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A system in the human brain for predicting the actions of others

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The ability to attribute mental states to others, and therefore to predict others' behavior, is particularly advanced in humans. A controversial but untested idea is that this is achieved by simulating the other person's mental processes in one's own mind. If this is the case, then the same neural systems activated by a mental function should re-activate when one thinks about that function performed by another. Here, using functional magnetic resonance imaging (fMRI), we tested whether the neural processes involved in preparing one's own actions are also used for predicting the future actions of others. We provide compelling evidence that areas within the action control system of the human brain are indeed activated when predicting others' actions, but a different action sub-system is activated when preparing one's own actions.

Understanding others' intentions is an important ability that involves representing the mental states of others in one's own mind (forming a 'theory of mind'[1]). There are two competing views of how we do so. 'Simulation theory' suggests that we directly simulate others' cognitive processes by deploying the same cognitive mechanisms, whereas 'Theory theory' suggests that we use inferential and deductive processes that do not involve simulation[2–4]. The problem of understanding others' intentions can be translated into the more tangible problem of predicting others' actions. Identifying the neural mechanisms used to predict the actions of others may then enable us to distinguish between the two theories.

When primates perform or observe a meaningful action, such as grasping a piece of food, the same neurons in the ventral premotor cortex (PMv) that fire during the action also fire during observation[5,6]. This premotor region is specifically referred to as area F5 in macaque monkeys; Brodmann area (BA) 44/45 in humans, a part of Broca's area, appears to have very similar properties[7–9]. The PMv is essential for 'standard' visuomotor stimulus-response tasks, in which the object specifies the action (for example, the shape and orientation of a piece of food specifies the necessary hand posture to grasp it[10]). Hence, preparing direct stimulus-response actions and imitating or understanding these actions when performed by other agents may depend on a common neural mechanism[11]. Thus for direct stimulus-response actions, the activity in PMv can be expected from Simulation theory of mental state attribution[12] in which third person mental processes are simulated by the first person. But visuomotor associations can be arbitrary and predictive[13], and such 'non-standard' learned stimulus-response associations require the dorsal premotor cortex (PMd) rather than PMv[14–17]. Observing non-standard instructional cues can allow predictions of another person's future actions: we can predict the behavior of other car drivers by observing the shared arbitrary visual cues (traffic lights). We have therefore used an associative visuomotor task, in which activation would be expected in PMd and not in PMv, to test for activity predicted by the Simulation theory hypothesis.

Before scanning, we trained subjects in pairs to learn the association between visual instruction cues (simple colored shapes, **Fig. 1**) and subsequent visually triggered motor responses (finger movements) in a delayed non-standard visuomotor association task. Subjects then performed the same task in the MRI scanner, in the belief that their training partner continued to perform the task in an adjacent room. In the task, each instruction cue signaled subjects to either prepare a particular finger action or to wait for the trigger cue to specify the required action. Only if specified by the instruction cue could the action be planned in advance[17]. Hence, an important aspect of the event-related design was that we were able to localize preparatory activity time-locked to instruction cues, separated from subsequent action triggers by varying the interval between these and other trial components. Furthermore, the instruction cue color specified which agent should perform the action: the person being scanned (first person), their training partner (third person) or a computer (non-biological agent[18]). We thereby stringently controlled the stimuli used to instruct each subject, as well as the stimuli used by subjects to predict and monitor each other's actions. Simulation of the third person's preparation for action would be expected to activate the same motor areas as those involved in first-person preparation[17]. Mental state attribution through other mechanisms, as proposed by 'Theory theory,' would be expected to activate areas outside the motor system; areas most consistently activated when subjects evaluate the intentions of others include the paracingulate cortex and the superior temporal sulcus.

RESULTS

We first verified that right-hand finger movements evoked the expected hemodynamic responses within the motor system, using an

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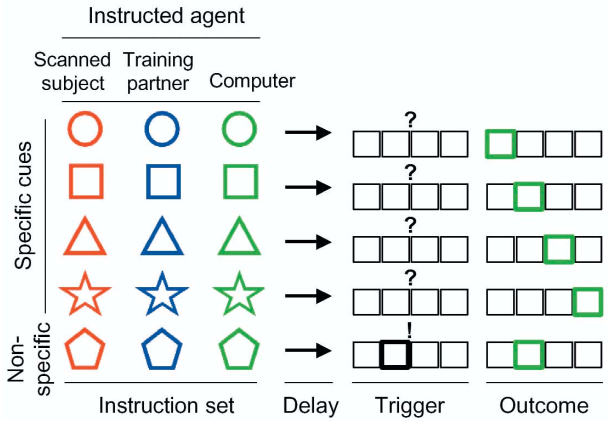

Figure 1 Trial structure and experimental design. Instruction cue color indicated which agent should perform the trial: scanned subject (first-person agent), training partner (third-person agent) or computer (non-agent control). Instruction cue shape indicated which action to perform. The cue-finger associations were the same for all agents. Twelve cues (top four rows) instructed a specific finger movement. Three cues (pentagons, bottom row) instructed the agent to wait until the trigger specified which key to press (the required movement could be any one of four, so subjects were not able to plan which key to press on the basis of the instruction cue). After the movement, the display screen also provided feedback, with the pressed key highlighted in green (correct) or red (incorrect).

event-related analysis of fMRI data that was time-locked to first-person trigger cues (**Supplementary Note**, **Supplementary Fig. 1** and **Supplementary Table 1** online). Next, to identify activity associated with preparing specific first-person actions, we contrasted specific versus nonspecific first-person instruction cues. We expected differential activity to be present in PMd[13,16,17,19], and this was the only area in the brain significantly activated in this contrast (**Fig. 2** and **Supplementary Table 2a**). Note that this activity was time-locked to the instruction cues, and not to the later performance of the actions. Thus, our design was both sensitive and selective.

We further validated our design by determining activity related to anticipation of third-person actions (the main effect of biological agency or 'intentional stance'[20,21]), comparing activity related to all third-person instruction cues (specific and nonspecific) with all computer instruction cues (**Fig. 3** and **Supplementary Table 2b**). In controlling for agency, it was important to exclude differences in attention between cues directed to the self and cues directed to others, so directly comparing third- and first-person conditions would be inappropriate (see Methods). Hence we compared third-person and computer conditions so that agency differed, but attention was externally directed for both.

We found differential agency-dependent activity in the superior frontal gyrus above the cingulate sulcus (**Fig. 3a**, paracingulate cortex, BA 9). There was also differential activity in the posterior superior temporal sulcus (**Fig. 3b**, STSp), ventral striatum and posterior cerebellar vermis (**Fig. 3c** and **Supplementary Table 2b**). Hence our experiment activates neural systems that have been often linked to mental state attribution[22–24].

As the final critical part of our design, we tested for activation differences between trials in which (i) the future actions of the third person or the computer were known and (ii) those trials in which they were not specified. From the simulation theory hypothesis, if areas required for one's own action preparation in our associative stimulus-response task are also used to predict others' actions, then PMd should be active following specific third-person instruction cues. We tested for this effect by looking for an interaction between agency and the specificity of instruction. We therefore contrasted the differential activation between specific and nonspecific instruction cues related to the third-person agent against the same differential for the computer (see **Fig. 4d,e** for comparison; **Fig. 2b** shows this differential activity just for the first-person trials).

Significant interactions were not found in PMd but elsewhere (**Fig. 4** and **Supplementary Table 2c**). First, there was significant activation of the same functional areas as activated in the main effect of agency (compare with **Fig. 3** and **Supplementary Table 2b**), including paracingulate cortex and STSp. Second, there was activation of a set of areas comprising a classical motor circuit and including dorsal prefrontal cortex (BA 9/46; **Fig. 4b**), a part of Broca's area (BA44/45, left ascending ramus of the sylvian fissure in pars triangularis; **Fig. 4c**) and right ipsilateral primary motor cortex (BA4). Event-related activation within these areas therefore depended both on the agency of the subject to perform the action, and on the specificity of that action.

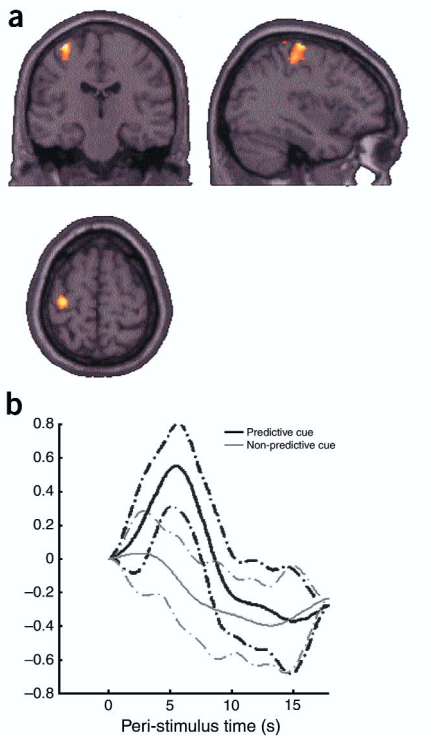

Figure 2 First-person preparation-related activity, contrasting predictive versus non-predictive first-person instruction cues. (**a**) The SPM{F} map is overlaid on the canonical T1 image from the MNI series, and shows activity in dorsal parts of the premotor cortex in the precentral gyrus. Coordinates (*x*, *y*, *z*): –34, –20, 68. (**b**) Best-fitting single-subject hemodynamic responses for predictive (black) and non-predictive (gray) instruction cues. Activity was sampled by randomly varying the interval from cue onset ($t = 0$) to scan onset. Over the course of the experiment, sufficient data was accumulated in the post-stimulus period to estimate the time course of the hemodynamic response. The solid traces depict a statistically estimated line of best fit through the data. The dotted-dashed lines depict the standard error of the mean (s.e.m.) of the data. Since the interval between cue onset and trigger onset was also varied, the activity was time-locked to instruction cues ($t = 0$) and not to the triggers.

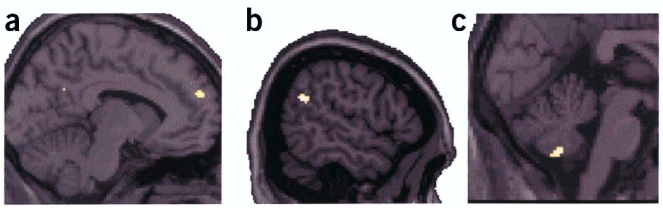

Figure 3 Main effect of biological agency: all third-person instruction cues compared with all computer-related instruction cues. Significant differential activity is shown in (**a**) paracingulate cortex, (**b**) posterior superior temporal sulcus (STSp) and (**c**) cerebellar vermis. All slices are sagittal sections. Coordinates (*x*, *y*, *z*): (**a**) –8, 56, 24 (**b**) 56, –54, 26 (**c**) 2, –60, –42.

DISCUSSION

Our results indicate that, in an associative stimulus-response task in which arbitrary visual cues specify future actions, neural activation during the preparation of specific, cued, first-person responses activated dorsal premotor cortex (PMd, **Fig. 2**). In contrast, activation related to the anticipation of the responses of another person performing the same task (the main effect of biological agency, **Fig. 3**) did not activate PMd, but instead activated paracingulate, superior temporal cortex and connected parts of the premotor cortex[25,26].

In fact, paracingulate cortex and superior temporal sulcus are the areas most consistently activated when subjects attribute mental states to others[22,27], but such studies differ from ours in important respects. First, in previous imaging studies, subjects were typically required to attribute complex mental states to others, in order to anticipate their actions[18,20]. In the present study, the rules of the task were straightforward, over-learned and performed without errors; hence the scanned subject unambiguously anticipated specific third-person actions. Second, we measured activity time-locked to the instruction cues, thus detecting time-specific mentalization by the first person of the other's state. Third, our design facilitates separation of first- from third-person action, as well as separates preparation from execution, whether by the first or third person. This allows direct comparison between activity evoked by the anticipation of first and third person actions, which has not been tested before. Finally, in some previous studies, the third party did not involve real agents. Stimuli were often cartoon characters or simple shapes with animate behavior[23,28], or spoken sentences necessitating the inference of mental states[23]. In our study, the third party was a real person known to the subject. Thus, despite the simple nature of our task, the activity that we saw in paracingulate cortex is consistent with mental state attribution.

We suggest that activation of the STSp (**Fig. 3b**), very close to an area activated by biological motion[29,30], may be due to predictive coding of imagined movement of the fingers. STS is strongly activated during imitation of actions[31], and may receive this predictive signal from the premotor cortex[31,32]. Although only the simple, static, visual cues and feedback stimuli in **Figure 1** were visible to the subject during scanning, subjects might have associated these stimuli with finger movements of their training partners remembered from the pre-scan training session, or with mental simulation of their own finger movements.

Interestingly, one recent report[33] localizes gray matter abnormalities in autistic individuals to specific regions within the paracingulate cortex, the inferior frontal gyrus and in the cerebellar vermis that were remarkably close to regions activated in the present study. Autistic individuals are impaired in their ability to attribute mental states to others[1,34]. This striking overlap raises the possibility that autistic individuals might have specific deficits related to anticipation of others' future actions in such associative visuomotor tasks. As far as we know, this possibility has not been tested.

Turning now to the interaction between agency and specificity, we suggest that the areas activated (**Fig. 4**) might reflect processing within two neural systems. The first may be a set of 'theory of mind' areas. These were in the same anatomical areas as activated in the main effect of agency (compare with **Fig. 3** and **Supplementary Table 2b**), including paracingulate cortex and STSp. Although they are not part of the classic motor circuit, both paracingulate and superior temporal cortex have robust and specific connections with dorsal and ventral sectors of the lateral premotor cortex[25,35]. Hence, mentalizing third-person action preparation could depend upon access to the motor system. Indeed, the second set of areas we found to be activated does comprise a classical motor circuit as well as dorsal prefrontal cortex (BA 9/46)—a region heavily interconnected with the premotor system in the macaque monkey[26,36]. Neurons in this area process action-related information at its most abstract level[37,38]. A part of Broca's area (BA44/45) was also activated in the present study; this area may be the homologue of

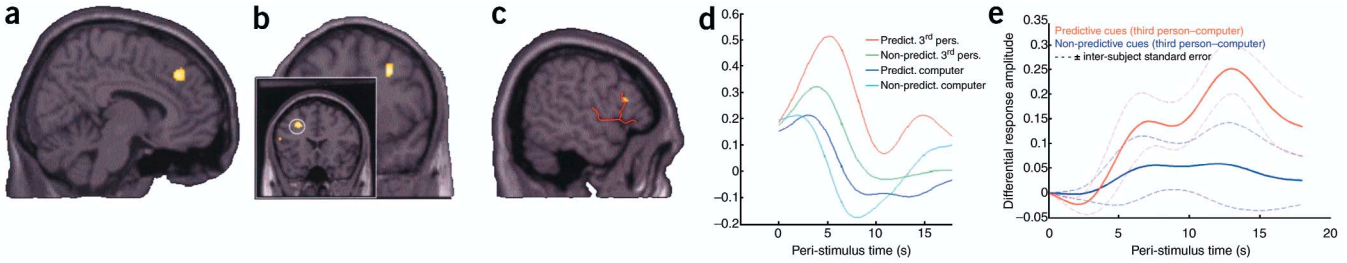

Figure 4 Activations for agency × predictability interaction. (**a**) Sagittal plane showing activity in paracingulate cortex (6, 34, 40), (**b**) coronal plane showing middle frontal gyrus (middle part, BA 9/46; background image, right hemisphere activation (30, 10, 42); inset, encircled left hemisphere activation (–26, 18, 38)). (**c**) Activity in Broca's area (BA 44/45 border delimited by ascending ramus of the sylvian fissure; horizontal and ascending components are depicted in red). (**d**) Instruction-related activity reflecting an interaction between the main effects of agency and predictability for action. Best-fitting single-subject hemodynamic responses evoked by the four types of cue are plotted for the paracingulate cortex (see panel **a** for location). The differential between responses to predictive (red) and non-predictive (dark blue) cues for the third person condition was greater than the differential between predictive (green) and non-predictive (cyan) cues for the computer condition. (**e**) Differential time courses for the same location averaged across subjects (solid lines show group mean, $n = 12$; dashed lines show inter-subject variability given by standard error). The differential between third-person and computer conditions was greater for predictive cues (red) than non-predictive cues (blue).

macaque premotor region F5, an intermediate level in the motor hierarchy receiving input from BA 9/46 (ref. 39). This area may be active for two possible reasons. First, predictive activity in advance of actions is typical of neurons in PMv[40] and, consistent with simulation theory[12], it is possible that this area was activated because the subjects needed to prepare or simulate their own actions, without execution, in order to predict the actions of their training partners. Second, Broca's area has also been reported to be activated by mental imagery of actions and during action imitation[8,11]; thus, the activation in our study could reflect the operation of the human mirror system. Mirror neurons may have fired in response to direct mental imagery of the training partners' unseen actions. Consistent with this view, mirror neurons are activated when monkeys make inferences about others' actions, even when the action itself is not observed[41]. In this situation it has been suggested that information flows from in STS, where there may be a visual representation of the imagined action, to the mirror system in Broca's area[31,32]. Both accounts are consistent with our data, but it is beyond the scope of the current study to distinguish between them. Finally, activation was also present in the lowest level of the cortical action system (right, ipsilateral, primary motor cortex, BA4), to which premotor areas such as F5 project[26]. Recent studies show that hand movements can also activate the ipsilateral primary motor cortex, particularly in complex motor tasks[42]. However, during mental simulation, low-level systems that implement action execution must be disengaged from the rest of the action planning system[43] and ipsilateral and contralateral primary motor cortex exert inhibitory influences on each other[44,45]. Hence we speculate that the observed activity in the ipsilateral primary motor cortex suppresses activity in the contralateral primary motor cortex, helping take the action implementation system 'off-line'.

In summary, we have shown that the human motor system is engaged when subjects use arbitrary visual instruction cues to prepare their own actions, and also when they use the same cues to predict the actions of other people. However, these two tasks engage separate sub-circuits within the premotor system. The preparation of one's own actions in this task preferentially activates PMd, as expected for mapping arbitrary cues to actions. In contrast, the prediction of other people's actions, using identical instruction cues, activates Broca's area (PMv) along with interconnected cortical prefrontal and primary motor areas. These results suggest that understanding the action-related mental states of others may not be explained by simulation theory alone. Although predicting the actions of others does involve the motor system (PMv), thus supporting simulation theory, activation of PMv instead of PMd suggests that pure simulation of the other person's mental state cannot be the mechanism used. Rather, it is likely that we understand the actions of others either by mental imagery of their actions or by the simulation of our own actions.

METHODS

Subjects. Subjects were 12 normal, healthy and right-handed volunteers (mean age, 24 years). Each gave written informed consent to participate; the study was approved by a local ethical committee.

Experimental design and task. We used a 2 × 3 factorial event-related design (**Fig. 1**). One factor manipulated subjects' ability to prepare and predict specific actions (specific versus nonspecific instructions[17]). Subjects were pretrained in pairs (first and third person), seated together in front of a computer, allowing prediction and direct observation of the other's actions. They learned a conditional, delayed-response motor task in which the shape of each of four arbitrary visual stimuli on the computer screen instructed them to perform one of four actions (four finger movements of the right hand, recorded by separate buttons) after a delayed trigger cue. Trigger cues were a visual representation of all four keys with a question mark (**Fig. 1**). A fifth instruction shape (pentagon) gave no specific information about the required action. On these trials, an exclamation mark rather than a question mark was used, and the target was highlighted (bottom row, **Fig. 1**). The second experimental factor directly specified which agent should perform the action. Thus, instruction cue color directed either the scanned subject (first person), the subject's partner (third person) or the computer. The paired associations between cue shape and response were identical for all agents. In each trial, the 300-ms instruction cue was followed, after a variable delay (0.1–9 s, uniformly distributed random intervals), by the trigger cue (300 ms) to allow separation of event-related responses. Immediately after each subject's response, the display indicated which key had been pressed, color-coded for correct (green) or incorrect (red). Subjects were thus able to monitor instruction cues, trigger cues and outcomes on all trials. Direct observation of their own or their partner's finger movements was possible during training but not during scanning. After training, one of each pair of subjects was scanned during the same task, while the other performed the task in the training room; the two subjects then exchanged places. Both were told that the training room computer was connected to the stimulus display computer in the scanner. However, in order to maintain experimental control during the scanning session, both 'third person' and computer response trials were actually computer-generated. Delays between triggers and computer-generated feedback stimuli were pseudorandom and their distribution matched to that recorded from a comparable group of subjects in a previous pilot study. Debriefing revealed that subjects did not detect any discrepancies in third-person or computer reaction times between prescan training and scanning.

We controlled for potential attentional confounds in two ways. First, comparisons between third-person and first-person conditions would be confounded by the direction of attention externally versus internally. Hence, we introduced the 'computer' condition as a control. In both third-person and computer conditions, attention was externally directed but only in the third-person condition was the subject evaluating actions of a human agent.

Second, to ensure that subjects did not simply 'switch off' during third-person or computer conditions, catch trials with error feedback incompatible with the agent's actions were introduced. To enforce attention across all trials types, subjects were instructed to monitor for any response errors made by their training partner or by the computer, for subsequent verbal report. The validity of the feedback image (**Fig. 1**) provided in each trial could only be evaluated by attending to all the events leading up to it. Thus, the scanned subject had to attend to the instruction cue for the other agents, and recognize that the feedback cue position and color were appropriate. By the end of training, their own performance was error-free and they accurately reported the presence of deliberately introduced incorrect feedback. During scanning, one erroneous trial was introduced for the each of first-person, third-person and computer responses. They successfully noted the occurrence of each catch trial in all conditions, suggesting that they attended every component of all trial types. Subjects failed to detect that the 'third-person' responses they observed during scanning were in fact computer generated.

Event timing and model definition. To optimally sample evoked hemodynamic responses (EHRs, the changes in BOLD signal evoked by task events), we introduced a random interval between scan onset and instruction cue onset, uniformly distributed from trial to trial over the range of 0–3 s (*i.e.*, one 'repetition time' (TR): the interval between the start of acquisition of one brain volume to the start of the next). Hence EHRs time-locked to the instruction cue were evenly sampled across repeated trials with an effective temporal sampling resolution much finer than one TR. To separate EHRs time-locked to the instruction cues from those time-locked to trigger cues, we also introduced random intervals between instructions and trigger cues uniformly distributed across three TRs (0.1–9 s). Thus, trigger cues were temporally uncorrelated with the preceding and the subsequent instruction cue, and were therefore modeled as independent event types. The six types of instruction cue (from the 2 × 3 factorial design, grouping all four specified finger responses together) and six associated trigger cues were each modeled as 12 separate event types. Our study did not require us to distinguish among different trigger-related

events, so EHRs related to detection of the visual trigger, motor responses and the visual outcome were modeled as a single compound event. Windowed Fourier basis functions were used to model EHRs. This strategy made minimal assumptions about the form of hemodynamic responses[17]. We monitored subject responses to identify trials in which motor responses were incorrect or late (RT > 1,000 ms), but the task was sufficiently over-trained and simple that subjects made no errors. The three trials in which false feedback was given were modeled as an additional covariate.

Functional imaging and analysis. For each subject, 750 T2*-weighted echo-planar images were acquired using a 3T Siemens Vision scanner with a GEM BEST sequence in a 37.5-min session (www.fmrib.ox.ac.uk). The field of view covered the whole brain: 256 × 256 × 125 mm, 64 × 64 × 24 voxels; TR = 3 s, TE = 30 ms, flip angle = 90°. High-resolution T1-weighted structural images were also acquired.

Scans were pre-processed using SPM99 (www.fil.ion.ucl.ac.uk/spm) by spatial realignment to the first scan[46], normalization to the ICBM template using both linear affine transformations and non-linear transformations[47]. Lastly, a Gaussian kernel of 10 mm was applied to spatially smooth the images.

Statistical analysis. A general linear model (GLM) was constructed in SPM99. Each event type was used to construct a series of regressors by convolution of event time delta functions with a Fourier set of five harmonic functions (2 sine, 2 cosine and 1 envelope function, 18-s post-stimulus time window). This strategy has been successfully used to model potentially complex hemodynamic activity without making stringent prior assumptions about its amplitude and time course profile[17,48]. All 65 regressors from each of the 12 subjects were incorporated into a GLM. Before the study, event times were carefully checked so that they resulted in an estimable GLM in which the independence (or 'rank') of the six event types was preserved. The degree of rank deficiency was assessed by examining the correlations among all regressors and found to be very low. After parameter estimation, F-contrasts were applied in the context of a fixed-effects group analysis to specify comparisons between the trial types. The resulting SPM{F}maps identified voxels in which linear combinations of the five basis functions resulted in estimated responses that were significantly different in the conditions of interest[17].

Localization. Anatomical details of significant signal changes were obtained by superimposing the SPM{F} maps on the T1 canonical MNI (Montreal Neurological Institute) template image. Results were checked against structural images of each subject. We used one atlas[49] as a general neuroanatomical reference, and another[50] for localization within the cerebellum. We used the designations of Brodmann as a rough guide to the location of cytoarchitectonic areas of the cortex.

Note: Supplementary information is available on the Nature Neuroscience website.

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COMPETING INTERESTS STATEMENT

The authors declare that they have no competing financial interests.

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Supplementary Information

A System in the Human Brain for Predicting the Actions of Others.

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Supplementary Results:

The event related analysis and experimental design were verified by testing for expected patterns of activation in the motor system due to instructed finger movements. We therefore compared trigger-related activation for all first person trials versus trigger-related activation for third person and computer-response trials. As expected, differential activity was found in most of the motor system, particularly robust in the left primary motor cortex (Figure S1, Table S1). Apart from the motor activity, there was also significant trigger-related activity in the visual cortex despite the same visual cues in all three trigger conditions (1st versus 3rd person and computer). This is likely due to increased attention to the trigger cue when 1st person actions are expected [1].

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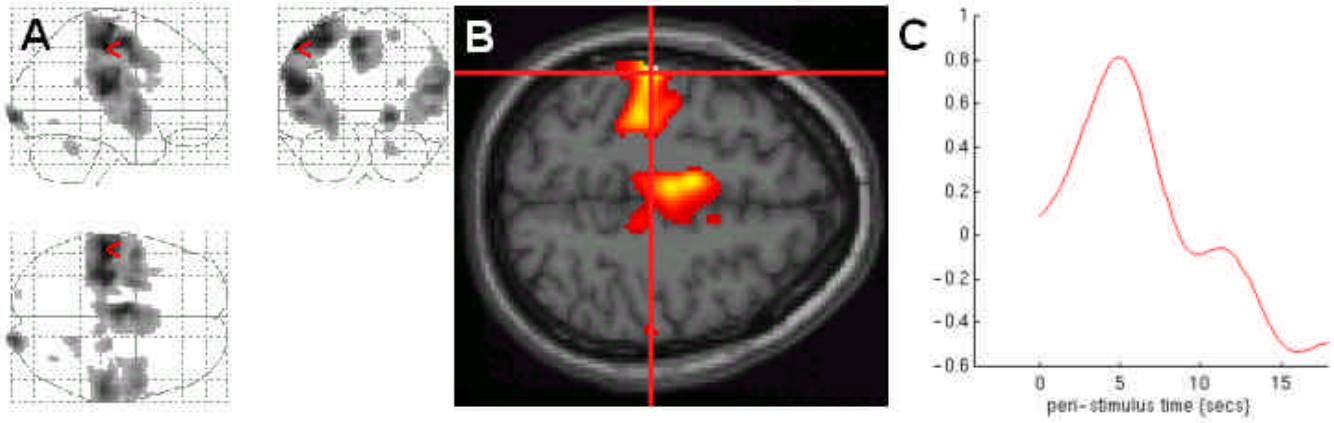
Supplementary Figure 1. Main effect of first person movements. Trigger-related activity for first person (visual trigger stimulus plus subject action) compared with trigger-related activity for 3rd person and computer (only visual trigger, no movement). **(A)** SPM{F} map for F-contrast displayed as a maximum intensity projection in a 'glass brain' Activity is evident in the motor system. **(B)** The same SPM{F} map is superimposed on the canonical brain of the MNI series (axial section, anterior = right). The voxel with maximum Z-score in the primary motor cortex is marked by the red cross-hairs. Activity is also seen medially in the supplementary motor area (SMA). **(C)** Best-fitting haemodynamic response from the voxel in **(B)** time-locked to the first-person trigger cue.



Supplementary Table 1: Activity time-locked to first person trigger cues (responses to visual trigger and outcome presentations plus subjects' finger movement motor responses) compared with third person and computer trigger cues (responses to visual trigger cue and outcome). Differential activity was seen throughout the motor system.

Area	Lateral -ity	F	equiv. Z	Coordinates: {x,y,z} mm		
Trigger-related activity (first person vs. [third person, computer])						
Cerebral cortex						
Precentral gyrus (primary sensorimotor cortex)	L	16.51	7.04	-54	-24	52
	L	14.28	6.46	-30	-24	58
	R	7.37	4.24	22	-22	60
	R	6.79	4.00	60	-12	42
	L	6.52	3.89	-20	-16	74
Inferior frontal gyrus (ventral premotor cortex)	L	10.9	5.47	-50	0	6
	R	9.28	4.94	56	0	8
Subcentral gyrus	R	8.00	4.48	60	-6	22
Supramarginal gyrus	R	11.95	5.79	60	-26	18
Insular cortex	L	9.55	5.03	-38	0	8
	R	8.21	4.55	42	0	8
	R	7.04	4.10	38	2	-6
Medial superior frontal gyrus (supplementary motor area, SMA)	L	14.05	6.40	-6	-10	56
Cingulate sulcus (cingulate motor areas, CMAa)	L	8.67	4.72	-2	8	38
Cingulate sulcus (cingulate motor areas, CMAd and CMAv)	L	11.46	5.65	-4	-2	44
Postcentral sulcus (posterior parietal cortex)	R	9.48	5.01	60	-18	30
Posterior cingulate sulcus	R	6.63	3.93	10	-28	50
Posterior subcentral gyrus (secondary somatosensory cortex)	L	14.59	6.54	-50	-22	20
Inferior occipital gyrus (primary visual cortex)	R	12.17	5.86	20	-98	-4
Basal ganglia						
Putamen	L	10.06	5.20	-28	-2	-8
	L	7.52	4.29	-32	-20	0
Cerebellum						
Cerebellar cortex (lobule HVI)	R	6.80	4.00	20	-52	-30

Supplementary Table 2: Table of results for activity time-locked to instruction cues (thresholded at p<0.05 corrected, except for Table S2 B where voxels were only present at a threshold of p<0.001 uncorrected).

Areas	*Laterality*	*F*	equiv. Z	Coordinates {x, y, z} mm
A: *Predictive vs. Non-predictive 1st Person Instruction cues*				
Dorsal premotor cortex (BA 6)	L	9.69	5.05	-34 –20 68
B: *Cue-related activity: Main effect of agency (3rd Person vs. Computer)*				
Cerebral cortex				
Superior frontal sulcus	R	4.99	3.20	22 38 30
Paracingulate cortex (medial superior frontal gyrus)	L	5.29	3.35	-8 56 24
Superior Temporal Sulcus (posterior)	R	5.45	3.42	56 -54 26
Parieto-occipital sulcus	R	4.90	3.16	10 -52 28
Parieto-occipital sulcus	L	4.85	3.13	-10 -56 28
Basal Ganglia				
Putamen	L	5.47	3.43	-20 10 -12
Cerebellum				
Posterior cerebellar vermis (lobule VIIIB)	R	5.49	3.44	2 -60 -42
C: *Predictability x agency interaction*				
Middle frontal gyrus (BA 9/46)	R	23.70	4.72	30 10 42
Middle frontal gyrus (BA 9/46)	L	23.66	4.72	-26 18 38
Superior frontal gyrus (lateral prefrontal corex)	L	21.23	4.45	-18 38 42
Precentral gyrus (sensorimotor cortex)	R	19.61	4.27	42 -26 42
Inferior frontal gyrus (BA44 / 45)	L	17.06	3.96	-54 20 16
Superior Temporal Sulcus (posterior)	L	16.53	3.90	-60 -46 28
Paracingulate cortex (medial superior frontal gyrus)	R	22.46	4.59	6 34 40