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Neural systems for preparatory control of imitation

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Humans have an automatic tendency to imitate others. Previous studies on how we control these tendencies have focused on reactive mechanisms, where inhibition of imitation is implemented after seeing an action. This work suggests that reactive control of imitation draws on at least partially specialized mechanisms. Here, we examine preparatory imitation control, where advance information allows control processes to be employed before an action is observed. Drawing on dual route models from the spatial compatibility literature, we compare control processes using biological and non-biological stimuli to determine whether preparatory imitation control recruits specialized neural systems that are similar to those observed in reactive imitation control. Results indicate that preparatory control involves anterior prefrontal, dorsolateral prefrontal, posterior parietal and early visual cortices regardless of whether automatic responses are evoked by biological (imitative) or non-biological stimuli. These results indicate both that preparatory control of imitation uses general mechanisms, and that preparatory control of imitation draws on different neural systems from reactive imitation control. Based on the regions involved, we hypothesize that preparatory control is implemented through top-down attentional biasing of visual processing.

1. Introduction

There is now a substantial body of work demonstrating that humans have an automatic tendency to imitate others. Converging evidence suggests that this phenomenon is mediated by the mirror neuron system (MNS) [1], which comprises a number of cortical areas that respond both when we observe an action and when we perform a similar action. As a result of the overlap between visual and motor action representations, simply observing an action leads to activation of the matching motor representation (i.e. the imitative response) [2–6].

The fact that observing actions activates an imitative response raises the question of how this motor activation is modulated, allowing subconscious mimicry in some circumstances [7] while avoiding perpetual imitation. Data from patients suggest that an active inhibitory mechanism exists to control imitation. This is demonstrated most strikingly by the observation of disrupted control of imitation in various disease states: rare patients with large frontal lobe lesions compulsively imitate observed actions [8,9] and, more commonly, excessive imitation occurs in patients with neuropsychiatric disorders and neurodegenerative disorders, in the form of echolalia (mimicry of speech) and echopraxia (mimicry of movements) [10–13].

Recent work has provided insight into the neural systems responsible for imitation control, focusing in particular on reactive control processes that inhibit imitation after an action is observed. Interestingly, reactive imitation control has been shown to rely on different mechanisms from control over other types of automatic response tendencies, including control processes evoked by Stroop [14,15] and spatial compatibility tasks [16]. The existence of a unique imitation control mechanism may be related to the social nature of automatic imitation [17,18], as well as to the fact that the specialized MNS supports automatic imitation. In line with this view, reactive control of imitation has been shown to

draw on social-cognitive functions related to distinguishing between self and other [19–21]. In addition, accumulating evidence suggests that imitation control may be implemented through modulation of activity in the mirror neuron system [16,21,22].

In contrast to reactive control, there has been no work to date looking at preparatory imitation control mechanisms that may be used before an action is observed. If one can foresee that an automatic imitative tendency will disrupt behaviour, a preparatory control mechanism may be able to bias processing so that the motor system is less sensitive to the influence of observed actions. This type of preparatory control mechanism could prevent action observation from activating the imitative response in the first place, in contrast to reactive control mechanisms previously studied that inhibit an unwanted imitative response after the action is observed. The excessive imitation observed in neuropsychiatric disorders could be attributed to either type of mechanism. A deficit in preparatory control would result in larger than normal motor activation in response to observed actions, whereas a deficit in reactive control would result in the behavioural expression of motor activity that can normally be suppressed. Given increasing evidence from the broader cognitive control literature that preparatory and reactive forms of control may involve at least partially distinct mechanisms [23–25], it is plausible that preparatory control of imitation involves mechanisms distinct from those previously studied in reactive control paradigms.

To explore preparatory control of imitation, we draw on cognitive dual route models that have been widely applied to stimulus–response compatibility (SRC) effects similar to those observed in imitation interference paradigms [26–28]. Automatic imitation refers to a special case of SRC where observing an action facilitates performance of a similar (compatible) action and slows performance of a different (incompatible) action. Dual route models attribute differences in compatible and incompatible response times to two parallel response activation routes. An intentional route (often called the 'short-term' or 'indirect' route) links stimuli and responses according to task rules—this route can accommodate any stimulus–response pair by applying the relevant stimulus–response rule held in short-term memory. At the same time, a parallel fast and automatic route (often called the 'long-term' or 'direct' route) activates the stimulus-compatible response as a result of long-term stimulus–response associations or similarities between properties of the stimulus and response. According to this dual route processing framework, behavioural compatibility effects occur as follows: when a task requires a stimulus-compatible response both the rule-based and automatic routes activate the correct response and therefore performance is fast and accurate. By contrast, when the task requires a stimulus-incompatible response, the automatic response route (which activates the incorrect, compatible response) must be inhibited, so that the slower, rule-based response can be performed.

Importantly, the automatic response route can be inhibited in preparation for trials in which it is likely to interfere with task goals [29–34]. In SRC tasks where subjects are instructed to perform either the compatible or the incompatible response to a stimulus, preparatory inhibition of the automatic response route occurs in two scenarios: (i) preparation for incompatible trials, because the automatic response will be incorrect and (ii) preparation for trials in which the required stimulus–response mapping (compatible or incompatible) is not provided in advance of the stimulus, because the automatic compatible response will be incorrect on half of trials. This preparatory suppression of the automatic route manifests as reduced behavioural compatibility effects on unknown mapping trials: the compatible response no longer benefits from automatic response activation making response times for compatible and incompatible trials similar. In the alternative circumstance—when the required mapping is known before the stimulus—the automatic response route can be suppressed selectively in preparation for incompatible trials, so that compatible trials have a speed advantage owing to automatic response activation [31,32].

According to these cognitive models, preparatory suppression of automatic response activation can be examined by comparing preparatory activity on incompatible trials and trials in which the mapping is unknown (which both involve preparatory suppression) compared to compatible trials, when the automatic response route is used and there is no suppression. Accordingly, we developed a novel SRC task using biological and non-biological cues to examine the neural correlates of preparatory control. We aimed to (i) identify neural correlates of preparatory inhibition of imitative tendencies to determine whether they are similar to previously described reactive imitation control mechanisms [15,16,19–21,35]; and (ii) compare biological and non-biological stimuli to determine whether preparatory control of imitation is distinct from preparatory control of non-imitative response tendencies, similar to the specificity observed in previous studies of reactive imitation control [15,16]. In addition, as distinct control mechanisms for biological and non-biological stimuli are proposed to be related to the specialized role of the MNS in automatic imitation, we also examined activity related to automatic response activation for biological and non-biological stimuli.

2. Material and methods

(a) Participants

Thirty seven participants (20 female; 18–34 years old) were recruited from the UCLA community through posted fliers and undergraduate subject pools. All subjects were right-handed, had normal or corrected-to-normal vision, no history of neuropsychiatric disorder and were not taking psychoactive medication. Three subjects were excluded because of technical failure preventing response recording, and two were excluded for poor performance (one failed to respond on more than 10% of trials and reported falling asleep; the second performed with less than chance accuracy on one condition and below 3 s.d. from the mean across conditions). Data from the remaining 32 participants were included in data analysis. Written informed consent was obtained from all participants and they received monetary compensation or course credit for their time. The study was approved by the UCLA Institutional Review Board.

(b) Behavioural paradigm

Subjects performed compatible or incompatible finger-lifting responses to either biological (finger) or non-biological (dot) stimuli. In the biological condition, videos depicted a left hand lifting either the index finger or the middle finger (figure 1*a*). In the non-biological condition, one of two small black dots moved upwards. The dots were arranged based on the index

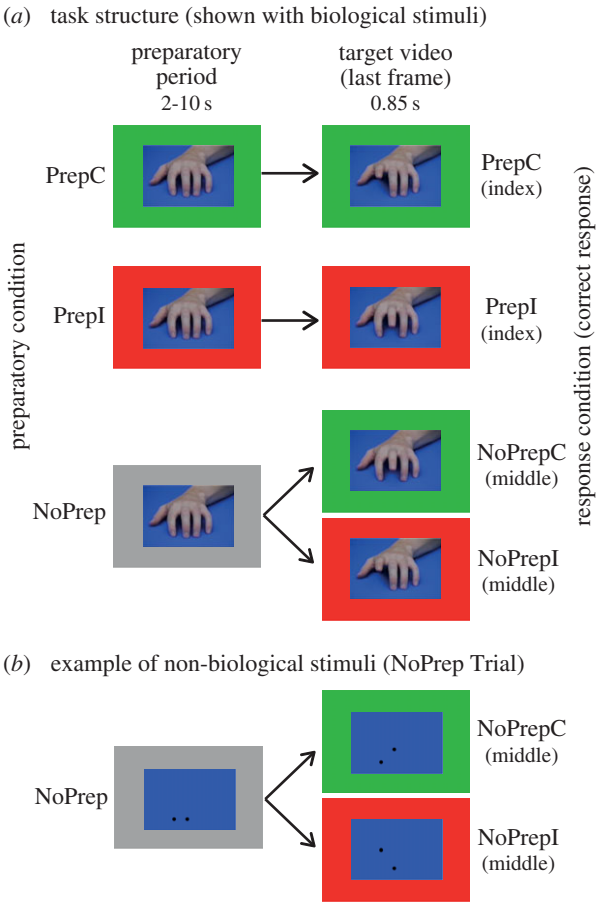

Figure 1. Task design. (*a*) Preparatory and response conditions are shown with biological stimuli. Preparatory and target period condition designations, as well as the correct response (in brackets), are noted in the margins. (*b*) Example of non-biological stimuli shown in NoPrep trial.

and middle fingertips in the hand videos, with similar initial and final positions and trajectories (figure 1*b*).

Subjects performed compatible or incompatible responses to the videos by lifting their own right index or middle finger. The appropriate stimulus–response mapping rule (compatible or incompatible) changed from trial to trial and was indicated by the colour of a thick border around the video (mapping cue). When the border was green, participants performed the compatible response: they imitated the hand videos (i.e. lift index finger in response to index finger video and middle finger in response to middle finger video) and performed the left/right spatially compatible response for the dot videos (i.e. lift index finger in response to left upward moving dot video and middle finger in response to the right dot). When the border was red, participants performed the incompatible response, counter-imitating the hand videos (e.g. middle finger response to index finger video) and performing the spatially incompatible response for the dot videos (middle finger response to left dot stimulus).

The ability for participants to prepare the upcoming response mapping was manipulated by varying the time at which the mapping cue was provided. Movement of the finger or dot in the video occurred 2, 5, 7 or 10 s after the onset of the first video frame, which depicted the finger/dots in a static starting position. Thus, the first frame of the video represented the preparatory period and the movement frames represented the response period. On two-thirds of trials, the mapping cue (green or red border) was provided during the preparatory period so that subjects could prepare the compatible (PrepC; one-third of trials) or incompatible (PrepI; one-third of trials) response mapping in advance of the stimulus. In the remaining one-third of trials (NoPrep), the border remained grey throughout the preparatory period and changed to green or red at the onset of the movement in the video. Therefore, on these trials participants did not know the response mapping before the target video (NoPrep condition). The resulting design included three different preparatory conditions (PrepC, PrepI and NoPrep) for each stimulus type (biological and non-biological) making a total of six conditions. The number of trials was balanced for each preparatory condition (32 trials per condition), as we were interested in preparatory period activity. However, because NoPrep trials are split into compatible (NoPrepC) and incompatible (NoPrepI) conditions upon presentation of the target video, a balanced design for preparatory conditions requires that the target condition is not perfectly balanced. For the target period, there are 32 trials each for PrepC and PrepI, but 16 trials each for NoPrepC and NoPrepI (figure 1*a*).

The target period was short (850 ms) to encourage preparation, and participants were instructed to prepare as much as possible for the upcoming response while waiting for the target movement. Trials were separated by a variable inter-stimulus interval (4, 6 or 8 s). Preparatory conditions were pseudo-randomized within a block that contained only one stimulus type (biological or non-biological) so that each preparatory condition followed each other with equal probability and compatible and incompatible targets followed one another with equal probability.

(c) Procedure

Immediately prior to scanning, subjects were familiarized with the task during a brief practice session. During each of four scanning runs that lasted 11 min, participants performed two blocks with imitative cues and two blocks with spatial cues. Participants held down two buttons with their index and middle fingers and responded by lifting a finger to release one of the buttons. Response times were recorded as the time of button release relative to the onset of movement in the video. Subjects were allowed a short break between runs. Each run was preceded by a reminder of the instructions.

(d) MRI data acquisition and preprocessing

Images were acquired on a Siemens 3T (Erlangen, Germany) Trio magnetic resonance imaging (MRI) scanner. Functional runs comprised 330 T2*-weighted echoplanar images (EPIs) (repetition time (TR) 2000 ms; echo time (TE) 28 ms; flip angle = 90°; 34 slices; slice thickness 4 mm; matrix 64 × 64; FOV 192 mm). To allow for T1 equilibrium, the first two volumes of each functional scan were automatically discarded before data collection began. Two sets of structural images were also acquired for registration of functional data: a T2-weighted matched-bandwidth high-resolution scan with the same slice prescription as the EPI (TR 5000 ms; TE 34 ms; flip angle = 90°; 34 slices; slice thickness 4 mm; matrix 128 × 128; FOV 192 mm); and a T1-weighted magnetization prepared rapid-acquisition gradient echo image (MPRAGE; TR 1900 ms; TE 2.26 ms; flip angle = 9°; 176 sagittal slices; slice thickness 1 mm; matrix 256 × 256; FOV 250 mm). Visual stimuli were timed and presented with Presentation software (Neurobehavioral Systems, Albany, CA, USA) through magnet-compatible liquid crystal display (LCD) goggles. Responses were recorded with a magnet-compatible response box (Current Designs, Philadelphia, PA, USA).

Image preprocessing and data analysis were performed with FSL v. 4.1.4 (Centre for Functional Magnetic Resonance Imaging of the Brain software library, www.fmrib.ox.ac.uk/fsl) [36]. Images were realigned to the middle volume to compensate for any head motion using MCFLIRT [37]. Images were then examined visually for gross motion artefacts that cannot be corrected for with simple realignment. When motion artefacts were detected, a nuisance regressor for each affected volume was included in the general linear model. In addition, one run for one subject was excluded for excessive motion (more than 10%

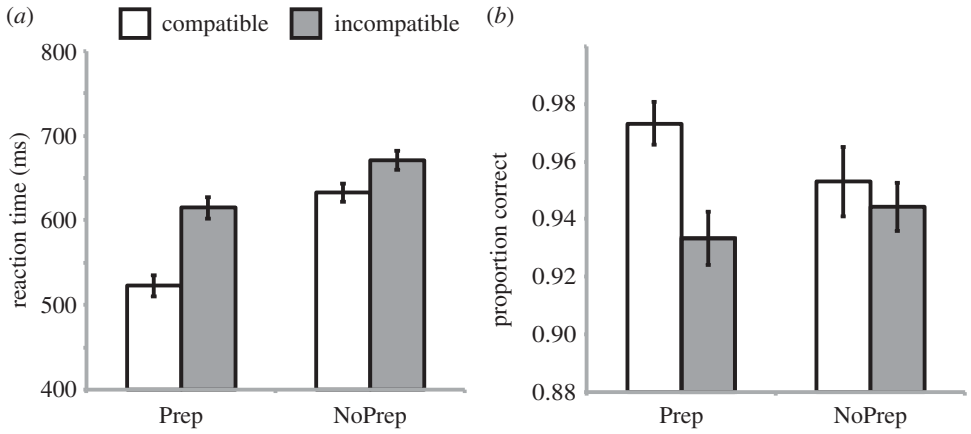

Figure 2. Behavioural data. Mean reaction time (*a*) and accuracy (*b*) for each target condition collapsed across stimulus type demonstrate reduction of compatibility effect for unknown mapping trials (NoPrep). Error bars represent standard error of the mean.

Table 1. Mean reaction time and per cent error (in parentheses) for each condition.

		compatibility condition		
stimulus	preparatory condition	C	I	compatibility effect (I-C)
biological	Prep	521 (2.9%)	611 (7.4%)	90 (4.5%)
	NoPrep	631 (4.9%)	673 (6.6%)	42 (1.8%)
non-biological	Prep	524 (2.5%)	619 (6.0%)	95 (3.5%)
	NoPrep	635 (4.5%)	669 (4.5%)	34 (0%)

volumes exhibiting motion artefacts). Data were temporally filtered with a high-pass filter cutoff of 100 s and spatially smoothed with a 8 mm full-width half-maximum Gaussian kernel in three dimensions.

(e) Statistical analysis

Statistical analyses were performed at the single subject level using a general linear model (GLM) with fMRI Expert Analysis Tool (FEAT, v. 5.98). Separate regressors modelled the preparatory and target periods of each condition, so that activity could be examined specifically in the preparatory period. This was made possible by jittering the length of the preparatory period between 2 and 10 s to reduce the correlation between preparatory and target period regressors. A separate regressor was included for each preparatory condition, resulting in six preparatory regressors (PrepC, PrepI and NoPrep for biological and non-biological stimuli). These regressors modelled the entire preparatory epoch. For target period regressors, compatible and incompatible responses following NoPrep periods were modelled separately, because the compatibility for these trials becomes evident once the mapping cue appears at the onset of the target video. As a result, there were eight regressors modelling target epochs, four each for biological and non-biological stimuli (PrepC Target; PrepI Target; NoPrepC Target and NoPrepI Target). Error trials were modelled separately with two regressors, one for the preparatory period and one for the target periods. Task regressors were convolved with a canonical double-gamma haemodynamic response function.

Contrasts specified were based on predictions formulated according to the dual route cognitive models described in the Introduction and are discussed in detail in the Results section. First-level contrast estimates were computed for each run and then registered to standard space (Montreal Neurological Institute, MNI) in three stages. The middle volume of each run of individual EPI data was registered first to the co-planar matched-bandwidth high-resolution T2-weighted image and subsequently, the co-planar volume was registered to the T1-weighted MPRAGE. Both of these steps were carried out using FLIRT (affine transformations: EPI to co-planar, 6 degrees of freedom; co-planar to MPRAGE, 6 degrees of freedom) [37,38]. Finally, registration of the MPRAGE to MNI space (FSL's MNI Avg152, T1 $2 \times 2 \times 2$ mm) was carried out with FLIRT (affine transformation, 12 degrees of freedom) and refined using FNIRT (nonlinear transformation). Contrast estimates for each subject were then computed treating each run as a fixed effect. Finally, a group-level analysis was performed to calculate a group mean for each contrast treating each subject as a random effect using FSL's FLAME (FMRIB's local analysis of mixed effects) stages 1 and 2 [39–41]. Except where noted, all images were thresholded at $z > 2.3$ ($p < 0.01$), corrected for multiple comparisons using cluster-based Gaussian random field theory controlling family-wise error across the whole-brain at $p < 0.05$. Discussion of preparatory activity is limited to regions in which preparatory activity is greater than baseline ($p < 0.05$ uncorrected) for at least one preparatory condition. This was accomplished by inclusively masking group maps after whole-brain statistical inference by each preparatory condition versus baseline.

3. Results

(a) Behavioural data

Mean accuracy and mean reaction time of correct responses were calculated in each condition for each subject. Trials with reaction time (RT) greater than 3 s.d. from the mean were excluded from analysis (0–2.8% of trials per subject). Separate three-way repeated measures ANOVA (cue type: biological,

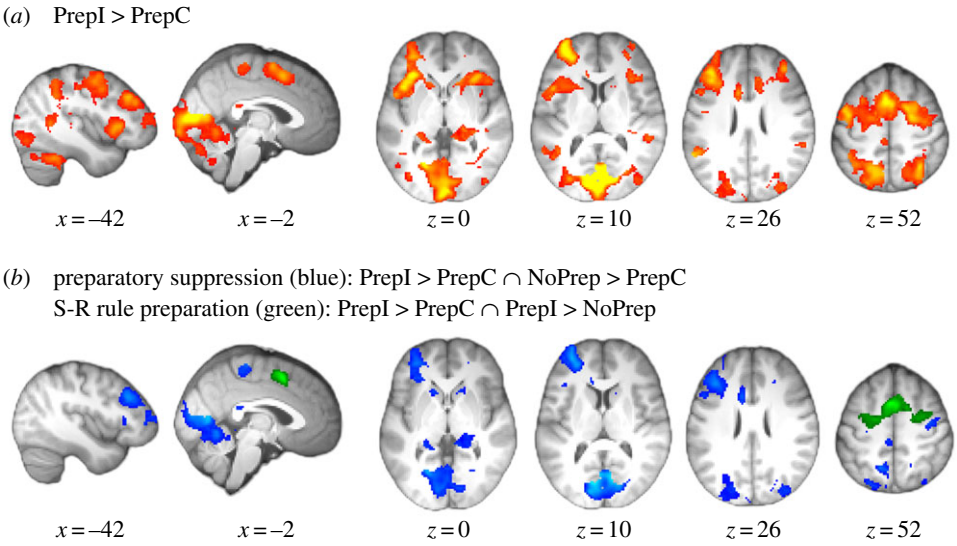

Figure 3. Preparatory period activity. (*a*) Compatibility effect (PrepI > PrepC) collapsed across cue types. (*b*) Neural correlates of preparatory suppression, which occurs during PrepI and NoPrep trials, is illustrated in blue as the conjunction between PrepI > PrepC and NoPrep > PrepC. Neural correlates of indirect route preparation, which occurs particularly during PrepI trials, are illustrated in green as the conjunction between PrepI > PrepC and PrepI > NoPrep.

non-biological × compatibility: compatible, incompatible × preparation: preparatory mapping cue, no preparatory mapping cue) were carried out for reaction time and accuracy using R statistical software (http://www.r-project.org).

Analysis of reaction times revealed significant main effects of preparation ($F_{1,31} = 174.4$, $p < 0.001$) and compatibility ($F_{1,31} = 205$, $p < 0.001$) owing to faster responses when preparatory information was available and for compatible trials, respectively (figure 2). In addition, consistent with previous reports and the preparatory suppression model [29–34], the two-way interaction between preparatory mapping information and compatibility was significant ($F_{1,31} = 82.2$, $p < 0.001$). This is due to greater compatibility effects for trials in which the mapping was known in advance (Prep trials) compared to when it is not known in advance (NoPrep trials). The three-way interaction was not significant, indicating that behaviour was similar for the two cue types (table 1).

For accuracy, there was a main effect of compatibility ($F_{1,31} = 16.9$, $p < 0.001$), reflecting better performance for compatible than incompatible trials. In addition, there was a significant preparation × compatibility interaction ($F_{1,31} = 7.2$, $p = 0.01$). Accuracy was similar for compatible and incompatible trials when preparatory mapping information was not available ($t_{31} = 0.92$, $p = 0.36$), consistent with the direct route suppression model in which the automatic route is suppressed and the two conditions use the same rule-based response route. By contrast, accuracy was better for compatible compared with incompatible trials ($t_{31} = 5.9$, $p < 0.001$) when preparatory information is available, consistent with the contribution of the automatic route to compatible, but not incompatible, responses.

(b) Functional imaging data

(i) Preparatory compatibility effect: preparatory suppression and rule-based route preparation

As outlined in the Introduction, dual route models propose that responding on incompatible trials involves (i) suppression of the automatic response route and (ii) implementation of the rule-based stimulus–response mapping. The fact that compatibility effects are reduced when preparatory mapping information is absent suggests suppression occurs during the preparatory period. In addition to suppression, it is also likely that preparation for incompatible trials involves preparation of the indirect route (rule-based stimulus–response mappings) since the information is available and the correct response must be selected via this route. In contrast to incompatible trials, preparatory suppression is absent on compatible trials as they benefit from automatic response activation. In addition, the ability to rely on the efficient automatic response route may reduce the degree of preparation of the rule-based stimulus–response mapping route. Following this framework, regions that are more active during preparation for PrepI than PrepC trials (PrepI > PrepC) should reflect neural correlates of preparatory suppression of the automatic response and/or preparation of the indirect route. Figure 3*a* illustrates reliable differences in BOLD signal located in multiple neural systems, including bilateral dorsal premotor cortex, posterior parietal cortex, anterior insula and the frontal pole for PrepI > PrepC (coordinates reported in table 2). In addition, visual areas, small clusters in supplementary motor cortex, and the bilateral caudate and hippocampus are more active during PrepI than PrepC.

The interaction between cue type and preparation ($\text{biological}_{\text{PrepI} > \text{PrepC}} > \text{non-biological}_{\text{PrepI} > \text{PrepC}}$) was not significant, and similar neural systems were observed when the compatibility effect was examined separately for each cue type (albeit, less robustly for spatial cues; see the electronic supplementary material, figure S1). Thus, we find no evidence for cue type specificity for either preparatory control or preparation of the stimulus–response rule.

To disentangle activity related to preparatory suppression from that related to preparation of the stimulus–response mapping, we introduced the NoPrep condition into the analysis. According to the cognitive model, suppression occurs not just during preparation for incompatible trials, but also when the mapping is unknown (NoPrep) as the automatic response would be incorrect half the time. Therefore, those regions related to suppression of the automatic response route in the PrepI > PrepC contrast should *also* be more active during NoPrep compared with PrepC trials. To identify these regions, we performed a conjunction [42] of the PrepI > PrepC and

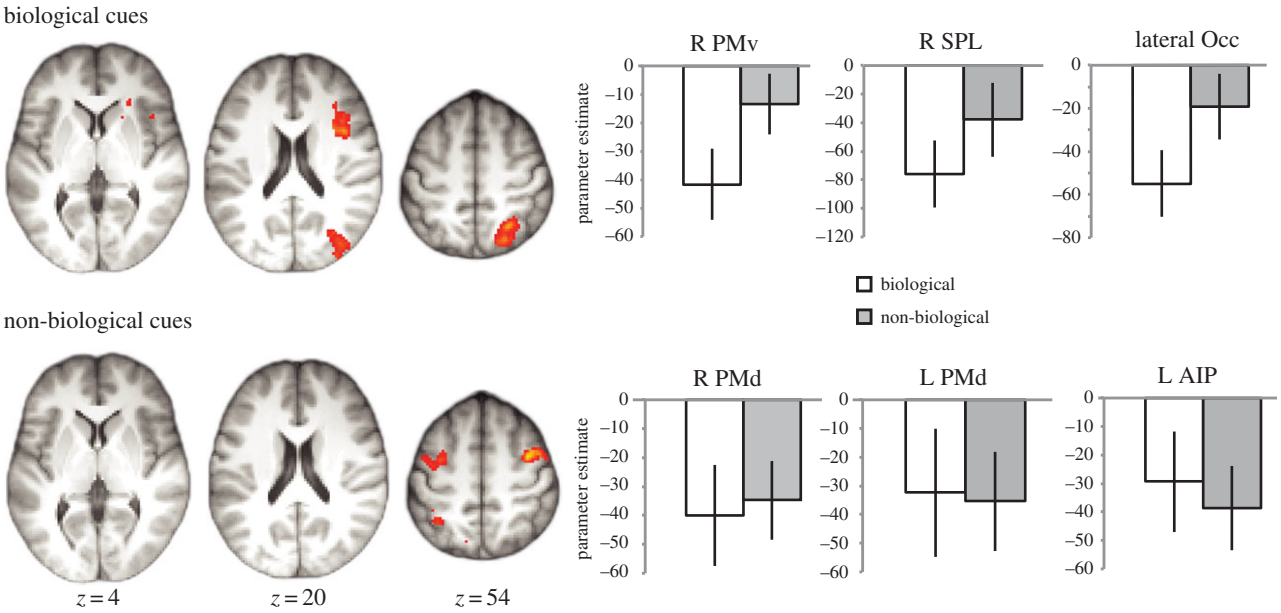

Figure 4. Neural correlates of automatic response activation. Brain maps depict regions in which preparatory period activation during PrepC trials correlates with subsequent response times on a trial-by-trial basis. In other words, higher activity in these regions predicts faster subsequent imitation (biological) or spatially compatible (non-biological) responses. Bar graphs depict correlation parameter estimates extracted from each cluster (negative relationship indicates higher activity associated with faster responses). R, right; L, left; PMv, ventral premotor cortex; SPL, superior parietal lobule; Occ, occipital; PMd, dorsal premotor cortex; AIP, anterior intraparietal sulcus.

NoPrep > PrepC contrasts. As shown in figure 3*b* (blue), activity following this pattern was observed in left dorsolateral prefrontal cortex (DLPFC), left frontal pole, bilateral posterior parietal cortex and visual cortex. In addition, small clusters are evident bilaterally in primary motor and caudate regions, as well as in the hippocampus.

In contrast to preparatory suppression of the automatic response tendency, regions in the PrepI > PrepC contrast that are involved in preparation of particular stimulus–response pairs should *not be active during NoPrep* trials, because the potential stimulus–response pairs are unknown. The alternative possibility—that all four potential mappings are prepared during NoPrep trials [43]—seems unlikely in this task given that the mappings are mutually exclusive (the same stimulus requires two different responses depending on the rule). Consistent with this view, the overlap of the contrasts PrepI > NoPrep and PrepI > PrepC (figure 3*b*, green) identified dorsal premotor cortex and supplementary motor area, regions previously implicated in stimulus–response associations in the conditional motor learning literature [44–48]. We also performed the PrepC > NoPrep contrast, as stimulus–response pairs could be prepared during preparation for compatible trials as well as during preparation for incompatible trials, but this yielded no significant effects. This is consistent with the hypothesis that preparation of the rule-based mapping route is minimal on compatible trials, when the more efficient automatic route activates the correct response as well (i.e. subjects can rely on the automatic route and therefore need not prepare the rule-based mapping).

There were no significant effects for the reverse compatibility effects (PrepC > PrepI) consistent with previous compatibility studies. The failure of this contrast to identify activity related to the automatic response route (which is theorized to be suppressed in PrepI compared to PrepC) may be because of insufficient sensitivity or the inability to distinguish inhibitory and excitatory inputs to a region using BOLD fMRI [49,50]. Therefore, we used an alternative strategy to identify activity related to the automatic response route.

(ii) Automatic response activation

To identify regions associated with automatic response activation (via the direct route), we used reaction time to locate regions in which greater preparatory activity is associated with faster subsequent reaction time on compatible trials. This method follows from the fact that reaction time benefit for PrepC trials is attributed to automatic response activation via the direct route. As shown in figure 4, patterns appear to be quite different for the two cue types. For biological cues, greater activity during preparation for the compatible response in the ventral premotor cortex is related to faster responses. By contrast, for non-biological cues, preparatory activity in the dorsal premotor cortex is related to faster subsequent compatible responses. The examination of the parameter estimates (figure 4, bar graphs) suggests some level of stimulus-specificity. While the parietal and dorsal premotor regions contribute similarly regardless of cue type, when cues are biological an additional fronto-parietal loop involving ventral premotor cortex also contributes to behaviour. The involvement of ventral premotor cortex for biological cues is particularly striking in the light of the fact that this region has been repeatedly associated with imitative behaviour and the mirror neuron system [51–53].

4. Discussion

Previous work suggests that inhibiting imitative response tendencies involves unique neural systems related to social cognition [19–21]. However, these studies focus on reactive control mechanisms, which are used after an action is observed. Here, we investigated the neural systems involved in control processes that are employed before an action is observed, to determine whether preparatory imitation control processes are

Table 2. Local maxima from significant clusters for direct route suppression, indirect route preparation and direct route activation.

		coordinates (MNI)			
contrast/region	L/R	*x*	*y*	*z*	*Z*-value
direct route suppression (PrepI > PrepC ∩ NoPrep > PrepC)					
frontal pole	L	−32	50	6	4.81
middle frontal gyrus	L	−40	28	30	3.95
anterior cingulate cortex	L	−12	28	20	3.64
dorsal premotor cortex	R	16	−6	66	2.95
	L	−24	−20	62	3.25
primary motor cortex	R	42	−8	48	3.17
	L	−2	−28	64	3.38
superior parietal lobule	L	−12	−58	56	3.83
	R	28	−68	46	3.34
lingual gyrus	L	−10	−50	−8	3.74
primary visual cortex	R	12	−74	8	4.69
	L	−12	−70	6	5.24
lateral occipital cortex	L	−20	−80	36	3.96
	R	28	−86	36	3.66
caudate	R	14	20	4	3.08
	L	−16	22	−6	3.42
hippocampus	R	20	−28	−6	3.29
	L	−22	−28	−4	3.14
cerebellum	R	12	−56	−14	3.80
	L	−22	−62	−22	2.91
indirect route (S-R rule) preparation (PrepI > PrepC ∩ PrepI > NoPrep)					
supplementary motor area	L	−4	10	50	4.25
	R	8	0	64	2.98
dorsal premotor cortex	L	−30	−4	68	4.16
	L	−16	−8	54	3.08
	R	26	−6	48	3.31
	R	48	0	42	3.21
direct route activation (PrepC activation correlated with RT)					
biological cues					
ventral premotor cortex/inferior frontal gyrus, pars opercularis	R	40	10	20	3.41
	R	34	8	28	3.19
superior parietal lobule/anterior intraparietal sulcus	R	26	−58	52	3.61
	R	32	−58	56	3.13
	R	24	−68	52	3.42
	R	32	−70	20	3.05
lateral occipital cortex/inferior parietal lobe	R	34	−70	26	3.18
	R	36	−78	16	3.60
non-biological cues					
dorsal premotor cortex	R	40	0	52	3.85
	R	40	−2	60	3.84
	L	−32	−4	54	3.0
	L	−38	−10	64	3.91
anterior intraparietal sulcus	L	−44	−38	36	3.22

similarly specialized. To do this, we drew from dual route models based in the spatial compatibility literature, which suggest that performing a stimulus-incompatible response involves (i) preparatory inhibition of automatic response activation and (ii) an increased reliance on stimulus–response mapping processes that apply a task rule. We were able to separate neural activity related to preparatory inhibition from that related to preparing the stimulus–response rule using a compatibility task with preparatory cueing. In addition, we determined the specificity of these processes by comparing biological and non-biological stimuli. Results suggest that the neural systems associated with preparatory inhibition of automatic response activation are similar for biological and non-biological stimuli. This contrasts with reactive imitation control processes which have previously been shown to rely on imitation-specific mechanisms [14–16].

In line with previous studies, participants were slower to respond with the incompatible compared with the compatible response, but this compatibility effect was substantially reduced when the stimulus–response mapping was not known before the stimulus [29–34]. The behaviour was similar for both biological and non-biological stimuli, which have both been shown to automatically activate the compatible response in previous studies [2,6,54,55]. As detailed in the Introduction, the pattern of behavioural results observed is attributed to preparatory suppression of the automatic response in preparation for incompatible and unknown mapping trials, because the automatic response is likely to be incorrect [31–33].

The neural correlates of preparatory suppression included left DLPFC, frontal pole, posterior parietal cortex and early visual regions. The pattern was similar regardless of whether cues depicted biological or non-biological stimuli, indicating that preparatory suppression mechanisms are not stimulus-specific: whether participants have to suppress the tendency to imitate or suppress the tendency to respond with the spatially compatible response, similar neural systems are involved. These findings contrast with previous studies of imitation control in several important ways. First, the neural systems involved in preparatory suppression of imitation are different from those observed in previous studies examining reactive inhibition of imitative tendencies. Reactive control is associated with activity in medial prefrontal cortex, the temporo-parietal junction, the frontal operculum and ventral premotor cortex [15,16,22,35]. In contrast, when the need for control is predictable and the imitative response tendency is strategically suppressed before an action is observed, we observe activity in the left DLPFC, left frontal pole, posterior parietal cortex and visual cortex.

Furthermore, there is a difference in the specificity of the preparatory imitation control mechanisms examined here and previously studied reactive control mechanisms. Neural correlates of reactive imitation control have been shown to be distinct from those involved in other conflict tasks, including both spatial compatibility [16] and Stroop tasks [15]. By contrast, we found no evidence of distinct mechanisms for biological and non-biological stimuli in this study, and the regions involved are similar to those reported to be involved in preparatory control across a variety of tasks [56–58]. Thus, while *reactive* imitative control relies on at least partly distinct mechanisms from other reactive control processes, the similar correlates for both biological and non-biological cues observed here suggests that *preparatory* control mechanisms are similar regardless of the stimulus that evokes the automatic response tendency.

Importantly, similar preparatory control mechanisms are unlikely to be attributable to identical processing of the biological and non-biological stimuli in this novel task. This is demonstrated by the observation that the neural underpinnings of automatic response activation are not identical for the two stimuli. Whereas the ventral premotor cortex contributes to compatible reaction times only when stimuli represent biological actions, activity in dorsal premotor cortex is related to faster responding regardless of the cue type. This finding fits well with premotor neurophysiology, as the ventral premotor cortex is one of the human homologues of area F5 where mirror neurons (which respond specifically to action observation) were discovered in monkeys [59,60]. Thus, while our data are consistent with the view that neural mirroring contributes specifically to automatic activation of imitative responses, preparatory control processes responsible for controlling automatic tendencies were not stimulus-specific.

The lack of stimulus-specificity is consistent with the associative learning theory of mirror neuron development. This theory argues that mirror neurons are just one component of a larger sensorimotor system that develops through a domain-general associative learning process. If one considers mirror neurons to represent a part of a larger general sensorimotor apparatus, it is not surprising that a singular non-specific control system develops to modulate the influence of this sensorimotor system on behaviour. However, we would also point out that a non-specific control mechanism does not preclude the alternative categorization of mirror neurons as a specialized system related to social cognition. Indeed, it is possible that a non-specific control system modulates early stages of processing, even if downstream processing stages are more specialized. Indeed, this latter hypothesis could explain why there is a difference in specialization for preparatory and reactive imitation control mechanisms. Reactive control occurs after the action is observed, and therefore after the specialized MNS is activated. As a result, reactive control uses a specific mechanism that modulates MNS activity. By contrast, when preparatory control processes are implemented, there has not been any action observation and therefore the mirror neuron system has not been activated. Without MNS activity, there may be no need for a specialized control system, and instead a general preparatory control mechanism is used.

While the precise details of this general preparatory control mechanism cannot be determined from the current results, we hypothesize that preparatory control involves top-down attentional biasing of early visual cortex. The DLPFC is activated during preparation across a range of conflict paradigms, including Stroop [56,61,62], flanker [58] and cross-modal [63] tasks and is proposed to be important for biasing processing in posterior regions depending on task demands. In line with previous work suggesting interactions between prefrontal and parietal regions in biasing of visual attention [64–66], we propose that activity observed in early visual cortex may be related to top-down suppression of visual input. The observation of involvement of early visual regions, which are not sensitive to biological content, is nicely consistent with the lack of stimulus-specificity observed. Suppression of early visual processing would lead to a reduction in the ability for any visual stimulus to

affect the motor system when the automatic visuo-motor response is likely to be incorrect (i.e. during preparation for incompatible and unknown mapping trials).

This may seem counterintuitive since activity is increased during PrepI and NoPrep trials, when suppression occurs. However, it can be difficult to predict the direction of BOLD signal change in a situation of top-down inhibition. The BOLD signal is correlated better with synaptic input than spiking activity [49], so when regional synaptic input is dissociated from neuronal spiking (as with inhibition) the BOLD signal may paradoxically increase [50]. Activity in line with this proposal was recently observed in a colour-word Stroop task [67], where participants responded to the ink colour of a written word that either matched the ink colour (the word 'RED' in red ink) or conflicted with the ink colour (the word 'BLUE' in red ink). On conflict trials, where the two visual features lead to conflicting responses, greater activity was observed not only in a functionally localized region responsible for processing the task-relevant feature—colour—but also in the visual word form area, a region involved in processing the irrelevant visual feature. More importantly, activity in the word form area was negatively correlated with reaction time, exactly as would be predicted if increased activity was related to more successful suppression of irrelevant feature processing.

In summary, our results indicate that preparatory control of imitation recruits different neural systems from previously studied reactive control mechanisms [15,35]. In addition, a singular non-specific preparatory suppression mechanism is involved in preparatory control regardless of the stimulus. Thus, in contrast to reactive imitation control, which relies on at least partially specialized mechanisms, [15,16] preparatory imitation control uses general mechanisms. Further work is required to test the hypothesis that preparatory control is implemented through top-down biasing of visual inputs to the motor system.

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