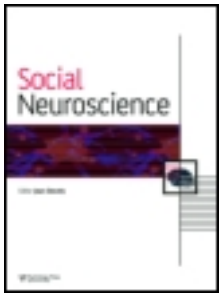

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Observed and self-experienced conflict induce similar behavioral and neural adaptation

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Observed and self-experienced conflict induce similar behavioral and neural adaptation

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In adapting our behavior to a rapidly changing environment, we also tune our behavior to that of others. To investigate the neural bases of such adaptive mechanisms, we examined how individuals adjust their actions after decision-conflicts observed in others compared to self-experienced conflicts. Participants responded to the color of a stimulus, while its spatial position elicited either a conflicting or a congruent action. Participants were required either to respond to stimuli themselves or to observe the response of another participant. We studied the difference between interference effects following conflicting or congruent stimuli, an effect known as conflict adaptation. Consistent with earlier reports, we found that the implementation of reactive control, following congruent trials, was accompanied by activation of the right inferior frontal cortex. Individual differences in the efficacy of response inhibition covaried with the level of activation in that region. Sustaining proactive control, following incongruent trials, activated the left lateral prefrontal cortex. Most importantly, adaptive controls induced by decision-conflicts observed in others, as well as the associated prefrontal activations, were comparable to those induced by self-experienced conflicts. We show that in both behavioral and neural terms we adapt to conflicts happening to others just as if they happened to us.

Keywords: Action observation; Conflict adaptation; Model-driven fMRI; Reaction time distribution analysis; Proactive control; Reactive control.

In our everyday life, we have to adapt our behavior to an ever-changing environment, importantly including the actions of other people. One way to study the underlying mechanism of adaptive behavior is by using conflict tasks. In conflict tasks such as the Simon task, either high- or low-conflicting information is presented to the participant. A typical observation is that reaction times (RT) are prolonged in high- compared to low-conflicting trials; this is known as the interference effect. However, the size of the interference effect depends not only on the nature of the current trial but also on the nature of the previous trial. The influence of such context-dependencies on the interference effect in conflict tasks has been investigated in various behavioral and neuroimaging studies (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Gratton, Coles, & Donchin, 1992; Kerns, 2004, 2006; Ullsperger, Bylsma, & Botvinick, 2005; for an overview, see Egner, 2008). We can make use of this finding to study social cognition by investigating these sequential

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effects following the observation of behavior, rather than following the behavior itself.

In general, the crucial observation is that following high- compared to low-conflict trials the interference effect is reduced (Gratton et al., 1992). This reduction has also been referred to as the conflict adaptation effect, or Gratton effect. A common explanation for these trial-to-trial adjustments is provided by the conflict monitoring model (Botvinick, 2007; Botvinck, Braver, Barch, Cater & Cohen, 2001; Verguts & Notebaert, 2008). According to this model, the detection of conflict on one trial triggers the implementation of additional cognitive control, resulting in reduced interference effects on the subsequent trial. If such additional control processes are already active by the time the next high-conflict trial starts, interference has less effect than after low-conflict trials where no additional control processes were proactively engaged. As such, we can distinguish two forms of control. On the one hand, we have reactive control, which is the inhibition of the irrelevant response dimension on the current trial. Effectively, this is the ability to cope with conflicting information on the current trial, without relying on prior mechanisms. On the other hand, there is proactive control, or conflict adaptation, which can be described as the inhibition of task-irrelevant information following a conflict (Braver, Paxton, Locke, & Barch, 2009; De Pisapia & Braver, 2006; Ridderinkhof, Forstmann, Wylie, Burle, & van den Wildenberg, 2011). Note that proactive control does not entail a task-general increase of control, but rather a task-specific inhibition of the irrelevant dimension of the current task (Hyafil, Summerfield, & Koechlin, 2009). These reactive and proactive control mechanisms can vary across subjects or states; for instance, they are affected by emotional stimuli and trait anxiety (Krug & Carter, 2010). These two forms of control are associated with different cortical areas, and we can study individual differences in the efficacy of these types of control. Effective proactive control has been shown to covary with increased left lateral prefrontal cortex (lPFC) activation (Kerns, 2006). On the other hand, we see specific activation in the right inferior frontal cortex (rIFC) during reactive control. The strength of this activation covaries with individual measurements of reactive control (Forstmann et al., 2008a; Forstmann, van den Wildenberg, & Ridderinkhof, 2008b).

To help extend these findings to our social experiment, we make use of the simulation account (Decety & Grèzes, 2006; Gallese, Keysers, & Rizzolatti 2004; Iacoboni et al., 2005; Press, Cook, Blakemore, & Kilner, 2011; Ramnani & Miall, 2004). According to the simulation account, the observation of another person's behavior induces internal states in the observer that are similar to those that would occur if the observer undertook the action himself. Interestingly, these internal states are also evoked if the other's actions are not directly observed, but heard or suggested instead (Kohler et al., 2002; Umilta et al., 2001). These findings suggest that it is not simply perceiving an action that evokes the same state, but rather knowing that it takes place. Although such simulation effects were initially reported for specific motor acts, they have also been found in more cognitive task aspects (de Bruijn, Miedl, & Bekkering, 2008; Sebanz, Knoblich, & Prinz, 2005; Sebanz, Knoblich, Prinz, & Wascher, 2006; Tsai & Brass, 2007; Tsai, Kuo, Hung, & Tzeng, 2008; van Schie, Mars, Coles, & Bekkering, 2004; Winkel et al., 2009). Interestingly, these effects are present when the subject believes she is observing human behavior, but not when she believes she is observing computer-generated behavior (Tsai et al., 2007, 2008). Such simulation is not necessarily automatic, and in some competitive task-contexts it can be disadvantageous (de Bruijn et al., 2008). Moreover, the simulation is not dependent upon the relevance of the other's information for the current task. Subjects show effects depending on the other's response even when it has no real relevance to the subject's own task (Sebanz et al., 2005).

The present study set out to investigate trial-to-trial adjustments, using a social Simon task, where part of the trials are not performed, but observed. We previously showed that comparable conflict adaptation is implemented when participants observe someone else performing the task as when they perform the task themselves. That is, conflict adaptation occurs even after we observe someone else in a high-conflict situation (Winkel et al., 2009). In the present paper, we will investigate whether observed and self-experienced conflict lead to the same neural correlates of control; that is, whether we can find similar neural correlates associated with proactive and reactive control following the presence or absence of observed conflict, as opposed to experienced conflict. Because we will be referring to a number of sequential trial types, we will adhere to the abbreviations we used before to describe the same paradigm. In these abbreviations, lower-case letters refer to the trial type of the preceding trial (s/o for "self" vs. "other"—c/i for congruent vs. incongruent). Upper-case letters refer to the trial type of the current trial (C/I for congruent vs. incongruent).

To index the efficacy of changing and adapting behavior after performing oneself or observing someone else, we zoom in on individual differences in reactive control. While other fMRI studies of adaptive control were limited to investigating mean RT in

the Simon task (e.g., Kerns, 2006), we used the delta plot method. Delta plots use quantile bins to quantify the reaction time distributions, providing more fine-grained information about the within-trial development of inhibition processes (for a review, see van den Wildenberg et al., 2010). In particular, the slope of the latest segments of the delta plot is associated with the fully developed reactive control (Forstmann et al., 2008a, 2008b). Subjects with lower (or more negative going) slope values exhibit stronger reactive control. We use this individual measure of reactive control as a covariate in our neuroimaging analyses (see the section "RT distribution analysis" for a more elaborate explanation).

Based on this setup, we formulate three hypotheses. Two of these relate to proactive and reactive control in general, and the third and most relevant hypothesis relates to the simulation of the first two during action observation.

First, we expect that following a high-conflict trial proactive control will be high. Previous research has shown activation in the left lPFC for effective proactive control, so we may expect to find such an activation for iI > iC trials. Because proactive control is already present during these trials, there will be less response capture early in the trial, and less need for reactive control as the trial progresses. As a result, we should not observe an increase in rIFC activation for iI > iC trials. Also, because proactive control is present before trial onset and not implemented during a trial, we should not see buildup of within-trial inhibition as displayed in the delta plots.

Second, after a low-conflict trial, there is little proactive control, so we do expect that reactive control will be engaged during conflict. This means that activation in the rIFC for cI > cC trials should covary with the individual behavioral efficacy in implementing within-trial (reactive) response inhibition. We measure this efficacy by the negative slope of the late segments of the delta plots (cf. Forstmann et al., 2008a, 2008b). To summarize, we expect to find a negative correlation here; the more negative-going the delta-plot slope is for a particular subject, the higher we expect the rIFC activation to be.

The above predictions relate to the study of conflict adaptation and proactive versus reactive control in general. Our third hypothesis relates specifically to our social paradigm and the simulation account. We expect that both in terms of behavior and in terms of neural activations, we will see similar adjustments after subjects perform the task themselves, and after they observe someone else perform the task. That is, we expect to see the same behavioral and neural signs of proactive control after observing a conflicting trial, and of reactive control after observing a non-conflicting trial.

MATERIALS AND METHODS

Participants

21 healthy volunteers were recruited. We obtained written consent from all participants prior to the experiment. The experiment was approved by the local ethics committee of the University of Amsterdam, and all procedures were conducted in accordance with the Declaration of Helsinki, as well as institutional guidelines. All subjects had normal or corrected-to-normal vision. No subject had a history of neurological, major medical, or psychiatric disorder, according to self-report. The data of four subjects were excluded from analysis due to excessive movement artifacts ($n = 1$) or high error rates ($n = 3$). The remaining 17 subjects were 9 females and 8 males (age: $M = 22.9$, $SD = 1.8$ years) who were all right-handed as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971).

Instructions, other player, and debriefing

Prior to scanning, participants were seated in an experimental room next to the scanner, where they were introduced to the Simon stimuli and the stimulus–response mappings, and allowed to practice the task for 10 min. The subjects were shown a picture and the name of a person. They were told that this person had already taken part in the experiment on an earlier occasion, and that the subject would observe the other person's recorded responses intermixed with their own trials during this session. We told the subjects that their own responses would also be shown to a subsequent subject. We then took the subjects' picture, telling them it would be shown to the next subject, who would observe their responses. The (fictive) name of the other person was matched in length to the name of the participant, and genders were counterbalanced.

The participants were instructed to observe the behavior of the other person. We ensured that participants paid attention to the other person's behavior by introducing catch trials. In these catch trials, the other person's response was incorrect. Participants had to indicate with their left middle finger that they detected an erroneous response from the other person. After the experiment, participants were asked to rate on an analog scale both the skill level of the observed

behavior and the extent to which they felt they were observing another person. Finally, we asked whether they believed that they were really watching another person's behavior.

Behavioral task

We used a modified version of the Simon task (Simon, 1967) without stimulus repetitions (Figure 1). The Simon target stimuli consisted of a green, red, blue, or yellow circle presented to either the left or right of a central fixation cross. Participants were instructed to press the left button to green or red stimuli, and the right button to blue or yellow stimuli, with the index fingers of the left and right hand, respectively. Stimulus color (green/red vs. blue/yellow) was the relevant aspect of the signal, while the spatial location (left vs. right) was the irrelevant dimension. On congruent trials, the responses were spatially compatible with the position of the target (for instance, a green circle designating a left button press and appearing on the left side of the screen). On incongruent trials, the responses were spatially incompatible with the target location (for instance, a yellow circle designating a right response but appearing on the left). The distance between the fixation cross and the stimulus covered a visual angle of 2.8°.

On each trial, the presentation of the stimulus was preceded by a cue, indicating whether the subject was to perform this trial herself, or to observe prerecorded behavior (Winkel et al., 2009). In the "self" condition, participants received their own name presented in the middle of the screen. Their name signaled that they had to press a response button as soon as one of the colored Simon stimuli was presented. In the "other" condition, participants received the name of the other player. This name signaled that participants had to observe the behavior of the other person and were not required to press a response button when they saw one of the colored Simon stimuli. The trials comprised 8 types ($2 \times 2 \times 2$), as determined by the player of the previous trial (s/o), the congruency of the previous trial (c/i), and the congruency of the current trial (C/I). As there is no response during the observed trial, these trials do not generate behavioral data. We therefore only examine trials where the subject performs the trial herself, labeled "self" if it was preceded by another of self performed trial, or "other" if it was preceded by an observation trial. The sequence of trial conditions was produced pseudo-randomly, with the constraint that each of the eight trial types occurred equally often. Moreover, consecutive trials never used the same color, to avoid stimulus-repetition effects and hence preclude an interpretation of the Gratton effect in terms of repetition priming (Wühr & Ansorge, 2005;

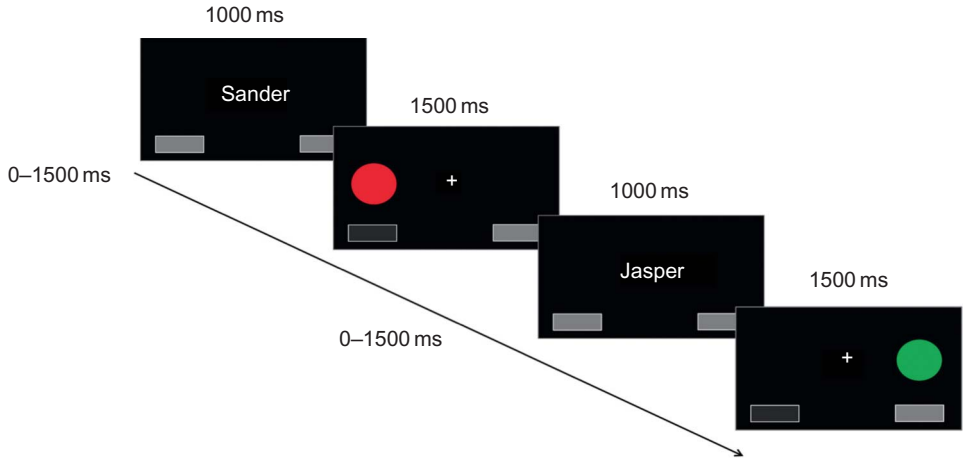

Figure 1. Schematical drawing of the Social Simon task. Each trial started with the presentation of a name cue. In the "self" condition, participants saw their own name presented on the middle of the screen. In the "other" condition, participants received the name of someone else. This name signaled that participants just had to observe the behavior of this person, instead of responding themselves, when the Simon stimuli were presented. In both the "self" and the "other" conditions, participants viewed two response buttons that were presented below the colored Simon stimuli. According to the response button that was pressed by the participants themselves, the corresponding virtual response button was highlighted. In the "other" condition, one of the virtual response buttons was also highlighted indicating the other person's response. Four different colored circles were used that were presented either on the right or left side of the central fixation cross. Green and red circles were mapped to a left response button press, whereas yellow and blue circles were mapped to a right response button press yielding either congruent—i.e., color and spatial location overlap—or incongruent conditions—i.e., color and spatial location do not overlap. The sample trial sequence displayed here is an ocI type trial, meaning that the subject (Jasper) first observes another's congruent trial, and then performs an incongruent trial himself.

also see Cho, Orr, Cohen, & Carter, 2009). To allow for this, two colors were mapped to each response direction. The other's RT was drawn from a uniform distribution between 220 and 460 ms.

In both the "self" and the "other" condition, participants viewed two response buttons that were presented below the colored Simon stimuli (see Figure 1). When the response button was pressed by the participants themselves, the corresponding visually presented response button was highlighted. In the "other" condition, one of two visually presented response buttons was also highlighted to indicate the other person's response. It is important to note that the participants were instructed not to respond during these trials except when observing an error.

All stimuli were presented on a back-projection screen that was viewed via a mirror system attached to the magnetic resonance imaging (MRI) headcoil. The timing of the sequence of trials was triggered from the MRI control every 4 s. The trials started with a variable oversampling interval of 0, 500, 1000, or 1500 ms to obtain an interpolated temporal resolution of 500 ms. A fixation cross was presented during the variable oversampling interval. Participants were asked to maintain fixation. The name cue was presented for 1000 ms in the middle of the screen. Directly following the name cue, the target stimulus was displayed until the response interval exceeded 1500 ms. During this time, the participants had to respond with their right or left index finger.

The experiment consisted of 296 trials including 20 null events that were pseudo-randomly interspersed, as well as an average of 11 catch trials. Both the "self" and the "other" condition consisted of 138 trials, each with 69 incongruent and congruent trials, respectively. Experimental trials following catch trials and null events were discarded. The null events were included to compensate for the overlap of the blood-oxygenation-level-dependent (BOLD) signal. The functional scanning lasted about 40 min, separated in two experimental blocks. Every block started with two dummy trials that were excluded from the analysis.

RT-distribution analysis of interference effects

We employed the dual-route model of interference effects (e.g., Eimer, 1995; Kornblum, Hasbroucq, & Osman, 1990) in the Simon task, and used the associated RT-distributional analyses to quantify and dissociate the effects of reactive control after a congruent versus an incongruent trial.

Critical to this dual-route model is the observation that fast responses to incongruent stimuli tend to be associated with *below-chance* accuracy (e.g., Gratton et al., 1992), which can be understood as response capture, the initial tendency to respond with the hand corresponding to the direction where the stimulus appears. To overcome direct response capture by task-irrelevant information, many authors have assumed implicitly or explicitly that the response as activated by irrelevant stimulus features is subsequently inhibited (e.g., Eimer, 1999) and aborted (e.g., Logan & Cowan, 1984). Such inhibition is not immediate, but develops over the course of a trial (e.g., Eimer, 1999). Because of this gradual build-up, slower responses will benefit more from selective response inhibition than faster responses (for a review, see van den Wildenberg et al., 2010). Consequently, correct slow responses to congruent stimuli will be less facilitated by the position-driven response capture, whereas correct slow responses to incongruent stimuli will be less delayed. This is especially the case following congruent trials, since the direct location-driven response is still activated; hence, on incongruent trials, a stronger need to inhibit the incorrect response is required. Therefore, a strong reduction of the interference effect after congruent, i.e., low-conflict trials, can be taken as a measure of selective response inhibition. A different data pattern is expected for incongruent trials following incongruent trials, when inhibition is already implemented. In these trials, the interference effect after incongruent trials should already be reduced or even negative depending on the strength of control implemented after high-conflict trials.

Given these dynamics, interference effects decrease by selective response inhibition more strongly after congruent than incongruent trials. They are also decreased more strongly in slow than in fast responses (van den Wildenberg et al., 2010). A good way to capture these dynamics is to use so-called delta plots. We construct these plots by plotting the interference effect as a function of response speed, separated in quantile bins (de Jong, Liang, & Lauber, 1994; Ridderinkhof, 2002; Wiegand & Wascher, 2007). Because reactive control results in a reduction of the interference effect in slow responses (outlined above), the magnitude of the interference effect levels off and *reduces* for slow responses. Because more effective reactive control results in a more pronounced reduction of interference effects in slow responses, as argued above, we can use the negativity of the slope as an index of the effectivity of individual reactive control. Because the gradual buildup takes place during reactive, but not proactive control, we expect to find such a pattern only for trials following

congruent trials (cI-cC), but not following incongruent trials (iI-iC). We can therefore only use this individual difference measure to investigate reactive, but not proactive control.

Bayesian hypotheses testing

For statistical testing between the "self" and the "other" condition, we report Bayesian posterior probabilities in addition to conventional *p* values on the distribution analysis to show that the effect for the "self" and for the "other" condition were identical. When we assume that the null hypothesis and the alternative hypothesis are equally plausible a priori, a default Bayesian *t*-test (Wetzels, Raaijmakers, Jakab, & Wagenmakers, 2009) allows one to determine the *posterior* plausibility of the null hypothesis and the alternative hypothesis. We denote the posterior probability for the null hypothesis as $p^{\text{Bayes}}(\text{H}_0)$. When, for example, $p^{\text{Bayes}}(\text{H}_0) = .9$, this means that the plausibility of the null-hypothesis has increased from.5 to.9. We report these posterior probabilities because they address several problems with conventional *p* values (Iverson, Lee, & Wagenmakers, 2009). Most importantly, posterior probabilities allow one to directly quantify evidence in favor of the null-hypothesis, instead of only "failing to reject" it.

MRI scanning procedure

The fMRI measurements were acquired in a single scanning session on a Phillips Intera 3.0T (Royal Phillips Electronics N.V., Amstelplein 2, Amsterdam, the Netherlands) using a standard SENSE head coil. For the fMRI experiment, 30 axial slices were acquired (220 ˆ 2 mm FOV, 96 × 96 matrix, 3-mm slice thickness, 0.3-mm slice spacing) parallel to the AC–PC plane and covering the whole brain. We used a single-shot, gradient recalled echo planar imaging (EPI) sequence (TR 2000 ms, TE 28 ms, FA 90°, transversal orientation). Prior to the functional runs, a 3D T1 scan was acquired (T1 TFE, 250ˆ2 mm FOV, 256ˆ2 matrix, 182 slices, 1.2-mm slice thickness, TR 9.69, TE 4.6 ms, FA 80°, sagittal orientation).

Functional magnetic resonance analysis

Analysis was carried out by FEAT (FMRI Expert Analysis Tool), Version 4.0, part of FSL (FMRIB's Software Library, www.fmrib.ox.ac.uk/fsl). The first two volumes were discarded to allow for T1 equilibrium effects. The remaining images were then realigned to compensate for small head movements (Jenkinson, Bannister, Brady, & Smith, 2002). Data were spatially smoothed, using a 5-mm, full-width-half-maximum Gaussian kernel. The data were filtered in the temporal domain using a high-pass filter with a cutoff frequency of 1/60 Hz to correct for baseline drifts in the signal. Finally, the functional data were pre-whitened by using FSL (Woolrich, Behrens, Beckmann, Jenkinson, & Smith, 2001).

All functional data sets were individually registered into three-dimensional (3D) space, using the participants' individual high-resolution anatomical images acquired at the beginning of each scanning session. The individual 3D reference data set was used to normalize the functional data into MNI space by linear scaling (affine transformations) (Jenkinson & Smith, 2001). The statistical evaluation was performed with a general linear model. The design matrix was convolved with a synthetic hemodynamic response function and its first derivative. Individual contrast maps were generated separately for the "self" and the "other" condition, as well as averaged across "self" and "other." Moreover, the main effect of congruency (C/I) was split for trials following congruent (cC/cI) or incongruent trials (iC/iI). This was done for the average of the "self" and the "other" condition as well as separately for the "self" and the "other" condition. Higher-level analysis was carried out with FLAME (FMRIB's Local Analysis of Mixed Effects) (Beckmann, Jenkinson, & Smith, 2003; Woolrich et al., 2004).

For the whole-brain analysis of the factor congruency (I > C) as well as the factor observation (s > o) and the interaction between the factor congruency on the current trial (I > C) and congruency on the preceding trial (i > c) separately for the "self" and the "other" condition, we only report cortical regions with a height threshold of $z > 2.3$ and a cluster probability of $p < .05$, corrected for whole-brain multiple comparisons. Due to splitting the data up in sequential and "self"/"other" trial types, the contrasts of congruency on the current trial following incongruent trials (proactive control) and the covariance analysis following congruent trials (reactive control) both had a limited trial count. We therefore constrained our analysis, limiting it to activations in the prefrontal cortex, as informed by our hypotheses. We applied a height threshold of $z > 2.3$ with a cluster extent threshold of 1000 mm^3, corresponding to 34 voxels (Forman et al., 1995).

The aim of the covariance analyses was to test for individual differences in the efficacy to implement within-trial control, in the form of selective response. To this end, the individual contrasts (incongruent vs.

congruent) for trials following congruent trials were subjected to two separate covariate analyses, one for the "self" and one for the "other" condition, to investigate the dynamics of selective response suppression. For each participant, slope values for the different delta-plot segments (also see behavioral analysis) were included as individual covariates. Finally, to verify the functional significance of this analysis, the individual effect size of the overall Simon interference effect was also entered separately as a covariate in the fMRI regression model.

To compute the percent signal change of the hemodynamic response of the rIFC, all significant voxels of the delta plot covariance analysis were determined separately for the "self" and the "other" condition. We then extracted the time course of the signal underlying these activated voxels for each participant from the modeled data. The percent signal change was calculated in relation to the mean signal intensity across all time steps for these voxels. These values were used to illustrate the correlation underlying the covariate analysis (Figure 3).

Contrary to the behavioral analyses, neuroimaging allows us to examine what regions activate during the observation of specific trials during the other player's (observed) behavior. We performed the same analyses of reactive control (cC > iC) and proactive control (iI > iC) on those trials where the current trial is observed instead of performed.

RESULTS

Behavior

Omnibus repeated-measures ANOVA was used for testing factor effects on error rates and RT. Effects of primary interest were the main effects of the factor congruency (I vs. C), the factor previous player (s vs. o), the interaction effect between the factor present congruency and the factor preceding congruency (the Gratton effect), and the three-way interaction between the factors present congruency, preceding congruency, and previous player.

Error rates

Overall, error rates were low with a mean of 6.9%. There was no main effect of the factor present congruency in error rates (Table 1). There was a main effect of the factor observation with 7.92% errors on trials following an "other" trial compared to 5.97% errors on trials following a "self" trial $F(1, 16) = 8.58$, $p = .01$. Error rates did not vary systematically with the two- and three-way interactions Fs < 1. Error rates on catch trials were 30%.

RTs

As expected, a significant two-way crossing-over interaction between the factors present congruency and the factor preceding congruency indicated that the present congruency effect seen after congruent trials is reversed after incongruent trials (the Gratton effect), $F(1, 16) = 6.71$, $p = .05$ (Table 1). As a result of this reversion, the main effect of present trial congruency was canceled. It is important to note that the RTs for "self" trials after performing the task oneself (570 ms) compared to RTs for "self" trials after having observed someone else (575 ms) did not differ $t(16) = 0.0$, $p = 1$, $p^{\text{Bayes}}(\text{H}_0) = .78$. The factor previous player also failed to modulate the effect of the factor present congruency or the interaction between the factors present congruency and preceding congruency. This indicates that the Gratton effect does not differ between "self" and "other" trials.

Finally, to test for attention effects while observing the behavior of someone else, we computed the

TABLE 1
Reaction times and error rates. This table shows the reaction times and error rates for the eight trial types. c or i indicates that the previous trial was congruent or incongruent, while C or I indicates the current trial's congruency

	Reaction Time		*Error rate*	
Trialtype	*Following Self*	*Following Other*	*Following Self*	*Following Other*
cC	571	576	5.66%	9.47%
iC	586	582	6.18%	7.25%
cI	571	580	7.49%	8.13%
iI	558	569	4.54%	6.83%
Average	571	577	5.97%	7.92%

correlation between the difference in conflict adaptation effect following "other" and following "self" trials, on the one hand, and the catch trial performance on the other hand. We hypothesized that if people are less engaged in the other's performance, they would show both lower catch trial performance, and a smaller adaptation effect following the other's conflict. Indeed, there was a significant correlation between the two factors, $R(20) = .527$, $p = .028$ (two-tailed), indicating that participants who attended very closely to the observed behavior also revealed stronger trial-to-trial adjustments following the other's behavior.

RT distribution analyses

Further repeated-measures ANOVAs focused on delta plots to capture the temporal dynamics of the congruency effect after congruent (cI-cC) versus incongruent (iI-iC) trials, for both the "self" and the "other" condition.

Proactive control: Conflict following high-conflict trials

A repeated-measures ANOVA examined delta plots after incongruent trials for the "self" and the "other" condition. Consistent with previous findings, the interference effect did not show changes as a function of response latency, due to the presence of proactive control. Instead, interference effects remained constant across the entire RT distribution (Figure 2). The slopes of these horizontally going delta plots did not distinguish between "self" and "other" trials or between fast and slow RT segments ($F < 1$, $p > .33$).

Reactive control: conflict following low-conflict trials

A repeated-measures ANOVA examined delta plots after congruent trials for the "self" and the "other" condition. The interference effect steadily decreased from 12 ms in the fastest segment to –3 ms in the slowest segment in the "self" condition, and from 6 ms to –6 ms in the "other" condition (Figure 3). In the reactive control condition, delta-plot slopes were generally negative-going ($p = .01$ when tested against a zero slope-value) and did not distinguish between "self" and "other" trials, $t(16) = 0.0$, $p = 1$, $p^{\text{Bayes}}(\text{H}_0) = .84$, or between fast and slow RT segments ($F < 1$, $p > .23$).

Neuroimaging data

fMRI data

An initial set of analyses was augmented with a model-driven fMRI approach to examine the neural underpinnings of trial-to-trial adjustments after action observation compared to after action performance. The first set of analyses focused on the main effect of present congruency (I > C) trials, as well as the conditional main effect of present congruency separately for the "self" and "other" condition. Consistent with our behavioral results, direct comparison of I > C trials did not yield significant activations (for a discussion, see also Forstmann et al., 2008a). No significant results were obtained when comparing I > C trials separately for the "self" condition and the "other" condition either.

A second set of analyses focused on trial-to-trial adjustments: the present congruency effect after an incongruent trial (proactive control: iI > iC), and the present congruency effect after a congruent trial (reactive control: cI > cC). For the former, it was predicted that strong cognitive control should already be implemented pre-emptively, resulting in activation of the left lPFC, but not the rIFC. Also, the effect in the "self" condition (siI > siC) should be comparable to that in the "other" condition (oiI > oiC). The results revealed activation only in the left lPFC, for both the following "self" condition (43 voxels) and the following "other" condition (46 voxels). Note that although the peak activations for "self" and "other" were both in the left lPFC, the activation for the following "self" condition was located more ventrally (–56, 14, 14) compared to the following "other" condition (–44, 18, 40) (Figure 2).

To account for activation elicited after low-conflict trials, a different analysis procedure was used (see also Forstmann et al., 2008a, 2008b). After low conflict, there will be little proactive control. We expect to see the buildup of reactive control within an incongruent trial, relative to a congruent one (cI > cC). Because of this buildup, the individual subjects' slope values of the slowest segment of the delta plot indicates their individual efficacy in reactive control. Using this measure as a covariate, we are able to image its neural correlate. Two separate covariance analyses were performed: for the "self" condition and the "other" condition. For the "self" condition, the results revealed increased activation in the rIFC (50 voxels) with decreasing slope values in the slowest segment of the delta plots (see also Forstmann et al., 2008a, 2008b). Although the covariance analysis did not

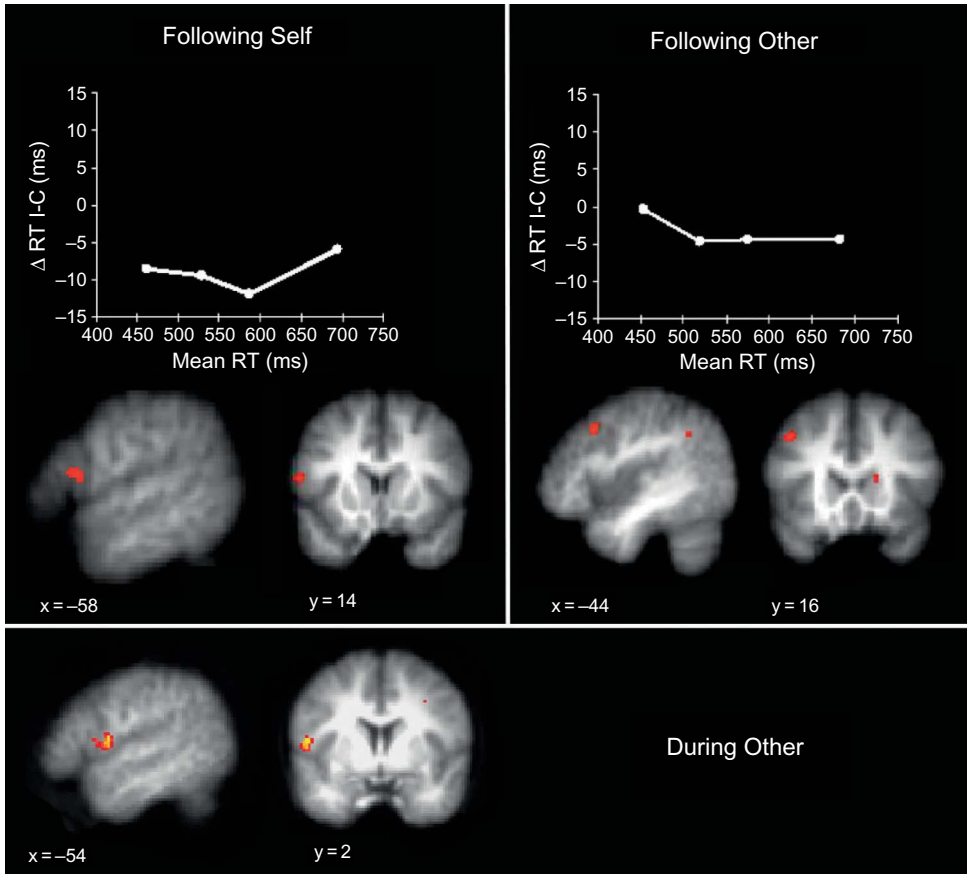

Figure 2. Sustained proactive control. The two graphs depict the delta plot separately for the "self" condition and the "other" condition after incongruent trials. As can be seen from the plots, no temporal dynamics were observed. Below the graphs, the main contrasts between incongruent versus congruent trials after an incongruent trial separately for the "self" condition and the "other" condition are depicted. The bottom panel shows the same contrasts during the other's trial. Shown is the averaged activation across 17 participants rendered onto a template brain ($z > 2.3$, cluster > 34 voxels). The only significant prefrontal activations are obtained in the left lPFC (following self: $x = –56$, $y = 14$, $z = 14$; following other: $x = –44$, $y = 18$, $z = 40$; during other: $x = –54$, $y = 2$, $z = 10$). Coordinates are given in MNI space.

reach significance for the "other" condition, when we defined a region of interest (ROI) based on the rIFC region we found in the "self" condition, we did find a significant correlation between the activation there in the following "other" condition, and the delta plot slopes following the "other" ($R = –.40$, $p = .05$) (see Figure 3).

This finding is in line with previous results reflecting stronger inhibition with a decreasing interference effect with longer RTs. Most importantly, the result is again comparable between the "self" and the "other" condition (Figure 3), as revealed in similarly negative correlations between the BOLD signal change derived from the rIFC and the individual slope values of the slowest delta-plot segment in "self" and "other" conditions. The similarity between the two conditions is confirmed by Bayes statistics: $t(16) = 0.38$, $p = .71$, $p^{\text{Bayes}}(\text{H}_0) = .83$.

To further verify the functional significance and specificity of the slowest segment of the delta plot as a specific indicator for selective response inhibition, both the overall mean RT and the individual effect size of the individual interference effect were also entered (separately) as covariates in the fMRI regression model as a control variable. Importantly, these analyses did not yield any significant effects, $F < 1$, confirming that only one specific parameter (i.e., the slope of the slowest segment of the delta plot, as specifically predicted) corresponded with rIFC activation.

In addition to the analysis of the neural activation during the subject's behavior, we also analyzed the activations during the action observation. Observation of trials that would produce strong reactive control (cOI > cOC) did not yield any significant activations. Observation of trials associated with maintenance as opposed to release of proactive control (iOI > iOC) did

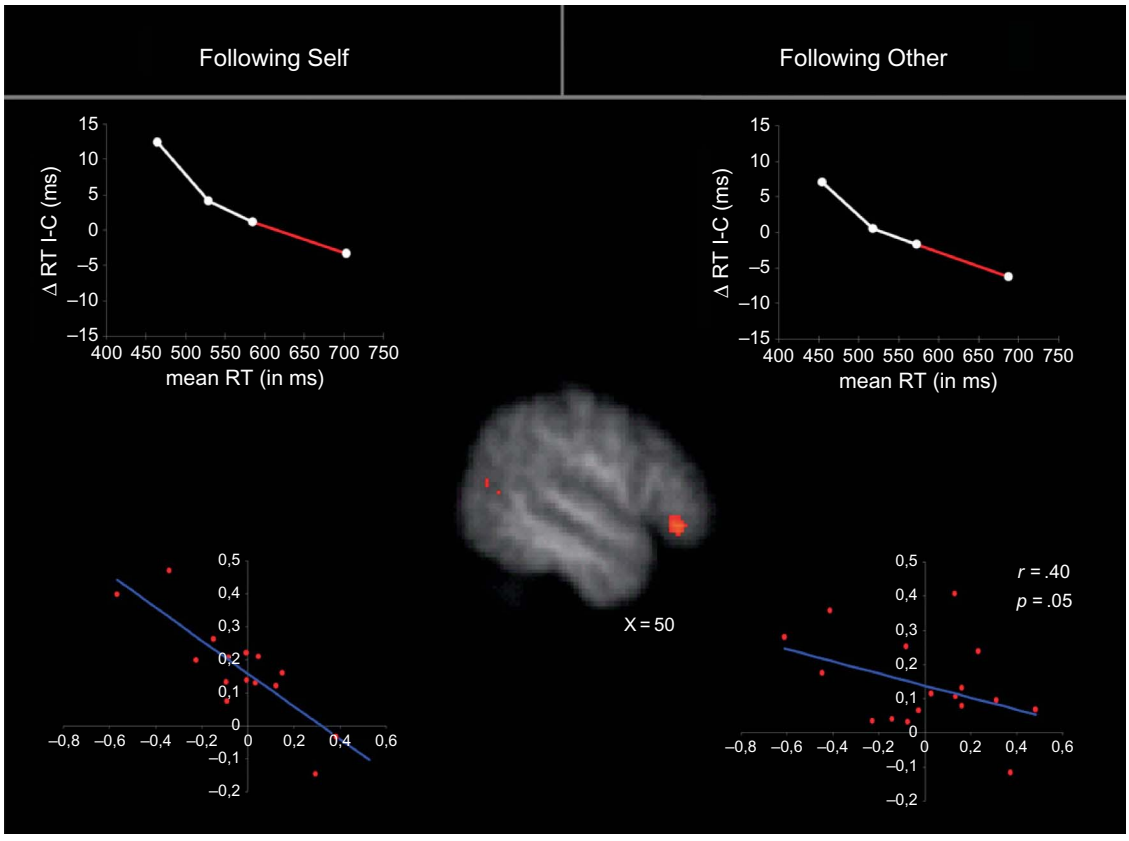

Figure 3. Implemented reactive control. Covariate analysis with individual parameters derived from 17 participants for each segment of the delta plot (only the correlation between the slowest segment of the delta slope separately for the "self" and "other" conditions and the percent signal change is depicted). Upper panel displays the delta slopes separately for the "self" and the "other" condition after congruent trials. Middle panel shows the averaged activation across 17 participants rendered onto a template brain ($z > 2.3$, cluster > 34 voxels) of the covariance analysis with the slowest segment of the delta plot. The only significant prefrontal activation is obtained for the slowest segment of the delta plot in the rIFC ($x = 50$, $y = 32$, $z = -6$) for the "self" condition. Coordinates are given in MNI space. Lower panel shows Pearson correlations (two-tailed) between the percent signal changes (y-axis) derived from the right inferior frontal cortex (rIFC) and the demeaned delta slopes (x-axis) for the "other" condition.

yield a significant region of activation in the left lPFC (90 voxels), spatially consistent with the activation found during the subject's own proactive control trials (–54, 2, 10).

DISCUSSION

In this study, we found neural correlates of proactive and reactive control to be similarly active following performed and observed behavior. We augmented standard fMRI analysis with a covariance-based analysis approach to investigate trial-to-trial adjustments in action observation. A social version of the Simon task was used in which participants were instructed either to observe the behavior of someone else or perform the task oneself on high- or low-conflict trials. This manipulation allowed us to compare modulations of congruency effects after high- versus low-conflict trials. Most importantly, we could investigate whether adaptive processes are comparable when observing someone else perform the task or performing the task oneself.

Behaviorally, we found that subjects showed conflict adaptation both following their own, and following observed behavior. This replicates our previous findings using this paradigm (Winkel et al., 2009). One difference between the behavior following own and observed trials, was that subjects showed higher error rates following observed trials. We believe this may be an aftereffect of reduced attention during the observed trial.

Our first hypothesis was that the left lPFC is involved in proactive control. As such, we expected

higher left lPFC during proactive control. That is, following high-conflict trials, we expected higher activation for high- compared to low-conflict trials. This hypothesis was confirmed by the present data. Following conflict, we found higher activation in the left lPFC for incongruent vs. congruent trials (iI > iC) (Figure 2). This finding is in line with the conflict-monitoring hypothesis as well as several studies showing that the left lPFC is involved in implementing control after encountering high conflict (e.g., Botvinick et al., 2001; Kerns, 2006). On the surface, one might argue that comparing iI > cI trials would more accurately model proactive control than comparing iI > iC trials. After all, the behavioral analysis of the conflict adaptation effect compares only the effect of different preceding trials. However, although the behavioral effect of proactive control is measured on the subsequent trial, the onset of the control lies in the preceding trial. If we were to analyze the iI > cI contrast, both compared trials would include the onset of proactive control, thus potentially occluding any activation differences resulting from the previously engaged proactive control. In our current contrast (iI > iC), we measure the onset of proactive control immediately related to the incongruent stimulus, but during a trial where the incongruent stimulus causes little reactive control (see Figure 3).

Our second hypothesis focused on the role of the rIFC in reactive control. More specifically, we expected activation strength in the rIFC to show covariation with the strength of reactive control. We demonstrated just such an effect in our data set, showing covariation of the rIFC with the negative slope of the late section of the delta plots (Figure 3). To further support the role of the rIFC in reactive control, a combined EEG/fMRI experiment examining the relationship between rIFC activity and lateralized readiness potential would be informative.

Finally, our third hypothesis stated that observing someone else or performing the task oneself would involve comparable adaptive mechanisms. Specifically, we expected to find left lPFC activation on current incongruent trials compared to congruent trials following the observation of someone else performing a high-conflict trial. We did indeed find left lPFC activations for both the "self" and "other" conditions. However, the activations in the "self" condition were located more ventrally than the activations in the "other" condition. A similar division, with more dorsal activations for representations of others, and more ventral activations for representations of self, has previously been demonstrated in the medial wall of the PFC (Overwalle, 2009). Although our results hint at such a division in the lateral prefrontal cortex as well, we cannot conclude its presence without subsequent experimentation.

In addition to the left lPFC for proactive control, we hypothesized that activation in the rIFC would correlate with individual negative slope values on incongruent trials after observing someone else performing on low-conflict trials. We did indeed find such a correlation. As can be seen from Figures 2 and 3, observing/simulating someone else's performance led to comparable trial-to-trial adjustments in both left lPFC and rIFC when subsequently performing the task oneself. This pattern of results was corroborated by comparable temporal dynamics for the "self" and "other" condition following high- as well as low-conflict trials (Figures 2 and 3, upper panels). Both our reactive and proactive control findings revealed very similar effects in the "self" and "other" conditions, supporting the simulation account in the field of conflict adaptation.

In addition to our findings of sequential effects following observed behavior, we also show activations related to proactive control during the observed behavior. This finding further supports the role of the left lPFC in proactive control. We did not find rIFC activations during the observed reactive control trials. One reason for this might be that reactive control reflects the buildup of reactive control throughout a trial. On the observed trials, there is already a global inhibition since the subject is not responding to the stimulus. This might prevent any reactive control effects from taking place during this time.

In general, the present data pattern corroborates and extends findings from previous behavioral, electrophysiological, and neuroimaging studies on action observation (Kerns, 2006; Schuch, & Tipper, 2007; Tsai & Brass, 2007; Tsai et al., 2008, van Schie et al., 2004; Winkel et al., 2009). Here, we also show that action observation in a conflict task leads to behavioral and neural adjustments as if we have encountered the conflict ourselves. More specifically, we can show that the same trial-to-trial adjustments take place after high- versus low-conflict trials in action observation as in our own action. Interestingly, also the temporal dynamics depicted by the delta plots reveal a similar pattern of trial-to-trial adjustments in action observation compared to performing oneself. We see the same activations during the observation of a trial that enables proactive control, as during the performance of such a trial.

The overall effect sizes found in the "other" condition are smaller compared to those found in the "self" condition. This is similar to a study by van Schie et al. (2004). This experiment compared the error-related negativity (ERN) elicited by the observation of an

error committed by someone else to that elicited by committing an error oneself. The authors argued that action observation yielded a comparable error signal to one's own errors; however, the overall effect size was much smaller. According to the simulation account (Gallese et al., 2004; Iacoboni et al., 2005; Ramnani & Miall, 2004), the observation of another person's behavior induces internal states in the observer that are similar to those that would occur if the observer undertook the action herself (cf. Schuch, & Tipper, 2007). We argue that simulation mechanisms can account for our present data, but that the simulation might result in weaker neural, as well as behavioral effects, compared to performing the task oneself. An explanation might be the vividness with which the observed behavior is simulated internally. This interpretation is supported by our findings with regard to performance on catch trials. We found that catch trial performance correlated significantly with the difference of the conflict adaptation effect following "other" and following "self" trials. The correlation indicates that participants who attended very closely to the observed behavior, as evidenced by their ability to detect errors, were also more affected by the observed conflict in their subsequent behavior.

Taken together, our results extend the notion of simulation mechanisms of higher-order cognitive control mechanisms. We provide evidence that well-known conflict adaptation processes are similarly represented in the brain, independently of whether we tune our behavior to our own conflict, or to someone else's. The left lPFC mediates prolonged proactive control similarly following another's behavior or one's own, whereas the rIFC covaries with reactive control similarly following one's own, and following observed conflict. In other words, we adapt to others' conflict as if it were our own.

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