The Angular Gyrus Computes Action Awareness Representations

Chlöé Farrer[1], Scott H. Frey[2], John D. Van Horn[3], Eugene Tunik[4], David Turk[5], Souheil Inati[6] and Scott T. Grafton[7]

[1]Centre de Neuroscience Cognitive, Université Claude Bernard Lyon 1, CNRS, 69675 Lyon, France, [2]Lewis Center for Neuroimaging & Psychology Department University of Oregon, Eugene, OR 97403-1228, USA, [3]Dartmouth Brain Imaging Center, Center for Cognitive Neuroscience, Dartmouth College, Hanover, NH 03755-3569, USA, [4]Department of Physical Therapy, New York University, New York, NY 10010, USA, [5]Social Cognition Laboratory, University of Aberdeen, Aberdeen, AB24 3FX, Scotland, [6]Center for Neural Science and Department of Psychology, New York University, New York, NY 10003, USA, [7]Department of Psychology, University of California, Santa Barbara, CA 93106-9660, USA

Involvement of the right inferior parietal area in action awareness was investigated while taking into account differences in the conscious experiences of one's own actions; especially, the awareness that an intended action is consistent with movement consequences and the awareness of the authorship of the action (i.e., the sense of agency). We hypothesized that these experiences are both associated with processes implemented in inferior parietal cortex, specifically, right angular gyrus (Ag). Two blood-oxygenation-level–dependent functional magnetic resonance imaging studies employed a novel delayed visual feedback technique to distinguish the neural correlates of these 2 forms of action awareness. We showed that right Ag is associated with both awareness of discrepancy between intended and movement consequences and awareness of action authorship. We propose that this region is involved in higher-order aspects of motor control that allows one to consciously access different aspects of one's own actions. Specifically, this region processes discrepancies between intended action and movement consequences in such a way that these will be consciously detected by the subject. This joint processing is at the core of the various experiences one uses to interpret an action.

Keywords: action, agency, fMRI, inferior parietal lobule, internal model

Introduction

Attending to one's own or another's actions gives rise to different forms of conscious experience. For instance, while playing a video game with a partner, I can focus on my own performance and evaluate if my intended action is consistent with both my actual performance as well as visual and proprioceptive feedback. This awareness of action performance does not call into question the source of the action (Jeannerod 2003). By contrast, I can also be aware of my inability to control another's actions. This sense of agency requires generating an inference of the perceived action's author. Empirical evidence suggests that these 2 states of awareness arise from 2 distinct levels of representation within the nervous system (Jeannerod 2003).

Investigations into the components of motor control involved in awareness of action suggest that awareness of both one's own and another's action may rely on predictive models that may be implemented by a mechanism analogous to forward internal models in motor control (Frith et al. 2000; Haggard 2005). These models represent aspects of one's own body and the external world (Wolpert et al. 1995) allowing the central nervous system

to predict the sensory consequences of a movement before its completion (Ito 1970; Miall et al. 1993; Wolpert et al. 1995; Jordan 1996). For self-generated movements, such predictions are thought to be derived from a copy of the motor command (efference copy) (Von Helmholtz 1886; Sperry 1950; Holst and Von Mittelstaedt 1950) and can be compared with real sensory feedback signals arising as a consequence of the movement itself and give rise to an error signal. Forward models are advantageous in that they enable rapid automatic corrections unconstrained by inherent delays in sensory feedback processing (Desmurget and Grafton 2000). Efferent information also plays a major role in self-recognition (Tsakiris et al. 2005). For example, involvement of forward models in awareness of action is suggested by studies in which sensory feedback, resulting from actors' movements, is manipulated to introduce erroneous discrepancies between the intended action and its perceived consequences. Discrepancies above a certain threshold induce an awareness in subjects that control of their action has somehow failed (Fourneret and Jeannerod 1998; Blakemore et al. 1999; Franck et al. 2001; Farrer et al. 2003b) but they still consider themselves as the author of the action. However, other studies show that a mismatch between intention and perceived consequences can also induce a perturbed sense of agency, with subjects no longer experiencing themselves as the author of the action (Sato and Yasuda 2003; Wegner et al. 2003). This experience is similar to a well-described psychiatric phenomenon wherein patients experience that another person is controlling their own actions (Schneider 1955). As in the video game example, a discrepancy between one's own intentions and the perceived consequences of an action can thus lead either to an awareness that one's own actions are failing, or, alternatively, to a perturbed sense of control or agency. How might a forward internal model instantiate these very different forms of action awareness in the nervous system? One possibility is that these distinct conscious experiences are associated with recruitment of common brain areas. Although, the neural structure(s) that construct the representations responsible for these experiences are presently unknown, awareness of action has been associated with parietal cortex (Frith et al. 2000; Sirigu et al. 2004), specifically, right angular gyrus (Ag). Increased activity in this area is observed when subjects become aware of not being in control of an action (Farrer and Frith 2002). Likewise, right Ag activity correlates with the magnitude of the discrepancy between intended and actual consequences of movement (Farrer et al. 2003a). These studies were, however, unable to

disambiguate between brain activity related to awareness of action discrepancy and awareness of action authorship. Although the same process of comparison between intended and actual consequences of movement is involved in these 2 aspects of action awareness, it is unclear whether these aspects recruit different inferior parietal areas.

To clarify involvement of the Ag in action awareness, 2 functional magnetic resonance imaging (fMRI) studies were undertaken in which we manipulated 1) the awareness of one's own movement being consistent with efference copy and feedback information and 2) the experience of authoring or not authoring the action (i.e., agency). Importantly, for both experiments the visual stimuli remained constant, and only the subjective experiences related to the subjects' actions were modified. In study 1, delays in visual feedback of manual actions were introduced to manipulate parametrically the relationship between predicted and actual sensory consequences of prehensile movements. This allowed us to disambiguate neural activity when subjects are aware versus unaware of these discrepancies with no bearing on authorship. In study 2 we introduced uncertainty regarding whether actions performed under conditions of delayed feedback were those of the subjects or another individual. This allowed us to determine neural correlates of action authorship.

Material and Methods of Study 1: Awareness of Action Discrepancy

Subjects

Fifteen healthy subjects (20.7 ± 5.7 years: 4 females, 11 males) identified as right hand dominant according to the Edinburgh Handedness Inventory (Oldfield 1971) participated in this experiment. None of the participants had a history of psychiatric or neurological disease and written informed consent was obtained from all the subjects. The study conforms to the Code of Ethics of the World Medical Association (Declaration of Helsinki) and the protocol experiment was approved by the Committee for the Protection of Human Subjects at Dartmouth College.

Task and Procedure

A manual peg removal task was performed with visual feedback delayed by 0 (unperturbed), 50, 100, 150, 200, or 300 or 400 ms. Subjects held with their left hand a black grid (17.2 cm width, 11.7 cm length) with 33 holes on which 25 white golf pegs were inserted randomly. The placements differed for each block within a run and across sessions so that the subjects could not memorize the locations of the pegs. Eight different grids were used per run. The image of the grid held by the subjects was filmed with an infrared camera located in front of the scanner; this image was sent outside the scanner room to a wide bandwidth direct Audio/Video delay unit that buffers video for up to 1 s in approximately. 16-ms steps. The image was then projected to the subjects via a LCD projector to a rear projection screen black at the head of the bore (Fig. 1). The subjects were required to view the image of the grid and to remove as many pegs as they could in 20 s and drop them into a box on their right side. The task required subjects to make very accurate movements that necessitate visual feedback to perform. The arm movements were minimized by restraints and support of the upper arm, neck, and head with cushions.

The study involved 7 different conditions that correspond to an absence of delay (0 ms) or a presence of a delay (50, 100, 150, 200, 300, or 400 ms). We used a block design with 8 different blocks of 20 s each, where the subjects were required to remove the pegs, alternating with rest conditions during which subjects did nothing and saw nothing. Each run involved the 7 different delays in a counterbalanced order and began with an additional 0-ms delay block. The order of presentation of the experimental runs was reversed between subjects. Immediately after each movement block ended, subjects gestured to indicate whether they perceived delays in the visual images of their movements:
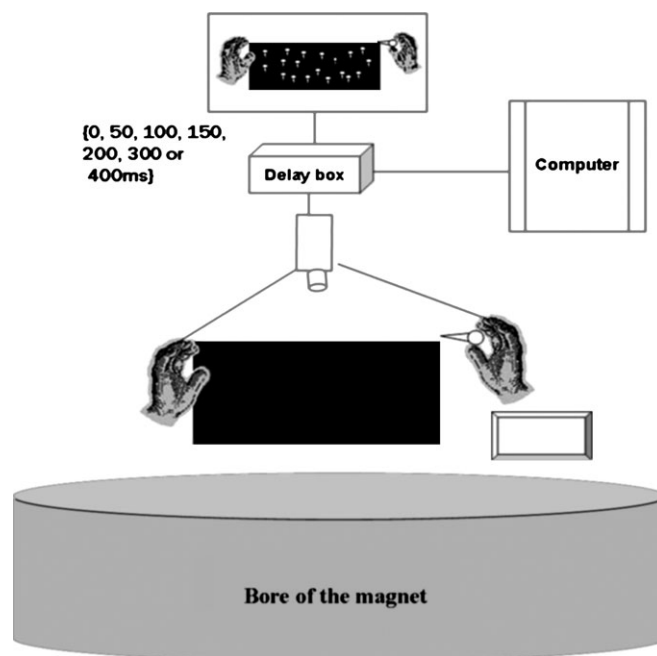

Figure 1. Experimental setup for the first study (awareness of action discrepancy). Subjects hold with their left hand a black grid on which 25 white golf pegs were inserted randomly. The image of the grid held by the subjects was filmed with an infrared camera located in front of the scanner; this image was sent outside the scanner room to a wide bandwidth direct Audio/Video delay unit that delays video for up to 1 s. The image was then projected to the subjects via a LCD projector to a rear projection screen at the head of the bore. The image on the screen was reversed so that the subjects could see their right hand on the right side of the screen. The subjects were required to view the image of the grid and to remove as many pegs as they could, and drop them in a box on their right side.

palm face down for a No response, and thumb up for a Yes response. The number of pegs removed during each block was recorded by the experimenter as a measure of performance.

To identify any area whose responses were sensitive to the number of grasping actions performed, we implemented a separate control run. This consisted of 8, 20-s control blocks alternating with rest. In each block subjects were required to remove all the pegs from the grid and no temporal delays were applied. The number of pegs in each block varied between subjects and was identical to the number removed during the task condition.

Functional Imaging

A 1.5-T GE scanner with a standard birdcage head coil was used for functional imaging. Head movements were minimized by use of a foam pillow and padding. Prior each functional run, 4 images were acquired and discarded to allow for longitudinal magnetization to approach equilibrium. For each functional run an ultrafast echo planar gradient echo imaging (EPI) sequence sensitive to blood-oxygenation-level–dependent (BOLD) contrast was used to acquire 25 slices per time repetition (TR) (4.5 mm thickness, 1 mm gap, in-plane resolution, 3.125×3.125 mm). The following parameters were used: TR = 2500 ms, time echo (TE) = 35 ms, flip angle = 90°. A coplanar, T_1-weighted, axial fast spin echo sequence was used to acquire 25 contiguous slices (4.5 mm slice thickness with 1.0 mm gap) coplanar with the EPI images: TE = Min full, TR = 650 ms, Echo Train = 2, field of view = 24 cm. A 124-slice, high-resolution ($0.94 \times 0.94 \times 1.2$ mm), whole-brain, T_1-weighted structural image was also acquired using a standard GE SPGR 3-D sequence.

Data Analyses

Image analyses and statistical analyses were performed using SPM99 (http://www.fil.ion.ucl.ac.uk/spm/software/spm99). For each subject, all functional volumes were realigned to the first volume to correct for interscan movement. Functional and structural images were coregistered and transformed (Friston et al. 1995a) into a standardized,

stereotaxic space (Montreal Neurological Institute [MNI] template) (Evans et al. 1994). Functional data were then smoothed with an 8-mm full width half maximum (FWHM), isotropic Gaussian kernel and temporally filtered with a cutoff period of 80 s. Three statistical analyses of the fMRI data were performed.

1. *Movement quantity-related activity.* The main purpose of the analysis of the control run was to detect brain areas that showed increased activity as a function of the number of visually guided grasping movements (i.e., pegs removed) by each subject.
2. *Movement-related activity.* The experimental blocks across all delays were grouped into one effect of interest. A simple contrast (experimental blocks—rest blocks) of the parameters estimates pertaining to each effect was then created for each subject.
3. *Delay-detection activity.* The subjects' responses in the detection of the delay allowed us to assess differences between trials where subjects perceived the delay and trials where they did not perceive a delay. Only the trials with 100- and 150-ms delays were used as they bracket the threshold of awareness (see Results part), for which subjects detected (DelayDet) or failed to detect (NodelayDet) delays. This allowed us to assess brain activity associated with the detection of the delay independently of the delay value.

In all of these analyses, the 6 realignment parameters resulting from the motion correction were modeled as effects of noninterest and the blocks were modeled by convolving a box-car function with a standard hemodynamic response function (hrf) (Friston et al. 1995b). Second-level group analyses were then performed for each analysis using a random effects model (Holmes and Friston 1998). The contrast images were entered into a one-tailed *t*-test. The set of *t*-values thus obtained constituted a statistical parametric map (SPM{*T*}). These SPM{*T*}s for each effect modeled were transformed into SPM{*Z*}s. Areas of activation were characterized in terms of their peak heights (*Z*-value maxima) with their positions specified in standardized coordinates (*x, y* and *z*). We report significant activations for the basic motor task (all delays vs. rest) corrected for multiple comparisons (*P* < 0.05). For effects of delay, where we had a priori predictions of anatomic localization, we report activations above a threshold corresponding to (*P* < 0.001, uncorrected for multiple comparisons), $Z \geq 2.80$ and minimal cluster size ($K_E \geq 10$).

Regions of Interest Analysis

The locations of peak activations in the left and right Ag were identified as regions of interests (ROIs) from the statistical parametric map resulting from the random effects comparisons of DelayDet versus NodelayDet conditions, *P* < 0.001 uncorrected for multiple comparisons, $K_E \geq 10$, minimum separation 8 mm. For each subject, BOLD response data from stimulation onset (SO) to 10-s post so were extracted from all voxels located within 8-mm radius spheres centered on peak locations using the MarsBar toolbox (http://marsbar.source-forge.net/faq.html). Repeated-measures analyses of variance (ANOVAs) were then performed on individual subject's time averaged data, pooled across all voxels within a given ROI.

Brain Activity Localization

Brain activity localization was identified by superimposing the SPM{*T*} maps onto the T_1-canonical MNI template image. The atlas of neuroanatomy by Duvernoy (1992) was used as a neuroanatomical reference. The atlas of Schmahmann et al. (2000) was used for the localizations within the cerebellum. Brodmann areas were identified after a conversion of MNI coordinates to Talairach coordinates (Talairach and Tournoux 1988) [$X' = 0.88X - 0.8$; $Y' = 0.97Y - 3.32$; $Z' = 0.05Y + 0.88Z - 0.44$] (http://www.mrc-cbu.cam.ac.uk/Imaging/Common/mnispace.shtml). MNI coordinates are used in the tables.

Results of Study 1: Awareness of Action Discrepancy

Behavioral Data

As expected, the accuracy of delay detection decreased as a function of delay magnitude. The mean threshold of awareness, defined as the delay value at which subjects correctly
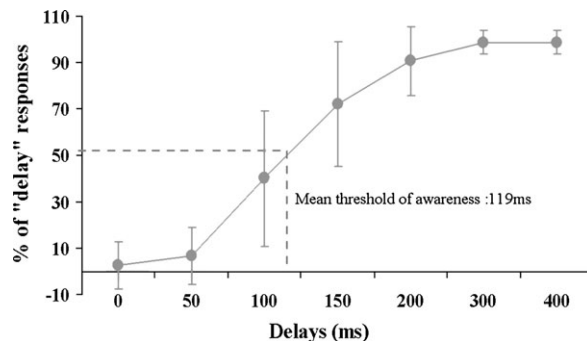

Figure 2. Percentage of "delay" responses (subjects declared there was a noticeable delay). The mean threshold of awareness, defined as the delay value at which subjects correctly detected the manipulation on 50% of blocks, occurred at 119 ms.

detected the manipulation on 50% of blocks, occurred between 100 and 150 ms (Fig. 2).

Consistent with the increasing demands on visuomotor control, the number of pegs removed decreased significantly as a function of increased delays in visual feedback ($F_{6,84} = 19.1$, *P* < 0.0001). On average, approximately 1.74 fewer pegs were removed for the maximal delay (400 ms) versus baseline (0 ms). Relative to no visual delay (0 ms), this difference reached significance at 100 ms ($t = -4.96$, df = 14, *P* = 0.00021). However, there was no effect of the detection of the delay on performance in those blocks with delay values near the threshold of awareness (i.e., 100 and 150 ms). The subjects removed the same number of pegs when the delays were detected and when they were undetected (*P* > 0.05).

Imaging Data

Movement-Related Activity

Contrasting all experimental conditions with the rest condition revealed significant activation in an occipito-parieto-frontal network typically associated with visually guided action (Grafton et al. 1996) (*P* < 0.05 corrected for multiple comparisons). A separate control run verified no significant changes in brain activity associated with the number of pegs removed even at a low threshold (*P* < 0.05 uncorrected).

Awareness of Action Discrepancy (Detection of Delay)

Brain areas involved in detecting a discrepancy were determined by contrasting blocks on which subjects perceived versus failed to detect the delay. These blocks were limited to trials with magnitudes near the threshold of awareness (i.e., 100 and 150 ms), for which subjects detected (DelayDet) or failed to detect (NodelayDet) delays. Detection of the delay independent of the delay magnitude [(DelayDet100 + DelayDet150) – (NodelayDet100 + NodelayDet150)] revealed activation in the right (Fig. 3) and the left inferior parietal lobule (IPL). These activations were localized in the Ag in both hemispheres. Individual analyses restricted to IPL revealed that 6 subjects showed bilateral IPL activation and 5 subjects showed unilateral IPL activation (4 on the right side and 1 on the left side; *P* < 0.005 uncorrected for multiple comparisons).

Activation in Ag cannot be explained by increased temporal discrepancy between action-related signals alone because there was no effect of delay on mean activations in the left and the right Ag. The effect of delay was instead associated with the activity in right posterior part of the superior temporal sulcus
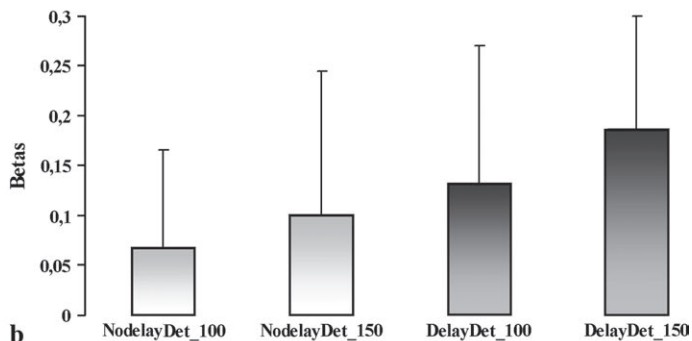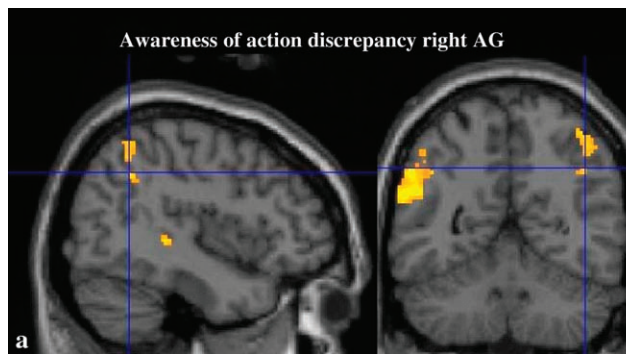
Figure 3. Awareness of action discrepancy. Activation of the right Ag when subjects are aware of a delay [(DelayDet100 + DelayDet150) – (NodelayDet100 + NodelayDet150)]. (*a*) The SPM{*T*} threshold at *P* < 0.001 (uncorrected) and superimposed on sagittal and coronal sections of the *T*₁ image shows the activation in the right Ag (*x*, *y*, *z*; 44, −54, 38). (*b*) Mean and standard errors of the beta values calculated in the right Ag (sphere of 8 mm centered at *x*, *y*, *z*; 44, −54, 38) for the no delay detection (NodelayDet_100 and NdelayDet_150) and the delay-detection conditions (DelayDet_100 and DelayDet_150).

Table 1
Awareness of action discrepancy

Area	Side	x	y	z	Z-score	K_E
Inferior parietal cortex (BA 39) Ag	R	44	−54	38	2.8	12
Inferior parietal cortex (BA 39) Ag	L	−40	−58	36	3.79	471
Supramarginal gyrus (BA 40)	L	−48	−38	54	3.08	16
Superior frontal sulcus (BA 9) DLPFC	L	−22	52	32	3.05	26
Superior frontal gyrus (BA 9) DLPFC	R	28	52	40	3	15
Orbital gyrus (BA 47)	L	−30	24	−12	2.85	17
Superior frontal gyrus (BA 6) preSMA	L	−10	26	64	3.28	19
Precentral gyrus (BA 6) PMd	L	−34	−20	66	2.83	15
Pulvinar	L	−14	−42	6	2.98	33

Notes: Detection of a delay independent of the delay value [(delaydet100 + delaydet150) – (nodelaydet100 + nodelaydet150)] (*P* ≤ 0.001 uncorrected for multiple comparisons at the voxel). PMd = dorsal premotor cortex; preSMA = pre supplementary motor area; DLPFC = dorsolateral prefrontal cortex. Coordinates [*x y z*] are reported in MNI space.

(BA 40) (Supplementary Table 3). Finally, awareness of action discrepancy was not restricted to the inferior parietal area, but also activated some premotor and prefrontal areas (Table 1).

Material and Methods of Study 2: Awareness of Action Authorship

Experiment 2 tested whether IPL areas, involved in awareness of action discrepancy, are also involved in awareness of action authorship. An initial behavioral experiment showed that introduction of large (≥800 ms) temporal delays has a strong influence on the subjects' sense of agency, leading them to consider the visual finger actions as another person's movements (Supplementary Fig. 1). We capitalized on this result to create an illusory situation in which subjects' perceptual experiences of agency were bistable. Subjects were led to believe that the image they were watching alternated between depicting their own movements with delay (preserved sense of agency: PresAgy) or those of another agent (perturbed sense of agency: PertAgy). In reality, they were always observing their own movements with easily perceived visual delays.

Subjects
Eighteen healthy subjects (27.9 ± 10.7 years: 11 females, 7 males) identified as right hand dominant according to the Edinburgh Handedness Inventory (Oldfield 1971) participated in this experiment. None of the participants had a history of psychiatric or neurological disease, and written informed consent was obtained from all the subjects in accordance with the Declaration of Helsinki. The experimental protocol was approved by the Committee for the Protection of Human Subjects at Dartmouth College.

Task and Procedure
The subjects' hands were filmed with an infrared camera located in front of the scanner; this image was sent outside the scanner room to the

Audio/Video delay unit and then projected to the subjects via LCD projector to a rear projection screen black at the head of the bore. This procedure is similar to an experiment performed by Nielsen (1963). The subjects were required to continuously perform alternate index and middle fingers movements during the whole run (150 s) without stopping, and watch their own movements with a certain amount of delay (1000 or 800 ms) that was maintained unchanged throughout the run. They were previously trained with a metronome to keep their movements constant (1 Hz) across the experiment. They were instructed that they could either see their own movements with a delay or another person's movements and that some undetectable shifts between their own and another person's movement sequences occurred randomly. Although in reality subjects only observed their own movements delayed, this situation induces them to spontaneously switch from a sense of self to other. They were thus required to press one key as soon as they believed that the observed movements were their own (PresAgy) or a second key if they believed actions belonged to another agent (PertAgy). These key presses therefore occurred each time the subjects' perceptual experience of agency changed (either from watching their own movements to watching another person's movements [PertAgy] or from watching another person's movements to watching their own movements [PresAgy]). The key press mappings were counterbalanced across subjects. No information about the probability of seeing their own movements and another person's movements was given to the subjects to prevent them from any anticipatory strategy. To prevent any other source of information that could have biased the subjects' decisions, 2 precautions were used. First, the subjects' right hands were covered with a snug fitting glove to prevent any morphological recognition cues. Secondly, a homogeneous background was created to prevent any "environmental" information from getting to the subjects; this was achieved by covering the subjects' neck down to their feet with an opaque black sheet. Only the middle and the index figures passed through 2 holes in the sheet. A block design with 2 different runs ("1000" and "800" ms) of 150 s each and repeated 5 times was used. In each run, the subjects performed the task during 120 s, and then a 30-s rest condition occurred during which the subjects did and saw nothing. Runs with a 1000-ms delay were alternated with runs with a 800-ms delay.

Functional Imaging
Functional imaging acquisition procedure was the same as study 1.

Data Analyses
Image analyses and statistical analyses were performed using SPM99 (http://www.fil.ion.ucl.ac.uk/spm/software/spm99). For each subject, all functional volumes were realigned to the first volume to correct for interscan movement. The resulting parameters were entered as variables of no interest in the statistical model. Functional and structural images were coregistered and transformed (Friston et al. 1995a) into a standardized, stereotaxic space (MNI template) (Evans et al. 1994). Functional data were then smoothed with an 8-mm FWHM,

isotropic Gaussian kernel. Low-frequency drifts were removed by incorporating linear and quadratic confound regressors that best estimate them.

The subjects' responses allowed us to group trials in which they experienced they were observing another person's movements, that is, perturbed agency (PertAgy) and trials in which they experienced they were observing their own movements, that is, preserved agency (PresAgy). A general linear model (GLM) was constructed with 6 event types modeled. "PresAgy1000," "PresAgy800," "PertAgy1000," and "PertAgy800" designated events in which the subjects felt they were observing their own movements or another person's movements with a delay of 1000 or 800 ms. "Rest1000" and "Rest800" corresponded to trials in which the subjects saw and did nothing. Each event type was used to construct a series of regressors by convolution of a box-car function with a standard hrf and its temporal derivatives. The 12 regressors plus the movements' realignment parameters and linear and quadratic regressors were entered into a GLM (Friston et al. 1995b). Parameter estimates pertaining to each type of effect of interest were calculated for each subject for each voxel producing an image of parameter estimates.

Two statistical comparisons were done.

1. *Movement-related activity.* The experimental blocks across all experimental conditions were grouped into one effect of interest and the main effect of task was then created for each subject.
2. *Perturbed agency.* The subjects' bistable perception allowed us to distinguish between trials where subjects experienced a perturbed agency (perceiving actions as belonging to another agent) versus a preserved agency (perceiving actions as belonging to themselves). This allowed us to assess brain activity associated with the perturbed sense of agency independently of the delay value.

Parameters for each of the trial types were estimated in a voxel-wise manner and used to produce contrast images for each subject. A random effect analysis was then applied (Holmes and Friston 1998). The contrast images for each effect were entered into a one-tailed t-test. The set of t-values thus obtained constituted SPM$\{T\}$. These SPM$\{T\}$s for each effect modeled were transformed into SPM$\{Z\}$s. We report significant activations for the basic motor task (all experimental conditions versus rest) corrected for multiple comparisons ($P < 0.05$). For other effects we had a priori predictions of anatomic localization, we report activations above a threshold corresponding to ($P < 0.001$ [uncorrected for multiple comparison], $Z \geqslant 3.10$ and minimal cluster size [$K_E \geqslant 10$]).

ROIs Analysis

The locations of peak activations in the left and right Ag were identified as ROIs from the statistical parametric map resulting from the comparison of PertAgy versus PresAgy conditions, $P < 0.001$ uncorrected for multiple comparisons, $K_E \geqslant 10$, minimum separation 8 mm. BOLD response data were extracted from ROIs as in study 1.

Brain Activity Localization

Brain activity localization procedures were the same as study 1.

Results of Study 2: Awareness of Action Authorship

Behavioral Data

Consistent with our preliminary behavioral results obtained prior to imaging (see supplementary material), subjects were more likely to perceive observed movements as their own (PresAgy) versus those of another (PertAgy), ($F_{1,17} = 8, 45$ $P = 0.0098$). However, there were no significant effects of delay (1000 or 800 ms) nor run nor of their interaction on the subjects' responses ($P > 0.05$), establishing that the magnitude of delay did not influence the subjects' responses and that they tend to make similar types of judgments over the duration of the experiment.

Imaging Data

Movement-Related Activity

An occipito-parieto-frontal network, typically associated with visually guided action (Grafton et al. 1996) was significantly activated when all experimental conditions were contrasted with the rest conditions ($P < 0.05$ corrected for multiple comparisons).

Perturbed Sense of Agency

Relative to trials on which there was a preserved sense of agency (see supplementary data), perceiving actions as belonging to another agent [(PertAgy1000 + PertAgy800) − (PresAgy1000 + PresAgy800)] produced results strikingly similar to study 1. Activation in the right hemisphere was maximal in the Ag (BA 39) and extended into the middle sector of the intraparietal sulcus (Fig. 4). Peak activation in the left hemisphere was also observed in the Ag (BA 39). Individual analyses restricted to IPL revealed that 10 subjects showed bilateral IPL activation and 5 subjects showed right IPL activation ($P < 0.005$ uncorrected for multiple comparisons). Additional activations were observed in frontal areas (Table 2).

Mean activations in the left and the right Ag were separately entered in univariate repeated-measure ANOVA with authorship (PresAgy vs. PertAgy) and delay (1000 vs. 800 ms) as within-subjects factors, to investigate the influence of the delay value
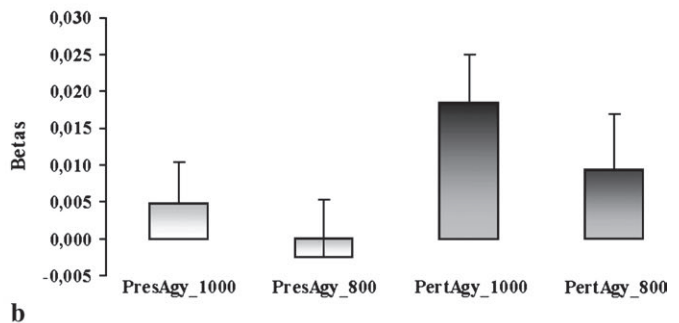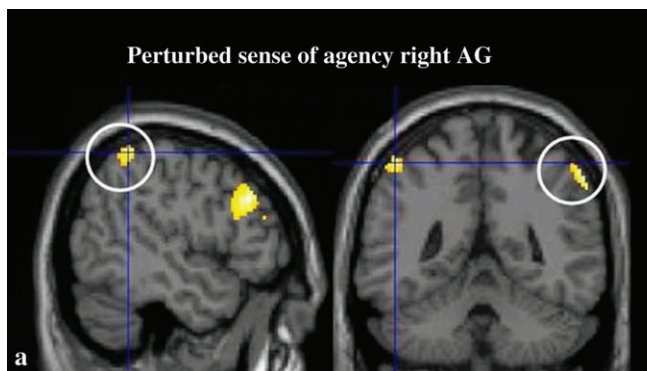

Figure 4. Perturbed agency. Activation of the right Ag when subjects experience a perturbed sense of agency [(PertAgy1000 + PertAgy800) − (PresAgy1000 + PresAgy800)]. (a) The SPM$\{T\}$ threshold at $P < 0.001$ (uncorrected) and superimposed on sagittal and coronal sections of the T_1 image shows the activation in the right Ag (x, y, z; 58, −46, 48). (b) Mean and standard errors of the beta values calculated in the right Ag (sphere of 8 mm centered at x, y, z; 58, −46, 48) for the perturbed sense of agency (PertAgy_1000 and PertAgy_800) and the preserved sense of agency conditions (PresAgy_1000 and PresAgy_800).

on Ag activation. Both analyses showed that neither the main effect of delay nor the interaction between the delay and the condition was significant ($P > 0.05$), showing that activation in the Ag cannot only be explained by increased temporal discrepancy between action-related signals.

Kinematics Data

The difference in BOLD activity when attributing an action to self or to another person might be due to subtle differences in kinematics performance by subjects in the 2 types of trials. To address this, we used an optoelectronic camera system (OPTOTRAK 3020, Northern Digital, Waterloo, Canada) to record the position of the index and ring finger-tips of 5 new subjects performing the task outside the scanner. Data was sampled at 100 Hz and low pass filtered (10 Hz) using a second-order butterworth filter. We computed for each finger its movement frequency, the maximal and minimal movement amplitude, and the normalized integrated jerk score (NIJ) for the preserved and perturbed sense of agency conditions. NIJ was computed as the sqrt$[(T^5/2L^2)\int J^2 \, dt]$, where J is the third time derivative of finger position, sqrt is square root, T is movement duration, L is finger path length, and the limits of integration are (0, T). This measure is an indicator of movement smoothness and is relatively independent of movement duration and amplitude (Kitazawa et al. 1993; Tresilian et al. 1997). Recordings were made over 4 runs of 150-s each (2 runs with a 100 ms delay and 2 runs with a 800 ms delay). The measures at the 800- and 100-ms delays were pooled together across the runs. Student's t-tests revealed no significant differences ($P > 0.05$) in all kinematics parameters between these 2 conditions showing that the perturbation of the sense of agency did not influence the kinematics.

Conjunction Analysis between Awareness of Action Discrepancy versus Awareness of Action Authorship

To evaluate whether the IPL activations associated with both forms of action awareness were similar in location, we compared the results obtained in the study of awareness of action authorship ($n = 18$) with those of the study of awareness of action discrepancy ($n = 15$). A random effects analysis modeling 2 populations was performed over the 2 studies. Images of parameter estimates for each contrast of interest were created for each subject and were then entered into a second-level analysis using a one-way ANOVA with the 2 hrfs as a factor. The contrast of interest for the first study consisted in the comparison between delays (100 and 150 ms) that were detected versus delays (100 and 150 ms) that were not detected. The contrast of interest for the second study consisted in the comparison between trials in which subjects experienced observing another person's movement and trials in which they experienced observing their own movements. SPMs of the T-statistic were constructed using a generalized Greenhouse–Geiser correction for heterogeneity of variance. Common network associated with these 2 forms of action awareness was revealed with a conjunction analysis using the inclusive masking procedure (thresholded at $P < 0.001$, uncorrected for multiple comparisons). For effects we had a priori predictions of anatomic localization, we report activations above a threshold corresponding to $P < 0.001$ uncorrected for multiple comparisons, $Z \geq 3.10$ and minimal cluster size $K_E \geq 10$.

The results showed that both forms of conscious experience of one's own actions activate the same Ag area (BA 39) (Fig. 5). Additionally to these parietal areas, frontal activations were also observed (Table 3).

Discussion

Results of our 2 studies indicate that processes implemented in the Ag give rise to 2 distinct types of action awareness.

Table 2
Perturbed agency

Area	Side	x	y	z	Z-score	K_E
Inferior parietal cortex (BA 39) Ag	R	58	−46	48	4.36	384
Intraparietal sulcus (BA 40) middle sector	R	44	−50	60	3.19	384
Inferior parietal cortex (BA 39) Ag	L	−48	−46	56	3.97	283
Middle frontal gyrus (BA 46) DLPFC	L	−48	28	30	4.5	1529
Middle frontal gyrus (BA 9) DLPFC	L	−44	22	36	3.81	1529
Frontomarginal gyrus (BA 10)	R	28	54	−2	3.34	220
Middle frontal gyrus (BA 8)	R	46	30	42	3.51	185
Inferior frontal gyrus (BA 44) PMv	L	−54	18	20	4.33	1529
Inferior frontal sulcus (BA 46) rostral part	R	38	50	−2	3.89	220

Notes: Perturbation of subjects' sense of agency [(PertAgy1000 + PertAgy800) − (PresAgy1000 + PresAgy800)] ($P \leq 0.001$ uncorrected for multiple comparisons at the voxel). DLPFC = dorsolateral prefrontal cortex. Coordinates [x y z] are reported in MNI space.
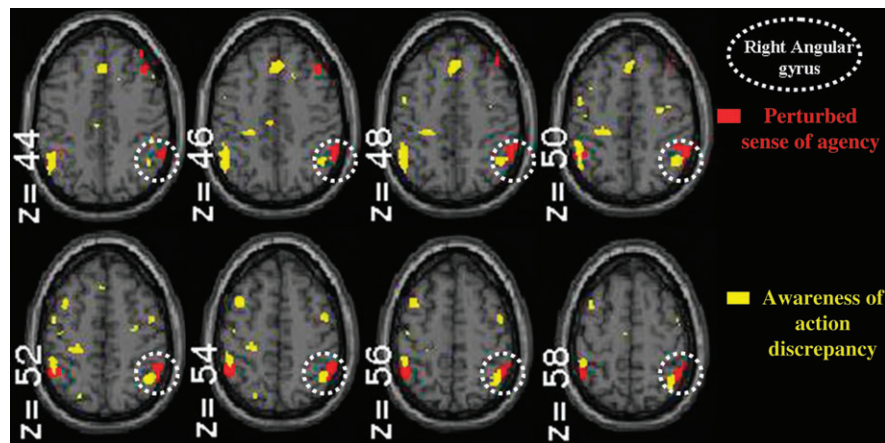

Figure 5. Conjunction analysis across the 2 studies. Awareness of action discrepancy (in yellow) and awareness of action authorship (in red) activate the same right IPL area: the right Ag (peak activation; x, y, z; 46, −50, 48; $P \leq 0.001$ uncorrected for multiple comparisons at the voxel; voxel extent threshold ≥ 10). Activations are shown superimposed on axial slices ($x = 46$, $y = −50$, $z = 44$–58). This analysis was corrected for variance heterogeneity using a generalized Greenhouse–Geiser correction.

Table 3
Conjunction analysis across the 2 studies

Area	Side	x	y	z	Z-score	K_E
Inferior parietal cortex (BA 39) Ag	R	46	−50	48	4.24	372
Intraparietal sulcus (BA 40)	R	48	−44	54	4.34	372
Inferior parietal cortex (BA 39) Ag	L	−48	−54	54	4.12	196
Frontopolar gyrus (BA 10)	R	28	58	2	3.78	47
Middle frontal gyrus (BA 8)	L	−38	24	50	3.2	42
Inferior frontal gyrus pars triangularis (BA 45)	R	52	22	16	4.29	109
Inferior precentral sulcus (BA 44)	L	−54	18	32	3.43	40
Precentral gyrus (BA 6) PMd	R	48	−2	52	3.32	13

Notes: Common network associated with both awareness of action discrepancy and perturbed agency was revealed with a conjunction analysis using the inclusive masking procedure and the generalized Greenhouse–Geiser correction for heterogeneity of variance ($P < 0.001$, uncorrected for multiple comparisons at the voxel). PMd = dorsal premotor cortex. Coordinates [x y z] are reported in MNI space.

Manipulating the correspondence between intended actions and their perceived consequences, by delaying the visual feedback, induced our subjects to access action awareness at different levels. In the first study subjects became aware of a perturbation of their own movements with a delay greater than 100–150 ms. This study confirms previous findings that show awareness of one's own movement perturbation at a similar range of delays (Franck et al. 2001; Leube et al. 2003). Although subjects were aware of the perturbation, they still attributed the visualized movements to themselves. In our second study, the delay was greatly increased and the source of the action was made ambiguous by making the subjects believe that they were observing either their own or another person's movements. This intermittently induced the illusion of misattributing movements to another agent. Their sense of agency was thus perturbed. This result echoes previous findings using a similar paradigm that found that subjects can either feel in control of an action that is in fact controlled by another agent or feel that another person is controlling an action that is controlled by themselves (Sato and Yasuda 2003; Wegner et al. 2003).

These 2 forms of action awareness may rely on the same predictive computational mechanisms that compare intended and actual consequences of action and these processes may be subserved by the same network. As predicted, a conjunction analysis revealed that the Ag is similarly involved in awareness of action discrepancy and awareness of action authorship. Additionally, these 2 forms of action awareness activated similar prefrontal and premotor areas.

Alternatively, increased Ag activation might reflect increased attentional processing of visual feedback (Desimone and Duncan 1995). However, it is unclear why subjects would pay greater attention given that the stimulus delay was constant and only their experiences of the movements as being perturbed or belonging to another differed. Additionally, subjects were required to pay equal attention to the hand movements for each condition of each study and the equivalence of their performance scores across conditions indicate that they complied. Furthermore, activation associated with observation of biological movements of the hand is localized in the posterior part of the superior temporal sulcus (Puce and Perrett 2003; Hamilton et al. 2006), whereas attention to action activates the intraparietal sulcus (Rowe et al. 2002). These 2 areas are more anterior than those we found associated with action awareness. It could also be argued that Ag activation only reflects process-

ing of the delay with increased delay processing for both detection of delay and perturbed sense of agency. However, analyses were carefully done while controlling for the amount of delay. The same delays were involved for trials for which subjects detected the delay versus had a perturbed sense of agency, and trials for which subjects did not detect the delay versus had a preserved sense of agency. Furthermore, in the second study, behavioral results showed that the magnitude of delay did not influence the subject's responses. Finally, these activations cannot be explained by differences in the quantity or quality of movements because no significant modulation of brain activity was associated with the number of pegs removed, even at a low threshold ($P < 0.05$ uncorrected) in the first study. In the second study, kinematics analyses revealed no influence of the perturbation of subjects' sense of agency on kinematics parameters (movement frequency, jerk, maximal, and minimal amplitudes).

Evidence of involvement of right IPL in action awareness has been shown in studies involving healthy subjects and patients. In healthy subjects right IPL has been found to be recruited in studies that distinguished between self and other-generated actions (see Jackson and Decety 2004, for review). A similar paradigm to the one used in the present study found activation in the Ag bilaterally when subjects do not feel in control of an action and attribute it to another agent (Farrer and Frith 2002). Studies with neurological and psychiatric patients show growing evidence that right IPL is associated with action awareness disturbances. Neurological patients (with all patients' lesions involving the right Ag) present altered awareness of voluntary action (Daprati et al. 2000; Sirigu et al. 2004). Schizophrenic patients who suffer from Schneiderian symptoms, in which patients feel that their own thoughts and/or actions are controlled by someone else, show abnormal activation of this region while manifesting these symptoms (Spence et al. 1997) or while making action-attribution judgments (Farrer et al. 2004).

Action awareness involves processing and comparing intended and actual consequences of action and arises in the present studies from the discordance between these signals. We propose that integration of these signals occurs in the Ag, as suggested both by the present results and by previous evidence of a correlation between Ag activation and the degree of discrepancy between intended and actual consequences of movement (Farrer et al. 2003a). Activity in Ag thus reflects a mismatch between these signals, being either abnormal in the case of neurological and schizophrenic patients, or artificially induced in the case of the present studies. What we further show is that Ag is equally associated with distinct conscious experiences of one's own actions arising from this discordance. Both awareness of action discrepancy and action authorship are associated with the same IPL area even though these experiences differ in their content.

It has been proposed that different internal models are used for different purposes and these models may be associated with different brain areas (Blakemore and Sirigu 2003). An implicit module in the cerebellum would be used for predictive control of voluntary action providing fast processing for action execution and prediction of the sensory consequences (Ito 1970; Kawato and Gomi 1992; Miall et al. 1993). An explicit module in the parietal cortex would monitor intention and motor plans at a higher level, detecting when actions match their desired goals (Sirigu et al. 2004). We propose that this region is involved in higher-order aspects of motor control that allow one to

consciously access different aspects of one's own actions closely related to intention and agent. Specifically, this region processes discrepancies between intended action and movement consequences in such a way that these will be consciously detected by the subject. This processing is at the core of the various experiences one can have about one's own action such as the sense of agency.

Supplementary Material

Supplementary material can be found at: http://www.cercor.oxfordjournals.org/.

Notes

Address correspondence to: Chlöé Farrer, Centre de Neuroscience Cognitive, UMR 5229 CNRS, 67 Bd Pinel, 69675 Bron, France. Email: farrer@isc.cnrs.fr.

References

Blakemore SJ, Frith CD, Wolpert DM. 1999. Spatio-temporal prediction modulates the perception of self-produced stimuli. J Cogn Neurosci. 11(5):551-559.

Blakemore SJ, Sirigu A. 2003. Action prediction in the cerebellum and in the parietal lobe. Exp Brain Res. 153(2):239-245.

Daprati E, Sirigu A, Pradat-Diehl P, Franck N, Jeannerod M. 2000. Recognition of self-produced movement in a case of severe neglect. Neurocase. 6:477-486.

Desimone R, Duncan J. 1995. Neural mechanisms of selective visual attention. Annu Rev Neurosci. 18:193-222.

Desmurget M, Grafton ST. 2000. Forward modeling allows feedback control for fast reaching movements. Trends Cogn Sci. 14(11):423-431.

Duvernoy M. 1992. Le cerveau humain. Paris: Spinger & Verlag.

Evans AC, Kamber M, Collins DL, MacDonald D. 1994. A MRI-based probabilistic atlas of neuroanatomy. In: Shorvon S, Fish D, Andermann F, Bydder GM, Stefan H, editors. Magnetic resonance scanning and epilepsy. NATO ASI series A, life sciences. Vol. 264. New York: Plenum. p. 263-274.

Farrer C, Franck N, Georgieff N, Frith CD, Decety J, Jeannerod M. 2003a. Modulating the sense of agency: a PET study. Neuroimage 18(2): 324-333.

Farrer C, Franck N, Paillard J, Jeannerod M. 2003b. The role of proprioception in action recognition. Conscious Cogn. 12(4):609-619.

Farrer C, Franck N, Frith CD, Decety J, Georgieff N, d'Amato T, Jeannerod M. 2004. Neural correlates of action attribution in schizophrenia. Psychiatry Res. 131(1):31-44.

Farrer C, Frith CD. 2002. Experiencing oneself vs another person as being the cause of an action: the neural correlates of the experience of agency. Neuroimage. 15:596-603.

Friston KJ, Ashburner J, Frith CD, Poline JB, Heather JD, Frackowiak RSJ. 1995a. Spatial registration and normalisation of images. Hum Brain Mapp. 2:165-189.

Friston KJ, Holmes AP, Worsley KJ, Poline JB, Frith CD, Frackowiack RSJ. 1995b. Statistical parametric mapping in functional imaging: a general linear approach. Hum Brain Mapping 2: 189-210.

Fourneret P, Jeannerod M. 1998. Limited conscious monitoring of motor performance in normal subjects. Neuropsychologia. 36(11):1133-1140.

Franck N, Farrer C, Georgieff N, Marie-Cardine M, Daléry J, d'Amato T, Jeannerod M. 2001. Defective recognition of one's own actions in patients with schizophrenia. Am J Psychiatr. 158:454-459.

Frith CD, Blakemore S, Wolpert D. 2000. Abnormalities in the awareness and control of action. Phil Trans R Soc Lond. 355:1771-1788.

Grafton ST, Fagg AH, Woods RP, Arbib MA. 1996. Functional anatomy of pointing and grasping in humans. Cereb Cortex. 6(2):226-237.

Haggard P. 2005. Conscious intention and motor cognition. Trends Cogn Sci. 9(6):290-295.

Hamilton AF, Wolpert DM, Frith U, Grafton ST. 2006. Where does your own action influence your perception of another person's action in the brain? Neuroimage. 29(2):524-535.

Holmes AP, Friston KJ. 1998. Generasibility, random effects and population inference. Neuroimage. 7:S754.

Holst E, Von Mittelstaedt H. 1950. Das reafferenzprinzip. Wechselwirkungen zwischen zentralnervensystem und peripherie. Naturwissenschaften. 37:464-476.

Ito MO.1970. Neurophysiological aspects of the cerebellar motor control system. Int J Neurol. 7(2):162-176.

Jackson PL, Decety J. 2004. Motor cognition: a new paradigm to study self-other interactions. Curr Opin Neurobiol. 14(2):259-263.

Jeannerod M. 2003. Agency and self awareness. In: Roessler J, Eilan N, editors. Issues in philosophy and psychology. Oxford: Oxford University Press. p. 128-149.

Jordan MI. 1996. Computational aspects of motor control and motor learning. In: Heuer EH, Keele ES, editors. Handbook of perception and action: motor skills. New York: Academic Press. p. 71-118.

Kawato M, Gomi H. 1992. A computational model of four regions of the cerebellum based on feedback-error learning. Biol Cybern. 68(2):95-103.

Kitazawa S, Goto T, Urushihara Y. 1993. Quantitative evaluation of reaching movements in cats with and without cerebellar lesions using normalized integral of jerk. In: Mano N, Hamada I, DeLong MR, editors. Role of the cerebellum and basal ganglia in voluntary movement. Amsterdam: Elsevier. p. 11-19.

Leube DT, Knoblich G, Erb M, Grodd W, Bartels M, Kircher TT. 2003. The neural correlates of perceiving one's own movements. Neuroimage 20(4):2084-2090.

Miall RC, Weir DJ, Wolpert DM, Stein JF. 1993. Is the cerebellum a smith predictor? J Mot Behav. 25(3):203-216.

Nielsen T. 1963. Volition: a new experimental approach. Scand J Psychol. 4:225-230.

Oldfield RC. 1971. The assessment and analysis of handedness: The Edinburgh Inventory. Neuropsychologia. 9:97-113.

Puce A, Perrett D. 2003. Electrophysiology and brain imaging of biological motion. Philos Trans R Soc Lond B Biol Sci. 358(1431):435-445.

Rowe J, Friston K, Frackowiak R, Passingham R. 2002. Attention to action: specific modulation of corticocortical interactions in humans. Neuroimage. 17(2):988-998.

Sato A, Yasuda A. 2003. Illusion of sense of self-agency: discrepancy between the predicted and actual sensory consequences of actions modulates the sense of self-agency, but not the sense of self-ownership. Cognition. 94(3):241-255.

Schmahmann JD, Doyon J, Toga A, Evans A, Petrides M. 2000. MRI atlas of the human cerebellum. San Diego: Academic Press.

Schneider K. 1955. Klinische Psychpathologie. Stuttgart: Thieme Verlag.

Sirigu A, Daprati E, Ciancia S, Giraux P, Nighoghossian N, Posada A, Haggard P. 2004. Altered awareness of voluntary action after damage to the parietal cortex. Nat Neurosci. 7(1):80-84.

Spence SA, Brooks DJ, Hirsch SR, Liddle PF, Meehan J, Grasby PM. 1997. A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). Brain. 120(Pt 11):1997-2011.

Sperry RW. 1950. Neural basis of the spontaneous optokinetic response produced by visual inversion. J Comp Psychol. 43:482-489.

Talairach J, Tournoux P. 1988. Coplanar stereotaxic atlas of the human brain. New York: Thieme Medical.

Tresilian JR, Stelmach GE, Adler CH. 1997. Stability of reach-to-grasp movement patterns in Parkinson's disease. Brain. 120: 2093-2111.

Tsakiris M, Haggard P, Franck N, Mainy N, Sirigu A. 2005. A specific role for efferent information in self-recognition. Cognition. 96(3):215-231.

Von Helmholtz H. 1886. Handbuch der Physiologischen Optik. Leipzig: Voss.

Wegner DM, Fuller VA, Sparrow B. 2003. Clever hands: uncontrolled intelligence in facilitated communication. J Pers Soc Psychol. 85(1):5-19.

Wolpert DM, Ghahramani Z, Jordan MI. 1995. An internal model for sensorimotor integration. Science. 269:1880-1882.