

# Implicit Agency in Observed Actions: Evidence for N1 Suppression of Tones Caused by Self-made and Observed Actions

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## Abstract

■ Every day we make attributions about how our actions and the actions of others cause consequences in the world around us. It is unknown whether we use the same implicit process in attributing causality when observing others' actions as we do when making our own. The aim of this research was to investigate the neural processes involved in the implicit sense of agency we form between actions and effects, for both our own actions and when watching others' actions. Using an interval estimation paradigm to elicit intentional binding in self-made and observed actions, we measured the EEG responses indicative of anticipatory processes before an action and the ERPs in response to the sensory consequence. We replicated our previous

findings that we form a sense of implicit agency over our own and others' actions. Crucially, EEG results showed that tones caused by either self-made or observed actions both resulted in suppression of the N1 component of the sensory ERP, with no difference in suppression between consequences caused by observed actions compared with self-made actions. Furthermore, this N1 suppression was greatest for tones caused by observed goal-directed actions rather than non-action or non-goal-related visual events. This suggests that top-down processes act upon the neural responses to sensory events caused by goal-directed actions in the same way for events caused by the self or those made by other agents. ■

## INTRODUCTION

Understanding our own and others' interactions with the world is a vital part of our day-to-day existence. We initiate our own movements to reach a goal and also watch others initiate their own movements to interact with the environment. Our own movements may include pressing a button to open a door, or we may watch someone else struggle with gripping a lid to open a jar. For our own movements, we form a sense of agency over the events that occur as a result of our own actions. We form the idea that we "caused" an event to happen through our movements (Gallagher, 2000). Recent investigations have suggested that we also attribute agency over others' actions in a similar way as we do for our own (Poonian & Cunnington, 2013; Sato, 2008). However, it is still poorly understood how the brain integrates our own and others' actions with their relevant sensory consequences to give us a coherent perceptual experience.

A recent theory has suggested that sense of agency involves two levels of attributions, an implicit "feeling of agency" and a more conscious judgment of agency (Synofzik, Vosgerau, & Newen, 2008). Implicit agency is hypothesized to relate to the causal association we make between our

own actions and their sensory consequences (Synofzik et al., 2008). An indirect measure of this association involves tasks where voluntary self-made actions and sensory consequences are perceived as occurring closer together in time when actions and effects are paired together (Moore & Obhi, 2012; Haggard, Clark, & Kalogeras, 2002). This perceived temporal shift of the action and consequence toward each other is thought to be because of the action and consequence being "bound together" in time (Haggard et al., 2002; for a review, see Moore & Obhi, 2012). This phenomenon, termed intentional binding, has also recently been measured when others execute actions, including just watching others' actions on a screen or in real life (Poonian & Cunnington, 2013; Wohlschlagel, Engbert, & Haggard, 2003) or when a person's action is part of a shared action with another (Obhi & Hall, 2011a; Strother, House, & Obhi, 2010). Although the association between movements and consequences has previously been demonstrated for both our own and others' actions, it remains to be seen whether the binding that occurs when another agent performs an action involves the same cognitive and brain processes found when we execute our own actions.

The causal attribution we make between actions and consequences is hypothesized to occur through predictive processes (Wolpert, Ghahramani, & Jordan, 1995). These processes include predictions that are made when planning an action (David, Newen, & Vogeley, 2008; Synofzik

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et al., 2008) as well as a comparison that occurs between the predicted sensory effect and the actual sensory effect (Blakemore, Frith, & Wolpert, 2001; Wolpert & Ghahramani, 2000). This comparison is thought to occur through efference copies or corollary discharges of the movement (von Holst, 1954; Sperry, 1950). These two aspects of predictive forward models, the prediction about an action (Wolpert et al., 1995) and the comparison of the predicted effects to the actual effect (Blakemore, Wolpert, & Frith, 2000), are thought to contribute to the causal associations we make between our actions and their sensory consequences (David et al., 2008). Recently it has been suggested that a similar predictive process is involved when we observe others' goal-directed actions; that is, we predict the goals, intentions, and consequences of others' actions through an internal representation of the observed action (Kilner, Friston, & Frith, 2007). In a similar way as is hypothesized for our own actions, it is also thought that we compare the actual consequences of an observed action to the effects that are predicted (Kilner et al., 2007).

Investigations into the neural processes associated with actions and their sensory consequences have predominantly focused on two EEG components. The first examines the neural activity leading up to a voluntary action representing the planning and preparatory activity before the initiation of a movement (Libet, Gleason, Wright, & Pearl, 1983; Deecke, Scheid, & Kornhuber, 1969). The second examines the neural processing of sensory consequences caused by self-made actions (Aliu, Houde, & Nagarajan, 2009; Martikainen, Kaneko, & Hari, 2005). These components have not only been measured during action-effect tasks to investigate the processing of actions and their consequences independently but also used as evidence to indicate the role predictive processes have when associating our actions with their corresponding effect.

Predictions about our own actions are thought to occur through motor commands from activity in premotor areas, such as the SMA (David et al., 2008). Neural activity before the execution of an action has previously been found in the SMA and pre-SMA (Cunnington, Windischberger, & Moser, 2005; Ikeda et al., 1999; Cunnington, Bradshaw, & Iansek, 1996; Deiber, Ibanez, Sadato, & Hallett, 1996; Ikeda, Luders, Burgess, & Shibasaki, 1992). In EEG tasks where self-made actions are executed, a slow-wave negative potential, known as the readiness potential (RP), begins approximately 2 sec before a movement and rises to a peak just before movement onset (Libet et al., 1983; Deecke et al., 1969).

Direct links between activity in SMA with attributions of self-agency have recently been made (Kuhn, Brass, & Haggard, 2012; Miele, Wager, Mitchell, & Metcalfe, 2011; Moore, Ruge, Wenke, Rothwell, & Haggard, 2010; Haggard & Clark, 2003). During implicit agency tasks, disruption of the SMA during preparation of an action results in less action-effect binding (Moore et al., 2010; Haggard & Clark, 2003). Therefore, it could be suggested

that slow-wave activity from SMA before an action event is involved in the attribution of agency we make between actions and effects.

Predictions about others' actions are hypothesized to involve mapping of the observed action onto areas that are also involved when a person executes their own movements (Kilner et al., 2007; Rizzolatti et al., 1996). Investigations using slow-wave potentials have examined activity before the presentation of another person's hand grasping an object. During this task, a contingent negative variation (CNV) was elicited before the presentation of the moving hand (Kilner, Vargas, Duval, Blakemore, & Sirigu, 2004). This suggests that the higher motor areas are active before observing another's actions (Kilner et al., 2004). The CNV is thought to index the anticipation of an upcoming stimulus (Brunia & van Boxtel, 2001), and during action observation, it is hypothesized to index activity from motor areas that are activated within the action observation network, again because of predictions about the observed action (Kilner et al., 2004). It therefore appears that slow-wave activity may be present before both our own actions and when watching others' actions; however, it is yet to be determined whether this activity is related to the attribution of agency for our own and others' actions.

The comparison between predicted and actual consequences of an action is also thought to contribute to the association between actions and consequences. In the comparator model, when the predicted and actual consequence match, we infer agency to ourselves. If there is a mismatch, we do not infer ourselves as the cause of a consequence (Blakemore, Wolpert, & Frith, 2002). Investigations into the prediction of consequences have focused on particular components of the sensory ERP. Reductions in the amplitude of the N1 component have been found for sensory effects caused by self-made actions (i.e., self-made effects; Bass, Jacobsen, & Schroger, 2008; Martikainen et al., 2005) in both the auditory domain (Hughes, Desantis, & Waszak, 2013; Knolle, Schroger, & Kotz, 2013; Baess, Horvath, Jacobsen, & Schroger, 2011; Lange, 2011; Aliu et al., 2009) and the visual domain (Gentsch, Kathmann, & Schutz-Bosbach, 2012; Gentsch & Schutz-Bosbach, 2011; Hughes & Waszak, 2011). Even greater decreases in amplitude have been found for self-made consequences that were predictable compared with self-made effects that were not (Hughes et al., 2013; Knolle et al., 2013; Gentsch et al., 2012). The decrease in N1 amplitude is thought to occur because of top-down predictions from the motor system suppressing the activity of the auditory cortex (Aliu et al., 2009). Many authors have concluded that the N1 suppression during action-effect tasks provides evidence that we process the consequences of our own actions through predictions within an internal model of action (Hughes et al., 2013; Aliu et al., 2009; Bass et al., 2008).

In relation to agency, greater ratings of agency and greater N1 suppression has been found for sensory effects

that were subliminally predictable for both action-related (Gentsch & Schutz-Bosbach, 2011) and non-action-related stimuli (Gentsch et al., 2012). The authors concluded that N1 suppression was evidence for top-down predictions about the sensory consequence and suggest that this attenuation plays a part in the conscious experience of agency (Gentsch et al., 2012; Gentsch & Schutz-Bosbach, 2011). Although these tasks investigated unconscious processing of agency, subjective ratings of agency were measured using an explicit judgment of causation and not by behaviorally measuring the temporal association between actions and effects, thought to index implicit agency, as assessed during intentional binding tasks.

Inferring causality for other agents' actions can also be measured either implicitly (Poonian & Cunnington, 2013; Obhi & Hall, 2011b) or explicitly (Sato, 2008, 2009), similar to agency measures for self-made actions. In both types of tasks, the causal attribution between others' actions and effects are hypothesized to involve predictions about the goal, intentions, and consequences of another agent's actions (Poonian & Cunnington, 2013; Sato, 2008). It remains to be seen whether the attribution of agency we form for others' actions is also indexed by similar brain mechanisms as found when we attribute causality over our own actions.

Very few investigations have examined whether the attenuation of the sensory ERP for self-made actions also occurs when watching another agent perform an action. Using a task where tones were thought to be caused by another person, no difference in N1 amplitude was found for those tones that were judged to be caused by the self compared with those that were judged to be caused by another (Kuhn et al., 2011). Similarly N1 suppression has been found for tones that occurred as a result of a joint action (Loehr, 2013). Another avenue of research has examined tasks that combine observation of biological actions with an auditory consequence. It has been found that watching visual lip movements that are combined with auditory speech sounds result in suppression of early components of the auditory ERP (Stekelenburg & Vroomen, 2007; van Wassenhove, Grant, & Poeppel, 2005). This multisensory integration of auditory and visual events extends to other biological motion tasks such as clapping or tapping an object (Stekelenburg, Maes, Van Gool, Sitskoorn, & Vroomen, 2013; Stekelenburg & Vroomen, 2007). It is suggested that the N1 suppression in these tasks is because of internal predictions about the expected outcome of the biological action (Stekelenburg & Vroomen, 2012; Arnal, Morillon, Kell, & Giraud, 2009; van Wassenhove et al., 2005). However, the suppression found for consequences of others' biological actions has never been linked to causal attributions that we implicitly make between others' actions and effects as can be measured in intentional binding.

Therefore the aim of this research was to investigate the neural processes involved in the causal attribution made between actions and effects when sensory events

are caused by a self-made action or an observed action. In Experiment 1, we investigated two separate EEG components while performing an implicit agency task: the activity before the onset of an executed or observed action and the ERP response to the sensory consequence of the action. If implicit agency in observed and self-made actions does indeed arise from the same underlying neural process, then we would expect to find similar preaction activity before the onset of an action (self-made or observed) and similar suppression of early components of the sensory ERP for each action. In a second experiment, we investigated whether the suppression of the N1 component was related specifically to an observed agent executing a goal-directed action rather than a non-action or non-goal-related visual event. To date, this is the first research to directly investigate the neural processes involved in intentional binding for both self-made and observed actions.

## EXPERIMENT 1

### Methods

#### *Participants*

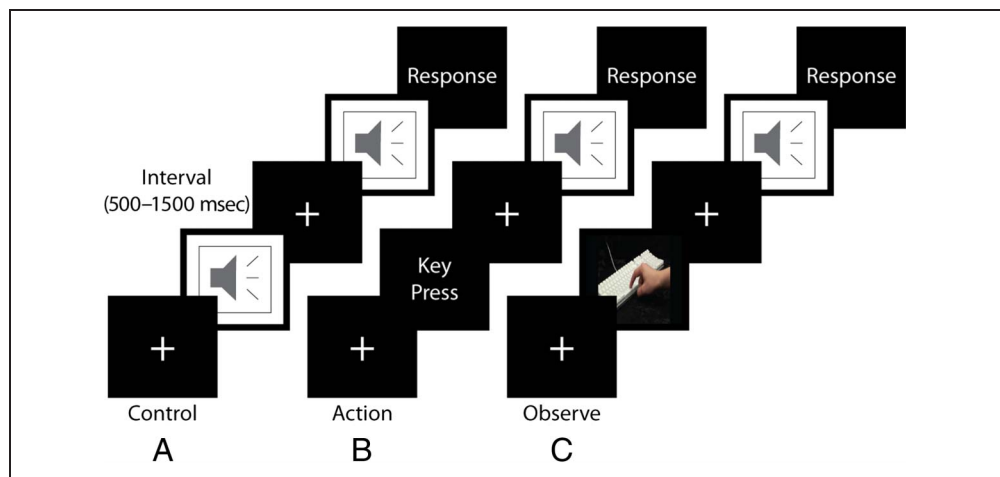
Twenty right-handed participants (seven men, age range = 18–29 years, mean = 23.5 years) with no known neurological disease or psychiatric conditions provided written informed consent to participate in this event-related EEG experiment. One data set was removed from further analysis after a failed recording session. Two further data sets were rejected for failing to reach the threshold for included trials following the EEG artifact rejection (artifact rejection threshold =  $\pm 120 \mu\text{V}$ , minimum 80 trials included). All analyses were conducted on the remaining 17 data sets. The study was approved by the University of Queensland medical research ethics committee (UQ Project No. 2008000703).

#### *Procedure*

To investigate the preaction/stimulus activity and the auditory ERPs in an implicit agency task, participants completed an interval estimation paradigm previously known to elicit intentional binding in observed and self-made actions (Poonian & Cunnington, 2013; Humphreys & Buehner, 2010). All trials began with a white fixation-cross presented centrally, whereas auditory stimuli were presented via two speakers on either side of the computer screen. All stimuli were presented, and responses were recorded using Cogent software ([www.vislab.ucl.ac.uk/cogent](http://www.vislab.ucl.ac.uk/cogent)).

Participants completed three separate conditions (Figure 1). In control conditions, participants were presented with a 150-msec auditory tone (440 Hz sine wave, sample rate = 44,100 Hz, bit rate = 16) created using MATLAB software (Mathworks, Natick, MA). After a pseudorandom delay of 500–1500 msec (varying by intervals of 100 msec),

**Figure 1.** Procedure for interval estimation binding task. In each condition, the first event consisted of either a tone (A), self-made button press (B), or observed button press (C). After a pseudorandom interval (500–1500 msec), a tone was presented. Participants were required to recreate the interval between the first event and tone by holding down a button for the length of the estimated interval.



another identical auditory tone was presented. After 1000 msec, participants were prompted on screen to recreate the stimulus interval by holding down the space bar with their right index finger for the length of time that they perceived the interval between the start of the first tone and the start of the second tone to have lasted.

In action conditions, participants were required to make a key press using their right index finger at a time of their choosing once the trial had started. After a pseudorandom interval (500–1500 msec), the 150-msec auditory tone was presented. When prompted, participants were required to recreate the perceived interval between their own key press and the onset of the tone.

In observe conditions, a video was presented of another person performing a key press. The video was created from a set of 37 bitmap images presented in rapid succession (duration = 1300 msec). Throughout the video, a white keyboard was shown in front of a black background. The video sequence involved a right hand appearing from the top right hand of the screen (Frame 5), reaching over to press the space bar on the keyboard (Frame 19), then moving back the way it came and leaving via the right side of the screen (Frame 36). The auditory click of the key press was also presented to coincide with the key press. The 150-msec tone was presented at a pseudorandom interval between 500 and 1500 msec after the fully depressed key press image was presented (Frame 19). When prompted, participants were required to recreate the interval between the observed key press, the time at which the key was fully depressed and the onset of the auditory tone.

In previous action-effect tasks, a movement-only condition is included to subtract out movement-related activity from the auditory ERP. As all of our tone stimuli were presented at least 500 msec after a movement had been executed, any motor contamination in the auditory ERP would be negligible; therefore, we did not collect a separate movement-only condition.

Each of the three conditions consisted of 132 trials, which were presented in separate blocks of 11 trials each.

Blocks were presented in a pseudorandom order and counterbalanced between participants.

### *Behavioral Analysis*

To measure binding, stimulus intervals were calculated as the difference between the time of the first event (first tone, self-made key press, or observed key press in the respective conditions) and the onset of the subsequent tone (500–1500 msec). These were then compared with response intervals, which were calculated as the time between the participants' downward key press and time of the upward release of the response key.

Reproduction errors were calculated as the difference between the stimulus interval and the response interval for each trial. Mean reproduction errors for all conditions across all participants were calculated. Negative reproduction errors indicate that participants perceived the interval between the first event and the tone to be shorter than it was (underestimate). A positive reproduction error indicated that participants perceived the stimulus interval to be longer than it was (overestimate). Mean reproduction errors for the 17 participants whose data were included in the EEG analysis were compared using a one-way repeated-measures ANOVA with the factor of Condition (control, action, observe).

### *EEG Acquisition*

EEG recordings were made using a BioSemi ActiveTwo system with 64 Ag/AgCl electrodes (BioSemi B.V, Amsterdam, Netherlands), placed according to the extended international 10–20 system. Data were sampled at a rate of 1024 Hz and analyzed offline using BESA software (BESA GmbH, Grafelfing, Germany); to calculate all average ERPs, an in-house software package was used to extract peak amplitudes of components. EOG recordings were measured using separate Ag/AgCl electrodes that were positioned above and below the left eye for vertical EOG and



adjacent to the outer canthus of each eye for horizontal EOG. Recordings of reference mastoids were also taken.

### *EEG Analysis*

EEG analysis consisted of referencing the raw EEG data to linked mastoid electrodes, across all data sets, including all conditions and electrodes. Blink artifacts were corrected for each participant using a filter, based on spatial topography (Berg & Scherg, 1994). Low-pass (40 Hz) and high-pass filters (0.05 Hz) were applied to all data. The high-pass filter in particular was set very low (0.05 Hz = 20 sec cycle) to retain slow-wave activity associated with preaction potentials but to attenuate slower baseline drift. Once data had been preprocessed, two types of epochs were created: preaction potentials and auditory ERPs. Epochs for preaction potentials began at 1500 msec before the onset of the first event and continued to 500 msec after the first event. Preaction epochs were baseline-corrected using a time window of 1500–1400 msec before first event onset. Epochs for auditory ERPs began at 500 msec before tone onset to 2000 msec after tone onset. These were then baseline-corrected using a time window between 100 and 0 msec before tone onset. Epochs were rejected if they contained artifacts caused by poor signal or movement (artifact rejection threshold =  $\pm 120 \mu\text{V}$ ), with each data set needing to contain at least 80 artifact-free epochs per condition. Of the remaining 17 data sets, the preaction potentials and auditory ERPs were created and averaged for each condition.

*Preaction potentials.* Grand averages of preaction potentials were defined in relation to the onset of the first event in each condition (tone, self-made key press and full depression of the observed key press). Mean amplitudes, for each electrode of interest, were taken across six time intervals of 150 msec each, beginning with 1400 msec through to 500 msec before the onset of the first event. The analyzed time windows ended at 500 msec because we needed to exclude any event-related changes in observe conditions because of the onset of the observed action stimulus.

Electrodes were grouped together into a grid of nine different locations across the scalp covering left/midline/right and frontal/central/parietal regions. Pairs of electrodes within each region were averaged together to further improve ERP signal (Baker, Piriyaunayaporn, & Cunnington, 2012). The pairs of electrodes for each region were left frontal (F1, F3), left central (FC3, C3), left parietal (CP3, CP1), middle frontal (AFz, Fz), middle central (FCz, Cz), middle parietal (Pz, CPz), right frontal (F2, F4), right central (FC4, C4), and right parietal (CP4, CP2). This grid covered premotor and motor areas, as well as parietal areas important to both intentional self-made actions and the observation of actions. The amplitude of participant's preaction potentials across electrode groups and time windows was compared using a 3 (Condition)  $\times$  6 (Time window)  $\times$

9 (Electrode group) repeated-measures ANOVA. Note that our primary goal in this analysis was to identify significant differences in ERP amplitudes between conditions, across particular electrode sites, and not more broadly to assess changes in scalp topography. Therefore, we did not normalize ERPs across electrode regions for topographic analysis (e.g., McCarthy & Wood, 1985).

*Auditory ERPs.* Grand-averaged auditory ERPs were created for all tone stimuli across the three conditions. The four ERP conditions created included the first tone in control conditions (first tone), the second tone in control conditions (second tone), the tone that followed a self-made action (action tone), and the tone that followed an observed action (observe tone).

Statistical analysis was conducted on two ERP components for all participants across nine frontocentral electrodes (F3, Fz, F4, C3, Cz, C4, CP3, CPz, CP4). For the N1 component, a 25-msec window centered around the peak of the grand-averaged waveform for all conditions was used (80–105 msec). For the P2 component, a 55-msec window centered around the peak of the grand-averaged waveform was used (145–200 msec). Mean amplitudes across these time windows were calculated for each participant and compared using two separate 4 (Condition)  $\times$  9 (Electrode) repeated-measures ANOVAs.

## **Results**

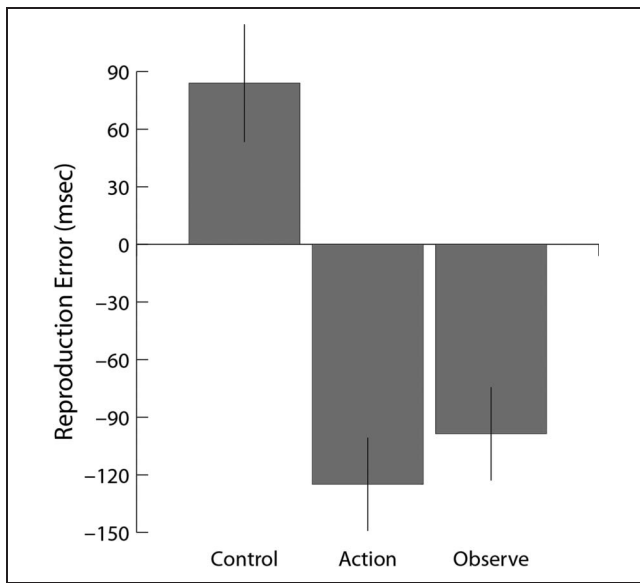
### *Behavioral Results*

Mean reproduction errors for the control, action, and observe conditions are shown in Figure 2. Participants appear to overestimate the interval between the two tones in the control condition. In the action and observe conditions, participants underestimated the interval between the movement-related events by shortening the perceived interval between an action (self-made or observed) and its consequent tone.

A one-way repeated-measures ANOVA revealed that reproduction errors significantly differed across Conditions,  $F(2, 32) = 13.67, p < .001$ . Tests of simple effects revealed significantly greater negative reproduction errors (shorter estimated intervals) for the action condition ( $-125$  msec) compared with the control condition (84 msec,  $p = .001$ ) and for the observe condition ( $-99$  msec) compared with the control condition ( $p = .006$ ). No significant difference in reproduction errors was found between the action and observe conditions. These results indicate that, in both the action and observe conditions, participants perceived the interval between the key press and tone to be significantly shorter than the interval between two tones.

### *EEG Results*

*Preaction potentials.* In grand-averaged preaction potentials, a slow-wave negativity in frontocentral electrodes



**Figure 2.** Mean reproduction error (and SEM) in msec, calculated as the difference between response interval and stimulus interval, across control, self-made action, and observed action conditions. In control conditions, participants overestimated the interval between two sensory events, whereas in action and observe conditions, they underestimated the interval between a self-made or observed movement and a sensory event.

was clearly evident before the onset of self-made actions (Figure 3). However, no such rising negativity was found before first tone onset and observed action.

A three-way repeated-measures ANOVA showed significant main effects, revealing differences between ampli-

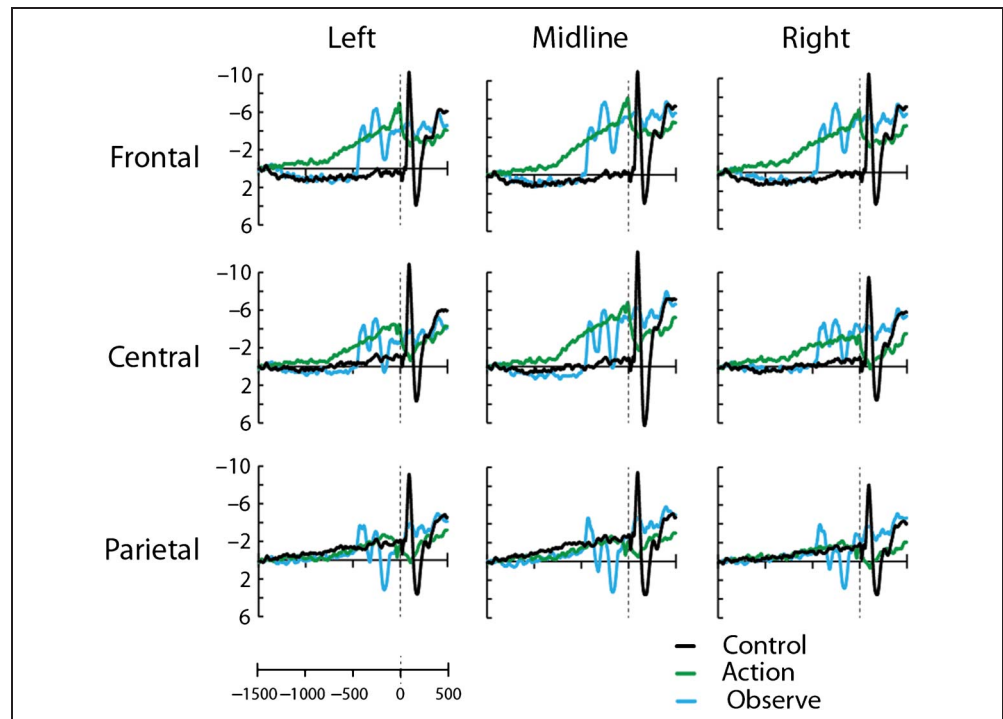
tudes of preaction potentials across Conditions,  $F(2, 32) = 6.00, p = .006$ , Electrode groups,  $F(8, 128) = 5.06, p < .001$ , and Time windows,  $F(5, 80) = 3.91, p = .003$ . A significant three-way interaction between Conditions, Time windows, and Electrode groups was also found in amplitudes for the preaction potential,  $F(80, 1280) = 10.31, p < .001$ .

To investigate these amplitude differences between conditions and electrode groups, we conducted follow-up two-way ANOVAs for each time window. Two-way interactions between Condition  $\times$  Electrodes were found across all time windows (for all comparisons  $p < .001$ ). Overall, comparisons of simple effects (using a Bonferroni correction) revealed significantly greater negative activity for the action condition compared with observe and control conditions. This difference was first evident in frontal electrodes at 1400 msec before self-made movement and later spread to central electrodes from 1100 to 500 msec (all  $ps < .05$ ).

As can be seen in Figure 3, this rising negativity continues on in the movement condition until movement onset. We did not further analyze the conditions after the 500-msec time window because of interference between the visual evoked potential elicited by the video of the observed condition.

Our results indicate a rising slow-wave negative potential (RP) in movement conditions; however, no such similar potential (CNV) was found in either tone or observed action conditions (before the video onset). These results suggest that the binding found when observing and executing an action is not directly indexed by preaction/stimulus activity in frontocentral electrodes.

**Figure 3.** Grand-averaged preaction potentials across a grid of 3 (left, midline, right)  $\times$  3 (frontal, central, parietal) groups of electrodes showing slow-wave potentials from 1500 msec prior till the onset of a tone (black), self-made button press (green), and observed button press (blue). Negative amplitudes are plotted upwards.



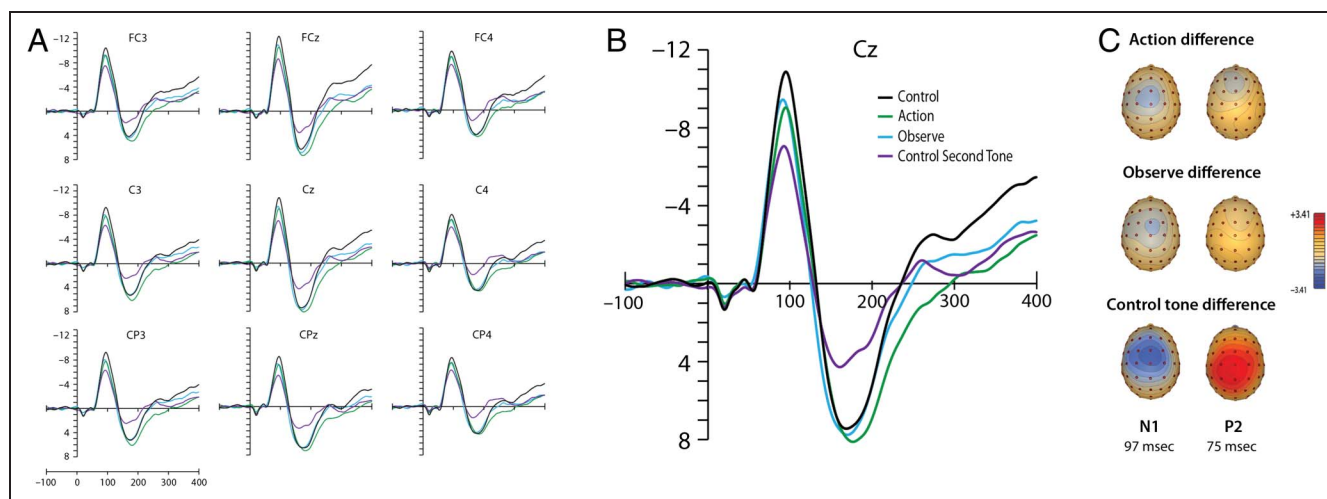
**Auditory ERPs.** Grand-averaged auditory ERPs are shown in Figure 4. The N1 component, a negative deflection peaking at 94 msec after tone onset, is evident across all conditions and is largest for all conditions in midline frontocentral electrodes. The amplitude of the N1 appears to vary between conditions. The P2 component, a positive deflection peaking at 175 msec after tone onset, is also evident across all conditions and appears to vary between the control tone condition and the remaining conditions. We further investigated these amplitude differences in each component, using two separate ANOVAs.

A two-way repeated-measures ANOVA showed significant main effects, revealing differences in amplitude for the N1 component across Conditions,  $F(3, 48) = 19.10$ ,  $p < .001$ , and Electrodes,  $F(8, 128) = 63.90$ ,  $p < .001$ . A significant two-way interaction between Condition and Electrode was found between amplitudes for the N1 component,  $F(24, 384) = 6.13$ ,  $p < .001$ . To investigate these differences in amplitudes between the conditions, we conducted pairwise comparisons (using a Bonferroni correction) between conditions for each electrode. For the N1 component (peak = 97 msec) frontocentral electrodes located on the left (FC3, C3) and middle of the scalp (FCz, Cz, and CPz) all showed the same pattern of results. Across these electrodes, the first tone in the control condition had significantly greater negative amplitudes compared with all the other conditions (all  $ps < .05$ ). The tones presented in both the observe and action conditions were also greater in amplitude than the second tone in the control condition (all  $ps < .05$ ). Across the right side electrodes (FC4, C4, CP4), no difference in amplitude was found between the first tone and tones

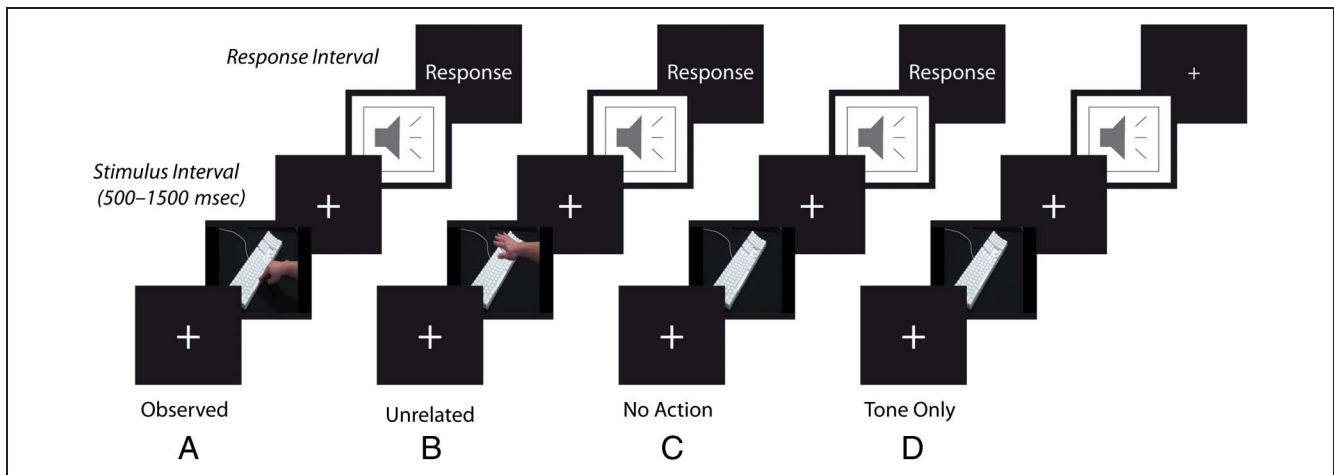
in both the observe and action conditions; however, there was still a difference between first tone and second tone in the control conditions across these electrodes ( $p < .05$ ). In electrodes CP3, FC4, and C4, the amplitude in observe conditions was greater than that for the second tone in the control condition ( $p < .05$ ), whereas in CP4 it was the amplitude in the action condition that was significantly greater than the second tone in the control condition ( $p < .05$ ). Across all electrodes, no difference was found in amplitude for the N1 component between observe and action conditions.

For the P2 component (peak = 175 msec), a two-way repeated-measures ANOVA significant main effects, revealing differences in amplitude across Conditions,  $F(3, 48) = 24.07$ ,  $p < .001$ , and Electrodes,  $F(8, 128) = 47.57$ ,  $p < .001$ . A significant two-way interaction between Condition and Electrode was found between amplitudes for the P2 component,  $F(24, 384) = 3.46$ ,  $p < .001$ . Bonferroni-corrected pairwise comparisons between conditions for each electrode showed the same pattern of results. The second tone in control conditions had a significantly smaller positive amplitude than all other conditions (first tone, action, and observe;  $p < .05$ ). No significant differences were found between first tone, action, or observe conditions in any electrode.

Our results indicate that presentation of a first tone results in a large N1, which decreases in amplitude when tones are the consequent of a previous event. Both executing and observing an action before the onset of a tone results in a reduction of the amplitude of the N1, but not to the same extent as a tone that was preceded by another tone. The N1 suppression in action and observe conditions



**Figure 4.** (A) Grand-averaged auditory ERP from a grid of central electrodes for first (control) tone, action tone, observe tone and second (control) tone events. (B) Grand-averaged auditory ERP for Cz electrode showing the N1–P2 components for each condition. Between all of the conditions, the tones that were first presented in the control task had the greatest N1 amplitude, with action and observe tones showing suppression of the N1 (decrease in N1 amplitude), and second (observe) tones with the smallest N1 amplitude. (C) Topography maps showing mean amplitude differences for the N1 (left) and P2 (right) component between the first tone in the control task and the action caused tone (top), the observed caused tone (middle), and the second tone in the control task (bottom). Negative differences (blue) show that there were similar amplitude differences in the N1 component for both observe and action tones. This difference occurs predominantly over frontocentral electrodes located around the midline and toward the left side of the head.



**Figure 5.** Interval estimation task for Experiment 2. Participants were presented with a video clip showing a key depression that was caused by either an observed action (A), paired with an unrelated action (B), or occurred with no action (C). After a pseudorandom interval (500–1500 msec), an auditory tone was presented and participants were required to estimate the length of time between the key press and the consequent tone. In a tone-only control condition (D), the image of the keyboard was presented on screen, and a single auditory tone was presented after a variable interval.

is predominantly over midline and left electrodes, suggesting a relationship between auditory processing of the tone and the hand of action or, in the observed video, the visual field where the hand appeared (right side). These results suggest that, when a tone is presented as a consequence of an action (self-made or observed), the neural response to the tone is reduced. Crucially, this amplitude reduction is the same regardless of whether actions are self-made or observed.

## EXPERIMENT 2

### Methods

#### Participants

Twenty-four right-handed participants (five men, age range = 19–26 years, mean = 20.95 years) with no known neurological disease or psychiatric conditions provided written informed consent to participate in this event-related EEG experiment. Two data sets were rejected for failing to reach the threshold for included trials following the EEG artifact rejection ( $\pm 120 \mu\text{V}$ ). All analyses were conducted on the remaining 22 data sets. The study was approved by the University of Queensland medical research ethics committee (UQ Project No. 2008000703).

#### Procedure

To investigate the N1 suppression found in observed actions more closely, a control experiment was conducted using visual–auditory events. Participants completed a similar interval-estimation task as in Experiment 1, but comparing four different types of visual–auditory stimuli

(Figure 5). In the control condition (tone only), participants were presented with an image of a keyboard (Frame 1). After a pseudorandom time interval (500–1500 msec), a single auditory tone was presented and no response was required. This condition was included to elicit an ERP response related to tones in the absence of actions (equivalent to the first tone of the control condition in Experiment 1). In the remaining three conditions, participants were presented with a video clip (presented as a set of 38 bitmap images presented in rapid succession) showing a key depression that was caused by either an agent, no agent, or paired with an unrelated action.

The observed action condition was identical to the observed condition of Experiment 1. In an unrelated action condition, a hand appeared from the right side of the screen at image (Frame 5), moved approximately 15–20 cm above the keyboard without touching it, and returned to exit the screen (Frame 36). While this action was occurring, the space bar on the keyboard depressed in a similar way to the key press in the observed action condition (Frame 19). In the no action condition, the image of a key depressing occurred by itself with no hand or other agent's action present at any time. For each condition, an auditory tone was presented after a pseudorandom interval between 500 and 1500 msec after the image of the key depression was shown. At the end of the trial, participants were required to recreate the stimulus interval between the key press and tone by holding down the space bar for the length of time they perceived the interval to have lasted, identical to Experiment 1. Two trials of each condition were presented in a random order in each block, with a total of 108 trials per condition presented in the entire experiment. All other stimuli were identical to the previous experiment.



## EEG Acquisition and Analysis

EEG recordings and analyses were conducted using the same software and hardware as outlined in the previous experiment. The raw EEG data were referenced to the average reference for all conditions and electrodes. Blink artifacts were corrected for each participant using a filter, based on spatial topography (Berg & Scherg, 1994), with low-pass (45 Hz) and high-pass (0.1 Hz) filters applied to each participant's data set. Auditory ERP epochs started at 500 msec before and went through to 1000 msec after tone onset. Baseline correction was conducted on a window between 100 and 0 msec before tone onset. Epochs were rejected if they contained artifacts caused by poor signal or movement (artifact rejection threshold:  $\pm 120 \mu\text{V}$ ), with each data set needing to contain at least 75 artifact-free epochs per condition. Two participants' data were excluded for not reaching these criteria.

Grand averages of the auditory ERP were created across each condition. The N1 component was analyzed by taking the average of three central midline electrodes (Fz, FCz, and Cz). These midline electrodes were selected as they centered around FCz, which was the electrode with the peak amplitude from the previous experiment. N1 amplitudes were calculated as the mean amplitudes over a 30-msec time window centered around the peak in the grand average, from 85 to 115 msec. The amplitude of the N1 component for the observed action condition was then compared, using Bonferroni (multiple comparison)-corrected  $t$  tests, to each of the other conditions. This analysis specifically tested whether N1 suppression for the consequent auditory event was greater when the key press was caused by an agent executing a goal-directed action rather than the key depression alone or when paired with an unrelated action.

## Results

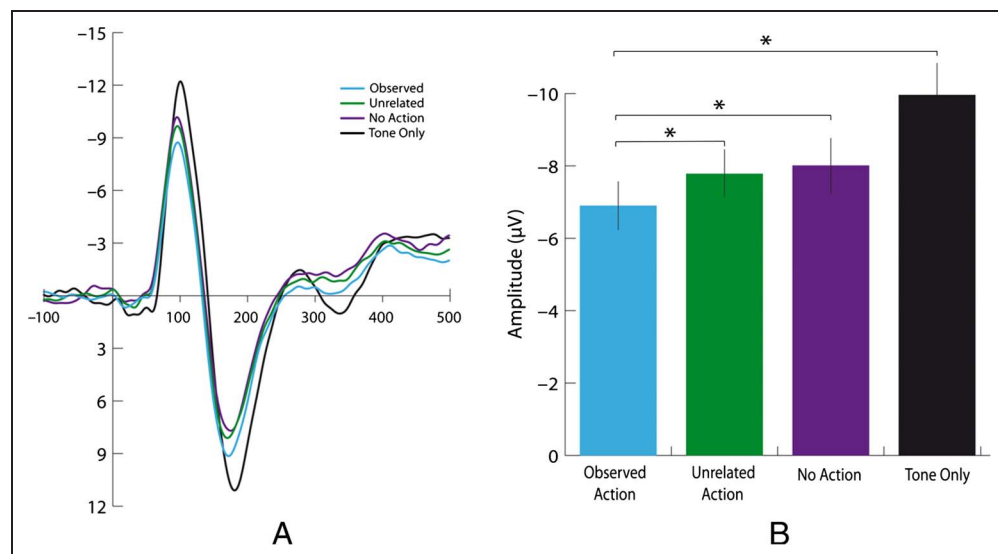
Grand-averaged auditory ERPs for electrode FCz are shown in Figure 6A, and the amplitude of the N1 compo-

nent is shown Figure 6B. A set of pairwise  $t$  tests were used to compare the amplitude of the N1 component in the observed action condition to all other conditions. The N1 amplitude was significantly lower (greater suppression) for the observed action condition ( $-6.9 \mu\text{V}$ ) compared with the tone-only condition ( $-9.95 \mu\text{V}$ ,  $t(20) = 3.88$ ,  $p = .001$ ), the no action condition ( $-8.01 \mu\text{V}$ ,  $t(20) = 3.38$ ,  $p = .003$ ), and the unrelated action condition ( $-7.78 \mu\text{V}$ ,  $t(20) = 2.70$ ,  $p = .014$ ). These results clearly indicate that N1 suppression to consequent tones is greatest for tones caused by observed goal-directed actions of another agent, rather than similar visual events that do not involve causal or goal-directed actions.

## DISCUSSION

In this study, we aimed to investigate the neural processes that index the causal attribution between actions and effects in both self-made and observed actions. To do this, we measured EEG activity while participants performed an intentional binding task. We found greater reductions in the estimated interval between actions and effects for both types of action, demonstrating intentional binding for our own and others' actions. Our results demonstrated N1 suppression over left frontocentral and midline sites for auditory stimuli that were preceded by either type of action (self-made or observed), with no significant difference in N1 amplitude reduction for tones caused by our own and others' actions. We also found a slow-rising negativity (RP) over frontocentral electrodes before the execution of a self-made action; however, we failed to find a slow-rising negativity indicative of anticipation of a stimulus (CNV) in either observe or control conditions. In a second study, we also found that N1 suppression was greatest for tones that were caused by an observed agent executing a goal-directed action, compared with similar visual events that did not involve causal or goal-directed actions. Our results

**Figure 6.** (A) The auditory ERP at FCz for each condition. Suppression of the N1 component amplitude was evident for all key press conditions compared with the tone-only condition (black). (B) Mean N1 amplitudes across conditions. Overall, the lowest N1 amplitude (greatest suppression) was found in the observed action condition (blue) compared with all other conditions (\* indicates significant differences,  $p < .016$ ).



demonstrate that the N1 component is suppressed when an auditory stimulus is preceded by a causal action (either observed or executed), whereas anticipatory activity from frontocentral sites before the onset of an observed action does not appear to be necessary for intentional binding in others' actions and may not directly index the neural processes involved in this phenomenon.

Our main finding shows that auditory consequences caused by observed actions result in N1 suppression that is not significantly different from that caused by our own actions. Using a behavioral measure of implicit agency, we again found that observed actions and self-made actions resulted in a decrease in the perceived interval between actions and consequences, indicative of intentional binding (Poonian & Cunnington, 2013). Our results are supported by previous investigations, which have determined that N1 suppression occurs when tones are judged as caused by ourselves (Gentsch et al., 2012; Gentsch & Schutz-Bosbach, 2011; Kuhn et al., 2011). Our finding that N1 suppression of the auditory ERP occurs when the tone is preceded by observing another person making a goal-directed action suggests that similar cognitive mechanisms are involved in the causal attribution we make between both self-made and observed actions and their sensory consequences. In both self-made and observed actions, there is a growing body of research suggesting that N1 suppression to auditory consequences occurs as a result of predictions about the consequence of the action (Hughes et al., 2013; Gentsch et al., 2012; Stekelenburg & Vroomen, 2012; Bass et al., 2008; van Wassenhove et al., 2005). Taken together, we suggest that the N1 suppression for tones caused by an action (self-made or observed) is because of the implicit feeling of agency that is elicited when making our own actions and when watching another person cause changes to the sensory environment. Our finding is the first evidence that N1 suppression of the auditory ERP occurs to the same extent in conditions where the tone is caused by the self or another and is reflected in a behavioral measure of implicit agency.

It could be argued that the N1 suppression we found in consequences that are caused by an observed or executed action may not be because of predictions within an internal model of action processing, but because of temporal prediction, auditory/visual integration, or repetition suppression. Previous research has found decreased N1 amplitude with tones that are predictable in time (Sowman, Kuusik, & Johnson, 2012; Ford, Gray, Faustman, Roach, & Mathalon, 2007). A recent study investigated this more closely and found that temporal prediction is not the sole reason that N1 suppression occurs when a self-made tone is processed (Lange, 2011). Our study supports this as our consequent tones were at variable times in relation to the first event; however, we still found N1 suppression for temporally unpredictable tones.

It is also possible that the N1 suppression found in our observed condition is a result of the tone being preceded

by a visual stimulus in general rather than specifically by the video of an observed action. Tasks involving audio-visual integration have been found to have different impacts on the amplitude of the N1 in comparison with unimodal auditory ERPs. In some tasks, the N1 is greater for auditory ERPs when preceded by a visual event (Giard & Peronnet, 1999), whereas in other tasks no difference is found in early components of the auditory ERP when auditory and visual stimuli are presented together (Senkowski, Saint-Amour, Kelly, & Foxe, 2007). Recently, N1 suppression was found when an auditory event was preceded by a visual event, but not when there was a delay in synchronicity between auditory and visual events (Vroomen & Stekelenburg, 2010), suggesting that N1 suppression in visual-auditory tasks is because of a causal relationship between the auditory-visual stimuli. We conducted the second experiment to investigate this more closely by comparing the amplitude of the N1 auditory ERP when the tones followed different types of visual stimuli. We found that the greatest N1 suppression occurred for auditory consequences that followed a key press caused by another agent executing a goal-directed action, compared with a key press paired with an unrelated action or with no action. We suggest that the N1 suppression found in tones caused by an observed action is related to the presence of a visual, biological motion stimulus executing a goal-directed action that results in an auditory event.

In regard to the second tone in control conditions, this auditory ERP resulted in large decreases in amplitude for both the N1 and P2 component in relation to the other tone conditions. We believe that the suppression of the consequent tone in control conditions is because of repetition suppression effects, which occur when cortical responses are decreased for repeated stimuli (Costa-Faidella, Baldeweg, Grimm, & Escera, 2011; Ulanovsky, Las, Farkas, & Nelken, 2004). In our control condition, the first and second tones were identical in frequency, which would explain why we found such a large decrease in amplitude for both the N1 and P2 components. This decrease in the P2 component was not found for tones caused by either action condition. Because of the differences in amplitude and electrode locations we measured between action-related tones and consequent tones in the control task, we believe that the suppression found in the second tone in the control task involves different mechanisms to those for action-related tones. Overall, comparing our results with previous research leads us to conclude that the N1 suppression for the action-related tones is not because of temporal predictability, multimodal integration, or repetition suppression. We instead propose that the N1 suppression of action-related tones is evidence of top-down predictions about whether an action results in a consequent tone, resulting in reductions in auditory cortical firing.

In addition, we did not find any P2 suppression for tones that were preceded by an action. It has been hypothesized that reductions in P2 amplitude are because of

conscious processing of the action-related sound (Knolle et al., 2013). Results vary in whether self-initiated sounds result in suppression of the P2 component (Knolle et al., 2013; Horvath, Maess, Baess, & Toth, 2012; Knolle, Schroger, Baess, & Kotz, 2012; Sowman et al., 2012) or not (Bass et al., 2008; Martikainen et al., 2005). We found no P2 suppression for both types of action-related tones, similar to other studies that found no difference in P2 amplitude to tones caused by self-made actions (Bass et al., 2008; Martikainen et al., 2005). It may be that because of our task being a measure of implicit agency, the differential effects were only found on the earlier N1 component. It is also possible that later processes involved in the conscious detection of agency may have differential effects on action tones and observed tones. In fact it appears that the judgment of self-agency does affect later components such as P3a but not the N1 (Kuhn et al., 2011). Recent work has also found that auditory events caused by another agent are perceived differently, with less perceptual attenuation, than auditory events caused by the self (Weiss & Schutz-Bosbach, 2012; Weiss, Herwig, & Schutz-Bosbach, 2011). In our results, we only investigated early ERP components, indicative of the attribution of agency between movement and sensory events. Our results suggest that the attribution of agency is generalizable not just to ourselves but also to other biological agents, as well as providing further support that early components of the sensory ERP relate to implicit attributions of agency.

We also examined activity leading up to the onset of an action, which is thought to provide evidence for anticipatory processes during the execution and observation of an action. We found no slow-rising negativity before an observed action, suggesting that preaction potentials, either a RP or a CNV, do not index intentional binding. We found no anticipated activity before the onset of the observed video, although the expected time of the video was at a fixed interval, it may be that the pre-SMA plays a differential role in binding of self-made actions to their consequences and observed actions to their sensory consequences. As intentional binding is evident in both self-made and observed actions, it may be that pre-SMA is part of a larger network involved in the awareness of our actions. Our results may also indicate that predictions for our own and observed actions involve different processes for binding an action to a sensory consequence and thus involves different anatomical areas. This action awareness network may include cerebellum and parietal cortex, areas that are thought to be involved in the prediction about intentions for both self-made and others (Knolle et al., 2012; Desmurget & Sirigu, 2009). Taken with our findings, we suggest that preaction activity may not be an index of the binding that occurs in general between actions and their consequences but may be specific for binding between self-made actions and their effects.

Together, our results suggest that the sensory consequences of observed actions and self-made actions are processed in a similar way. We found that both self-

made and observed actions involve implicit agency, with similar levels of binding, and result in similar effects on the neural response to the sensory consequence of the action. In a control experiment, we further showed that N1 suppression occurs for sensory consequences that are caused by another agent executing a goal-directed action. In contrast, activity before the onset of actions did not correspond with binding found in observed actions. We suggest that the N1 suppression during executed and observed action-effect tasks is because of top-down processes and, in particular, predictions about the consequences of an action within an internal comparator model. These expectations about events help us unconsciously make sense and predict the consequences of our own and others' interactions with the world.

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