliakoff, Jerrison, & Gowen, 2012). Critically, the movement speed and height were varied from trial to trial, but participants were not instructed about this; they were simply told to copy the goal. Adults with autism were able to accurately reproduce the horizontal extent of each movement—that is, they correctly imitated the action goal as instructed. This replicates findings of normal goal imitation in autism (Hamilton, Brindley, & Frith, 2007). However, typical adults spontaneously adapted their movement speed and height to more closely match the demonstrator on each trial, but participants with autism did not. This is particularly interesting because actions that are exceptionally high (reaching for an object as if moving over a barrier) are commonly studied as "irrational" actions. Thus, the participants with ASC in this study are emulating the action goals but not mimicking the irrational change in action height. This parallels differences in brain activation, when participants with autism respond normally to rational actions using parietal cortex but not to irrational actions using mPFC (Marsh & Hamilton, 2011). Overall, these studies suggest that typical participants spontaneously engage in both emulation and mimicry, but participants with autism tend to emulate actions without mimicking the unnecessary components.

To explore imitation of irrational actions in autism more directly, we turned to the phenomena of overimitation. Overimitation is a fascinating behaviour, where typical children and adults (but not apes) copy more than they need to. This has typically been studied using puzzle boxes or artificial fruit, where the participant sees a demonstrator open a novel box using some helpful actions and some unnecessary actions (Whiten, McGuigan, Marshall-Pescini, & Hopper, 2009). The use of

novel objects provides strong evidence that the observer learns something from the demonstrator. However, if a child does copy an irrational action on a novel object, it is not always easy to tell whether this occurred because the child is bad at causal reasoning (and does not know what is rational) or if they are genuinely motivated to copy the irrational action. Thus, we studied overimitation using familiar objects where causal reasoning demands are minimal. For example, in one task, the demonstrator takes a lunch box with a toy duck inside, opens the clips, taps on the lid twice (unnecessary), and then removes the lid and takes the duck out. The child is then given the reassembled box and asked "get the duck out as quickly as possible". Copying of the unnecessary tapping action was classified as overimitation. After the five different overimitation trials were complete, children saw the individual actions within a sequence again and were asked to judge whether each action was "sensible" or "silly". This gives a measure of whether the child knows that an irrational action really is irrational (silly). We found that typical 5–11-year-olds overimitate the adult's action, and they show this behaviour more if they are older, are more socially engaged, and have better causal reasoning (Marsh, Ropar, & Hamilton, 2014). This strongly suggests that overimitation is a socially motivated behaviour.

In a second study, we tested whether overimitation was different in children with autism (Marsh, Pearson, Ropar, & Hamilton, 2013). We used the same overimitation task with familiar objects and tested 30 children with ASC, 30 verbal mental age matched typically developing children (TD), and 30 age-matched TD. We found that the typical children overimitated on 40–50% of trials, while the ASC only overimitated on 22% of the trials. This difference was not due to exceptionally good causal reasoning in the ASC children, because they were also less accurate at distinguishing "sensible" from "silly" actions. Thus, this study provides further evidence that social forms of imitation are abnormal in autism. It is worth noting that other studies of overimitation in autism have yielded contradictory results (Nielsen, Slaughter, & Dissanayake, 2013), possibly because novel tool-use actions were used. If overimitation is reduced in autism, this implies that children with autism may lack the social motivation to imitate even when the actions they observe are simple and within their motor capacity. It is not yet clear whether in typical participants, overimitation is driven by a need to be congruent with the demonstrator (social affiliation) or by a need to be part of the group (social norms), nor which of these two aspects of social motivation might be different in autism. In either case, the reduction of overimitation in autism provides a clear contrast with earlier work showing good goal-directed imitation in autism, and it adds to the idea that different types of imitation depend on different neurocognitive systems.

Summary

The data reviewed above demonstrate that there are several different types of imitation behaviour, which depend on different brain and cognitive systems. Goal-directed actions are processed in inferior parietal cortex and are emulated normally by participants with autism. Irrational actions demand processing in medial prefrontal cortex where brain activity differs in autism, and irrational actions are less likely to be overimitated by autistic children. The kinematic features of without goals can be mimicked and may depend on processing in inferior frontal gyrus. There are also hints in these data that overimitation of irrational action is similar to mimicry of kinematic features of actions —both are abnormal in autism, and neither has a clear goal. The relationship between brain regions involved in mimicry and overimitation is considered below.

The fact that we can distinguish different types of imitation in terms of behaviour, brain systems, and impairment in autism gives fuel to evaluate the claim that there are multiple routes for copying behaviour. It would be hard to fit these different behaviours under a single "imitation system". Instead, it is likely that flexible models with more than one route will be a helpful way to understand neurocognitive systems for social interaction.

CLAIM 2: THE MIMICRY ROUTE IS SOCIAL

The second claim concerns the mimicry route and the emerging links between this route and social interaction. A key theme in understanding actions is the idea that actions are organized in a hierarchical fashion (Grafton & Hamilton, 2007) with basic kinematic representations contributing to more complex goal representations and then intention representations. It is also commonly assumed that the higher levels of the hierarchy are more "social" and more interesting from the point of view of social neuroscience (Hamilton, 2009). For example, understanding of intentions is closely linked to understanding of other mental states like beliefs, and thus theories of how intention understanding works have been developed in detail (Jacob & Jeannerod, 2005; Rizzolatti & Sinigaglia, 2010).

However, new data suggest that this hierarchical framework may have to be turned on its head. Both mimicry and overimitation behaviours draw on the lowest, kinematic levels of the action hierarchy, and both seem to be highly "social" behaviours (see also Gowen, 2012). This does not mean that mimicry and overimitation are linked directly to theory of mind; they may contribute to other aspects of social cognition like affiliation and group coherence. In contrast, emulation of goal-directed actions may be the least "social" level, as goals can be determined by consideration of objects and their environmental constraints. The present section examines in more detail the case that mimicry of the kinematic features of action is strongly modulated by and strongly interacts with other types of social information processing. Here I explore two specific claims. First, I suggest that the mimicry route is subject to top-down control from other brain systems for social information processing, with a particular focus on mPFC. Second, I suggest that these top-down control processes are particularly affected in autism, giving rise to abnormal mimicry behaviours and possibly reduced overimitation.

The social modulation of mimicry

Copying of simple, kinematic features of an observed action might seem to be a straightforward behaviour, but there is an increasing amount of evidence that this mimicry is subtly modulated by a large number of social cues. Mimicry has been studied under the rubric of the chameleon effect in live-interaction contexts (Lakin, Jefferis, Cheng, & Chartrand, 2003), and studies show more mimicry when interacting with someone from the same social group (Bourgeois & Hess, 2008) or someone with an attractive appearance (van Leeuwen, Veling, van Baaren, & Dijksterhuis, 2009) and less when interacting with someone with social stigma (Johnston, 2002). It is interesting to note that tasks used to study mimicry in this social psychology context could also be considered overimitation tasks (e.g., using the same colours to colour a picture; van Leeuwen et al., 2009). However, studies using live social interactions are hard to fully control and characterize. Within a cognitive psychology context, it is possible to study mimicry using well-controlled reaction time tasks developed by Brass and Heyes (Brass, Bekkering, & Prinz, 2001; Heyes, 2011). In these tasks, the participant makes a hand or finger movement in response to a preinstructed cue. An image or video of a hand or finger movement is presented at the same time, but should be ignored. Faster reaction times when a congruent stimulus is presented and slower reaction times to an incongruent stimulus give a robust measure of mimicry. It is then possible to test what factors modulate this mimicry effect.

In a series of studies, we have shown that eye contact is a rapid modulator of mimicry. Direct gaze occurring just before the movement cue leads to faster mimicry responses (Wang, Newport, & Hamilton, 2011), and this eye contact effect occurs only if the participant is gazed-at at the time of responding, not if the participant receives a direct gaze cue, which is then blocked before the response (Wang & Hamilton, 2014a). This suggests that the eye-contact effect is similar to an audience effect and may arise because participants feel that the other person is monitoring them. Using fMRI, it is

possible to examine the brain mechanisms underlying these effects. We found that the behavioural interaction between gaze and congruency is reflected in a blood-oxygen-level-dependent (BOLD) signal interaction in mPFC, STS, and IFG (Wang, Ramsey, & Hamilton, 2011). These three regions are strongly associated with social cognition. In particular, mPFC is known to respond to gaze and to mimicry; IFG is part of the human mirror neuron system; and STS processes biological motion as a possible input to the mirror neuron system. To determine how these regions were processing information during the task, we used dynamic causal modelling to compare 12 possible cognitive models. The "winning" model has several interesting features—first, mPFC provides a top-down input to both STS and IFG throughout the task. Second, the interaction of gaze and mimicry acts on mPFC and increases the strength of the connection from mPFC to STS. This suggests that mPFC is the core controller of the mimicry effect, acting on the lower level mirror system regions to either inhibit (as shown previously; Brass, Derrfuss, & von Cramon, 2005) or enhance the response to the observed action. For example, when direct gaze is present, mPFC may be engaged to enhance mimicry responses; in contrast, when direct gaze is absent, strong activation of mPFC is seen in the condition when participants must inhibit their natural tendency to mimic. This leads to the idea that mPFC plays a subtle and flexible role in the top-down control of imitative responding.

We further tested whether the role of mPFC in the control of mimicry generalizes to other types of priming stimuli. The domain of social priming has been somewhat controversial, especially the priming of action goals (Bargh, Schwader, Hailey, Dyer, & Boothby, 2012; Doyen, Klein, Pichon, & Cleeremans, 2012). Here we examine the impact of conceptual priming with prosocial or antisocial concepts on mimicry, where mixed results have also been found. For example, one study reports that priming with prosocial sentences leads to more mimicry (Lakin et al., 2003) while another suggests that viewing an antisocial event (ostracism) leads to more overimitation (Over & Carpenter, 2009). We first implemented a behavioural study to determine whether priming with prosocial or antisocial concepts affects mimicry. In this study, participants alternated between a scrambled sentence task and a mimicry task. The mimicry task was a simple reaction time task in which participants respond to a number on the screen while ignoring an adjacent finger movement—mimicry can be measured as the difference in reaction time on congruent and incongruent trials (Brass et al., 2001). The scrambled sentences described two people engaged in a prosocial or antisocial interactions. Initial results demonstrated that participants showed stronger mimicry after reading about an antisocial interaction than after reading about a prosocial interaction.

However, an independent study of how priming with prosocial or antisocial sentences affects mimicry in a simple reaction time task found the opposite result, with more mimicry following prosocial priming (Leighton, Bird, Orsini, & Heyes, 2009). Close examination of the priming stimuli suggested differences in the point of view present in the sentences—the sentences in our study described third-person events while Leighton et al.'s (2009) stimuli describe first-person or abstract events. Thus, in a second study, we directly manipulated the point-of-view in the scrambled sentence primes. We found that participants mimic more following first-person prosocial sentences or third-person antisocial sentences, but mimic less following third-person prosocial or first-person antisocial sentences. This result was confirmed in a third study with a novel video-description priming method, in which participants saw videos of helpful/unhelpful actions (based on Hamlin, Wynn, & Bloom, 2007) and described the actions from a first-person or third-person point of view. Together, these data suggest that the self-relatedness of the stimuli is critical for effective social priming (Wang & Hamilton, 2013). Furthermore, it is now clear that conceptual priming depends on the full meaning of the scrambled sentence and not just on the presence of a single prime word.

The results of this study are congruent with the active-self model of social priming, which suggests that the effect of primes depends on how they

engage the participant's self-concept (Wheeler, Demarree, & Petty, 2007). In this model, participants have a latent self-concept that includes the idea "I am prosocial". When a participant encounters a first-person prosocial prime, this prosocial self is activated and enhances mimicry. Also, when a participant encounters a third-person antisocial prime, he or she feels "I'm nicer than him" and activates the prosocial self, thus enhancing mimicry. In contrast, the first-person antisocial and third-person prosocial conditions do not engage the active self or enhance mimicry. This theory predicts that self-related brain regions should thus be engaged for the first-person prosocial and third-person antisocial priming conditions.

Using fMRI, it is possible to test these predictions. We adapted our scrambled sentence study to suit fMRI scanning and examined brain activation during both the priming phase (scrambled sentences) and the response phase (mimicry). In particular, we tested for a three-way interaction between point-of-view (first person/third person), social valence (prosocial/antisocial), and mimicry (congruent or incongruent responses). This pattern was found only in the anterior medial prefrontal cortex, a region linked to social schemas and self-related processing (Wang & Hamilton, 2014b). This region showed a similar but weaker pattern of activation during the priming phase of the study, and activation at priming predicted the behavioural effect seen during mimicry. These results match the predictions derived from the active-self model and support the claim that mPFC is particularly relevant for the top-down control of mimicry responses. It is also interesting to note that mPFC activation during the mimicry phase (implementation of top-down control) was larger than that during the priming phase (unscrambling sentences). This suggest that conceptual priming is not due to leftover processing from the priming, but rather is a more active process at the time when a response is made.

The STORM model

Based on these findings and others, we proposed a neurocognitive model of social responding called STORM (social top-down response modulation; Wang & Hamilton, 2012). This model has two key features (Figure 2). First, a visuomotor stream provides a mapping from perception of other people's actions to appropriate responses. This involves the flow of information from higher order visual regions (EBA/STS) to premotor cortex (IFG) and possibly inferior parietal cortex (IPL). In various literatures this is termed the perception–behaviour expressway (Bargh & Dijksterhuis, 2001) or the mirror neuron system (Rizzolatti & Sinigaglia, 2010). It is likely that the visuomotor links made here are determined by associative learning over the lifetime (Heyes, 2011), whereby participants associate the actions they perform with the sight of their own or other people's actions. Thus, this visuomotor stream specifies potential actions that can be performed, given the people and objects present in the world.

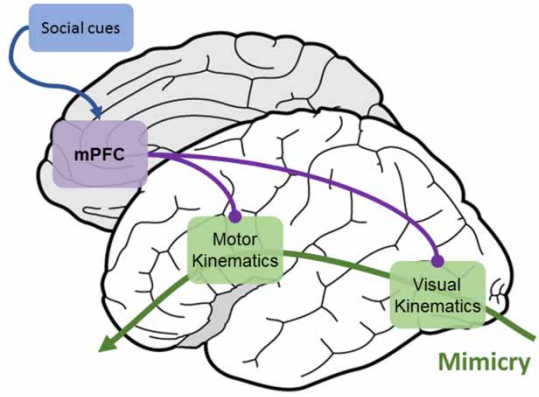

Figure 2. *The STORM model. In this model, the visuomotor stream (green) is influenced by mPFC (medial prefrontal cortex), which processes social cues and contributes to selecting an appropriate response. To view this figure in colour, please visit the online version of this Journal.*

Second, the STORM model proposes that the selection of which action should actually be performed is determined by top-down signals, originating from prefrontal cortex. In cognitive terms, mimicry results like the automatic imitation paradigms used here are commonly explained in terms of dual-route models (Heyes, 2011) in which a prelearnt visuomotor association route competes with a task-specific cue–response route. The top-down

control signal from mPFC could act to boost one of these routes, at either the input level or the output level. For example, boosting the processing of hand action inputs following direct gaze would make a mimicry response happen faster, as seen in Wang, Newport et al. (2011). This top-down control is able to take into account social cues such as eye contact, but maybe also social context and emotional valence, to determine which response to implement. This model has parallels in earlier models of motor control and executive function. Cisek and Kalaska proposed a motor control model in which a visuomotor stream specifies possible actions in parietal-premotor cortex, in parallel with an action selection mechanism in prefrontal cortex/basal ganglia (Cisek & Kalaska, 2010). Earlier, the supervisory attention scheme also comprised a stream of possible actions and a top-down selection mechanism (Norman & Shallice, 1986). The STORM model builds on these earlier models and proposes a specifically social mechanism of top-down control. A key question for future research is if and how social top-down control can be distinguished from other modes of executive function.

The STORM model can also help us understand the data concerning imitation behaviour in individuals with autism spectrum condition. As reviewed above, children with autism are able to imitate goal-directed actions, but show differences in their imitation of unnecessary and irrational actions. STORM suggests that these differences do not arise directly from a "broken mirror system" in autism or other differences in the basic visuomotor stream. Rather, participants with autism may differ in the top-down control mechanism that they use to interpret the rationality of actions and decide what to imitate. Neuroimaging evidence for this position comes from our study of observation of rational and irrational actions, where typical and autistic participants differed only in the engagement of mPFC when viewing irrational actions (Marsh & Hamilton, 2011).

Behavioural studies also suggest that modulation of copying behaviour is abnormal in autism. Cook and Bird used Leighton's scrambled sentence paradigm in two matched groups of typical and autistic adults. They found that, unlike typicals, autistic adults did not mimic more following priming with prosocial sentences (Cook & Bird, 2012). Using a similar task, Greccuci et al. showed that typical children mimic more following exposure to a fear face, whereas autistic children mimicked less (Grecucci et al., 2013). In both these studies, the basic mimicry response was present in the autistic participants but the modulation of this response by social cues was absent. These behavioural results can potentially be understood in terms of the STORM model and previous neuroimaging data, which suggest that mPFC is responsible for top-down control of mimicry. If mPFC is not engaged by social cues or by irrational actions in participants with autism, this could account for abnormal mimicry behaviour and overimitation behaviour in these individuals. This means there is no need to posit abnormalities of the mirror neuron system (MNS) in autism, and evidence for such abnormalities remains weak (Hamilton, 2013). Rather, differences in top-down control of imitation responses, a process implemented in mPFC, can account for the differences seen in imitation behaviour in autism. In cognitive terms, these differences in mPFC control of mimicry could reflect differences in the strength of the active self in individuals with autism, or differences in the motivation to engage with others, to be part of the social group and to manage ones reputation in a social group. Distinguishing between these remains a challenge.

CONCLUSION

Finally, this work leads on to a number of future questions. First, further specification of the STORM model will be required. The model so far suggests that top-down modulation of mimicry originates in mPFC, but it will be important to consider the role of other prefrontal and subcortical systems in controlling mimicry. For example, emotion (Heerey & Crossley, 2013) and reward (O'Connell, Christakou, Haffey, & Chakrabarti, 2013; Sims, Van Reekum, Johnstone, & Chakrabarti, 2012) can also modulate

mimicry behaviour. Second, the role played by mPFC in social interaction is complex. This region has been reported to be engaged in tasks involving rationality detection (Brass et al., 2007), control of imitation (Wang, Ramsey, et al., 2011), theory of mind (Frith & Frith, 1999), self-related processing (Amodio & Frith, 2006), and others. It will be important to determine whether there is a common factor or organizing principle that defines the computations and information processing being carried out in this region. A related question, highlighted above, concerns how this social top-down control relates to other forms of executive function.

Beyond these details of the STORM model, we can also consider the overarching question of why do we imitate, and why do we detect imitation from others—what value does imitation add to our behaviour? Why does mPFC need to control mimicry in such a precise and subtle fashion? An early treatise on imitation suggested that this behaviour has two functions—to learn about the world and to communicate mutuality with others (Uzgiris, 1981). Most research on imitation over the last 20 years has focused on the social learning component of imitation; the current review suggests that it is now critical to consider the social connectedness component too. The STORM model provides one possible way to understand the neurocognitive mechanisms that underlie interpersonal connections and thus to understand more about human social interaction.

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