Behavioral/Systems/Cognitive

Frontal Cortex Mediates Unconsciously Triggered Inhibitory Control

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To further our understanding of the function of conscious experience we need to know which cognitive processes require awareness and which do not. Here, we show that an unconscious stimulus can trigger inhibitory control processes, commonly ascribed to conscious control mechanisms. We combined the metacontrast masking paradigm and the Go/No-Go paradigm to study whether unconscious No-Go signals can actively trigger high-level inhibitory control processes, strongly associated with the prefrontal cortex (PFC). Behaviorally, unconscious No-Go signals sometimes triggered response inhibition to the level of complete response termination and yielded a slow down in the speed of responses that were not inhibited. Electroencephalographic recordings showed that unconscious No-Go signals elicit two neural events: (1) an early occipital event and (2) a frontocentral event somewhat later in time. The first neural event represents the visual encoding of the unconscious No-Go stimulus, and is also present in a control experiment where the masked stimulus has no behavioral relevance. The second event is unique to the Go/No-Go experiment, and shows the subsequent implementation of inhibitory control in the PFC. The size of the frontal activity pattern correlated highly with the impact of unconscious No-Go signals on subsequent behavior. We conclude that unconscious stimuli can influence whether a task will be performed or interrupted, and thus exert a form of cognitive control. These findings challenge traditional views concerning the proposed relationship between awareness and cognitive control and stretch the alleged limits and depth of unconscious information processing.

Key words: **awareness; cognitive control; cognition; consciousness; inhibition; vision**

Introduction

Over the last decades, research in psychology and neuroscience has shown that a substantial amount of cognitive processing goes on outside awareness (for review, see Dehaene and Naccache, 2001; Kouider and Dehaene, 2007). Although there appears to be general agreement that simple behavior is influenced by information of which we are unaware (but see Holender and Duscherer, 2004; Hannula et al., 2005), complex behaviors are often thought to result from conscious cognitive control (Umilta, 1988; Dehaene and Naccache, 2001; Jack and Shallice, 2001; Baars, 2002; Eimer and Schlaghecken, 2003; Tsushima et al., 2006). Cognitive control functions regulate and monitor our ongoing actions to optimize our behavior. Inhibitory control, the ability to cancel a planned or already initiated action, is an extreme form of cognitive control, in large part relying on the prefrontal cortex (PFC) (Casey et al., 1997; Konishi et al., 1999; Fuster, 2000; Liddle et al., 2001; Ridderinkhof et al., 2004; Picton et al., 2007) and associated exclusively with consciousness (Dehaene and Naccache, 2001; Eimer and Schlaghecken, 2003). A crucial issue in the field of unconscious/conscious cognition is whether such high-level cognitive (control) processes are evident in the absence of consciousness.

We designed a masked Go/No-Go task to study the operation of unconscious inhibitory control and recorded EEG to track the fate of masked No-Go signals in the human brain. Typically, event-related potential (ERP) studies using the Go/No-Go task report clear distinctions between Go trials and No-Go trials in the latency range of the N2/P3 (ERP components that peak 200–500 ms after stimulus presentation) at frontocentral electrode sites, which have been attributed to generators in the parietal, mediofrontal and ventral/dorsal lateral prefrontal cortex (Kiefer et al., 1998; Bokura et al., 2001; Nieuwenhuis et al., 2003; Lavric et al., 2004). In our task, participants had to respond as fast as possible to a Go signal, but were instructed to withhold their response when they perceived a No-Go signal, preceding the Go-signal. In our version of this paradigm (see Fig. 1*a*), the Go signal also functioned as a metacontrast mask, leading to undetectable No-Go signals at the short stimulus onset asynchrony (SOA), and perfectly visible No-Go signals at the long SOA. Our main interest was in the comparison between Go trials and unconscious No-Go trials because this would reveal whether unconscious No-Go signals could trigger frontal inhibitory control mechanisms.

Previous behavioral and imaging studies have shown that the way unconscious stimuli are processed is affected by top-down settings of the cognitive system, such as temporal/spatial atten-

Received March 25, 2008; revised June 24, 2008; accepted July 1, 2008.

We thank Sandra Cornelisse and Johannes Tesselaar for their help with data acquisition and the members of the Amsterdam Center for the Study of Adaptive Control in Brain and Behavior and the Cognitive Neuroscience Group for helpful discussions. We thank Stephen Whitmarsh for helping us with the source analysis.

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DOI:10.1523/JNEUROSCI.1278-08.2008

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tion, task strategy and the task being performed (Naccache et al., 2002; Kunde et al., 2003; Sumner et al., 2006; Nakamura et al., 2007). To test whether the relevance of the masked No-Go signal influences the depth to which it is processed, we performed a control experiment, in which the physically identical masked stimulus was task irrelevant (see Fig. 1*b*). This experimental setup enabled us to test whether (1) high-level inhibitory control processes can be triggered unconsciously, (2) unconscious No-Go signals reach prefrontal areas, and (3) task relevance influences the depth of processing of unconscious stimuli.

Materials and Methods

Participants. Thirty undergraduate psychology students of the University of Amsterdam (15 in each experiment; 22 females) participated and gave their written informed consent before participation. All were right handed, had normal or corrected-to-normal vision, and were naive to the purpose of the experiments. All procedures were executed in compliance with relevant laws and institutional guidelines and were approved by the local ethical committee.

Stimuli. Stimuli were presented on a gray box (59.1 cd/m^2; visual angle, 3.78°) against a black background (2.17 cd/m^2) at the center of a 15 inch BenQ TFT monitor with a refresh rate of 60 Hz. The monitor was placed at a distance of ~90 cm in front of the participant so that each centimeter subtended a visual angle of 0.64°. Participants were told that they would see a black annulus (the Go signal; 2.17 cd/m^2; visual angle, 1.30°; duration, 100 ms) and that they would have to respond as fast as possible by pressing a button with their right index finger. In the masked Go/No-Go task, participants were instructed to withhold their response when they perceived a gray circle (the No-Go signal; 41.85 cd/m^2; visual angle, 0.60°; duration, 16.7 ms) preceding the Go signal. The SOA between the No-Go signal and the Go signal was either short (16.7 ms) or long (83 ms). The No-Go circle exactly fitted within the Go-annulus, which typically results in efficient metacontrast masking (Enns and Di Lollo, 2000). We used perceptually weak No-Go signals and very strong Go signals, which is known to result in a monotonic masking function, leading to undetectable stimuli at short SOAs (Francis, 1997; Di Lollo et al., 2004). Two postexperimental detection tasks showed that No-Go signals remained undetectable at the short SOA, but were clearly visible at the long SOA. For simplicity, we labeled the condition with the long SOA as the conscious No-Go condition, and the condition with the short SOA as the unconscious No-Go condition. Stimuli were presented using Presentation (Neurobehavioral Systems)

The experiment consisted of three sessions on separate days with a maximum interval between sessions of 1 week. The first two sessions were behavioral sessions only (1 h per session); EEG was measured during the third session (3 h session). Participants performed seven experimental blocks per session, each containing 200 trials, 70% of which were Go trials, 15% were conscious No-Go trials, and 15% were unconscious No-Go trials. Trial duration was jittered between 1400 and 2200 ms (in steps of 200 ms), randomly drawn from a uniform distribution, rendering the presentation of the stimuli unpredictable. After each block, participants received performance feedback [mean reaction time (RT), percentage correct stops on conscious No-Go trials]. Participants were not informed about the presence of unconscious No-Go trials and did not receive any feedback about performance on these trials during testing. At the end of the third session of the masked Go/No-Go task, participants performed several detection tasks to assess the visibility of No-Go signals when masked with a SOA of 16.7 ms, as well as a SOA of 83 ms.

In the control experiment, participants were told to inhibit their responses to the black cross (2.17 cd/m^2; visual angle, 0.50°; duration, 16.7 ms). By using a different No-Go signal (a black cross instead of a gray circle), we prevented any association of the unconscious gray circle with response inhibition, as response inhibition was associated exclusively with the conscious black cross in this experiment (Fig. 1*b*). All other parameters and procedures were exactly the same as in the masked Go/No-Go task.

Behavioral tests of gray circle visibility. To test whether participants were truly unaware of the No-Go signals presented just before the Go signal, participants performed two detection tasks after the final experimental block of the masked Go/No-Go task. First, participants performed a yes–no detection task (one block of 200 trials). Trial timing and number of trials per condition was exactly the same as in the masked Go/No-Go task. Participants were instructed to press the right button whenever they thought a No-Go signal was presented. In all other cases they did not have to press the button. Second, two blocks of a forced-choice discrimination task were done. Before starting this task, participants were informed about the presence of No-Go signals appearing very shortly before the Go signal on some trials during the experiment. None of the participants reported to be aware of these No-Go signals during the Go/No-Go experiment. In the forced-choice discrimination task, each block consisted of 50 masked No-Go trials (SOA of 16.7 ms) and 50 Go trials. Stimuli were presented in pseudorandom order and trial timing was exactly the same as in the experimental sessions. Participants were instructed to press the left button when they thought that a No-Go signal shortly preceded the Go signal and press the right button when they thought this was not the case. Participants were told that in 50% of all trials, a Go signal was preceded by a No-Go signal and were instructed to consider this in their response.

Behavioral analysis. Because conscious No-Go signals are quite difficult to perceive at the beginning, participants will produce false alarms on Go trials, which means that they sometimes inhibit their response on trials on which no No-Go signal was presented (but participants thought that there was one). To reliably measure unconsciously triggered response inhibition, unconscious response inhibition is quantified in terms of a relative inhibition rate (percentage of trials inhibited) on uncon-

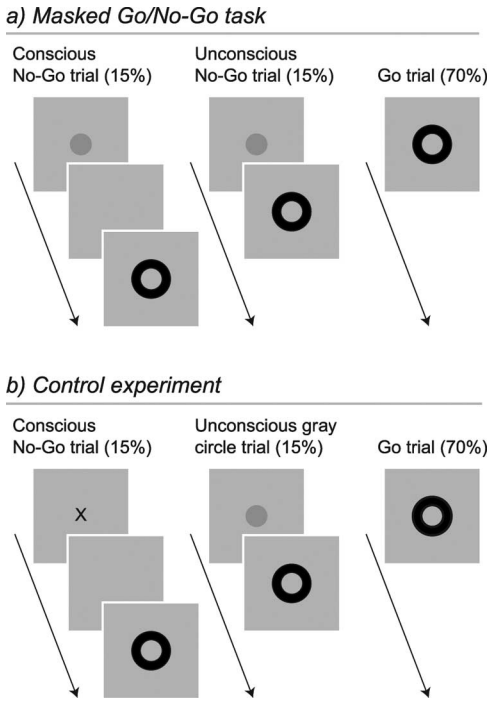

Figure 1. Stimuli and trial timing of the masked Go/No-Go task and the control experiment. The gray circle and black cross duration was 16.7 ms. Go signal duration was 100 ms. In conscious No-Go trials, the SOA between the No-Go signal and the Go signal was 83 ms. Participants had to respond to the Go signal (black metacontrast mask) but were instructed to withhold their response when a No-Go signal preceded the Go signal. In the masked Go/No-Go task, a gray circle served as a No-Go signal, whereas in the control experiment, the No-Go signal was a black cross. Therefore, the masked gray circle was associated with inhibition in the masked Go/No-Go task and thus served as an unconscious No-Go signal. In the control experiment, the unconscious gray circle was not associated with inhibition (and was task irrelevant) because participants were instructed to inhibit their responses on a black cross. Comparing processing of unconscious gray circles between both experiments enabled us to test whether (1) high-level inhibitory control processes can be triggered unconsciously, (2) unconscious No-Go signals reach prefrontal areas, and (3) task relevance influences the depth of processing of unconscious stimuli.

Table 1. General performance measures for the masked Go/No-Go task and the control experiment

	Session 1		Session 2		Session 3	
Measure	Experiment	Control	Experiment	Control	Experiment	Control
IR Go	1.9 (0.4)	0.9 (0.2)	0.7 (0.2)	1.2 (0.6)	0.7 (0.2)	1.4 (0.5)
IR con. No-Go	60.6 (2.8)	76.5 (1.7)	64.2 (2.0)	75.4 (1.6)	63.9 (2.8)	73.0 (1.4)
IR unc. signal	2.25 (0.6)	1.0 (0.4)	1.1 (0.3)	1.1 (0.6)	0.8 (0.3)	1.4 (0.7)
RT Go	326 (8.1)	278 (9.8)	278 (8.2)	254 (9.7)	273 (5.5)	247 (7.1)
RT con. No-Go	235 (7.7)	191 (7.8)	197 (8.9)	173 (6.6)	196 (6,9)	169 (7.0)
RT unc. signal	324 (7.5)	271 (9.9)	277 (8.5)	249 (10.0)	272 (5.7)	243 (7.1)

Experiment, Masked Go/No-Go experiment; Control, control experiment; IR, inhibition rate (percentage of inhibited trials); con. No-Go, conscious No-Go trial; unc. signal, unconscious No-Go trial in the masked Go/No-Go experiment and an unconscious gray circle trial in the control experiment. The SEM is shown in parentheses. The reaction times are shown in milliseconds.

scious No-Go trials compared with Go trials, which are perceptually similar (see Results). So, if subjects inhibit more responses on unconscious No-Go trials (successful stops) compared with Go trials (false alarms), this means that unconscious No-Go signals triggered response inhibition. This yields a more conservative measure than just comparing the number of inhibited trials on unconscious No-Go trials with a zero baseline. All trials for which there was no response by the start of the next trial are incorporated in our analyses of response inhibition. Repeated-measures ANOVAs were performed on square root percentages of responding on Go trials, unconscious No-Go trials and unconscious gray circle trials with within-subjects factors of trial type and session. For the response time analyses RTs <100 and >1000 ms were excluded. Repeated-measures ANOVAs were performed on the mean RT on correct Go trials, mean RT on responded unconscious No-Go trials, and mean RT on responded unconscious gray circle trials with within-subjects factors of trial type and session. Forced-choice detection performance was analyzed by signal detection methods and tested for significance using a one-sample *t* test.

EEG measurements and analyses. EEG was recorded and sampled at 256 Hz using a BioSemi ActiveTwo 48-channel system. Forty-eight scalp electrodes were measured, as well as four electrodes for horizontal and vertical eye-movements (each referenced to their counterpart) and two reference electrodes on the ear lobes. After acquisition, the EEG data were referenced to the average of both ears and was filtered using a high pass filter of 0.5 Hz, a low-pass filter of 20 Hz and a notch filter of 50 Hz. Eye movement correction was applied on the basis of the horizontal and vertical electro-oculography, using the algorithm of Gratton et al. (1983). Thereafter, we did an artifact correction on all separate channels by removing segments outside the range of ± 50 μV or with a voltage step exceeding 50 μV per sampling point. Baseline correction was applied by aligning time series to the average amplitude of the interval from -300 ms to the -100 ms preceding Go signal onset. This is well before the presentation of the gray circle in the conscious No-Go as well as the unconscious No-Go/gray circle trials. All preprocessing steps were done with Brian Vision Analyzer (Brian Products).

All subsequent analyses were conducted on difference waves obtained by subtracting the ERP on unconscious No-Go/gray circle trials from the ERP on Go trials. To increase the signal-to-noise ratio we created two regions of interest (ROIs): a frontocentral ROI consisting of several frontal electrodes (FCz, FC1, FC2, Fz, F3, F4, AF3, and AF4) and an occipitoparietal ROI consisting of several occipital and parietal electrodes (Iz, I1, I2, Oz, O1, O2, PO7, P5, P7, PO8, P6, and P8). ROIs were selected on the basis of previous literature (Eimer, 1993; Kiefer et al., 1998; Falkenstein et al., 1999; Bokura et al., 2001; Nieuwenhuis et al., 2003; Lavric et al., 2004; Fahrenfort et al., 2007) and the topography of the difference waveform (Go minus unconscious No-Go) reported in Figure 4. We performed random-effects analyses by using sample-by-sample paired *t* tests (two-tailed) from 0 to 500 ms after Go signal presentation to test at which time points difference waves differed significantly from zero. To solve the multiple-comparison problem, we applied a false discovery rate (FDR) correction (Fahrenfort et al., 2007). This method corrects for the number of tests being performed on the basis of the expected proportion of false alarms or type I errors (for a more detailed explanation of this method, see Genovese et al., 2002). All EEG analyses were done using Matlab (Mathworks).

Source analysis was performed using the BrainStorm software package (freely available at http://neuroimage.usc.edu/brainstorm/). The source imaging model consisted of 10,000 distributed current dipoles whose locations and orientations were constrained to the cortical mantle of a generic brain model, built from the standard MNI brain (Montreal Neurological Institute). First, the cortex, skull and scalp surface envelopes of this generic brain model were warped to the standard geometry of the electrode locations in the cap that was used in the experiments. Next, weighted minimum-norm cortical current estimates were computed from the EEG time series using the boundary element method, in which we used the scalp, skull and cerebral spinal fluid as compartments (for an extensive review on these procedures, see Baillet et al., 2001).

Results

Although both tasks are considerably more difficult than a "standard" Go/No-Go task, participants were able to perform the tasks well, demonstrated by typical inhibition rates (percentage of inhibited No-Go trials) of ~60–75% on conscious No-Go trials, while still being fast on Go trials (~270 ms) (for detailed behavioral results for both experiments, see Table 1).

Behavioral effects of unconscious No-Go signals

If unconscious No-Go signals are capable of triggering response inhibition, participants should sometimes inhibit their responses on unconscious No-Go trials. More precisely, a significantly higher inhibition rate on unconscious No-Go trials than on Go trials should be evident, although the two are perceptually identical. In the masked Go/No-Go task, participants stopped relatively more frequently on unconscious No-Go trials than on Go trials across all sessions ($F_{(1,14)} = 4.97$, $p = 0.043$). This was not the case for the unconscious gray circle trials in the control experiment ($F < 1$) (Fig. 2, left). This indicates that No-Go signals can actively trigger response inhibition unconsciously, resulting in complete response termination on a small number of trials. In the masked Go/No-Go task, the absolute percentage of successful stops on unconscious No-Go trials and false alarms on Go trials decreased slightly across sessions ($F_{(2,28)} = 8.25$, $p = 0.002$) (Table 1), probably because of practice.

When participants are not able to fully suppress the Go behavior, they might still slow down their responses as a result of a "partial" inhibition process. This should reveal itself as an increase in RTs on unconscious No-Go trials compared with Go trials. Note, however, that this predicted increase in RT may be counteracted by another effect reported previously: responses to masks generally tend to speed up when preceded by unseen primes (Fehrer and Raab, 1962). This effect is known as the Fehrer–Raab effect. In our experiments, all trials contain masks (the Go signal), but on unconscious No-Go trials, gray circles precede masks. Consequently, baseline RTs on unconscious No-Go trials, disregarding any unconscious inhibition effect, should be faster than RTs on Go trials. So, initially, unconscious

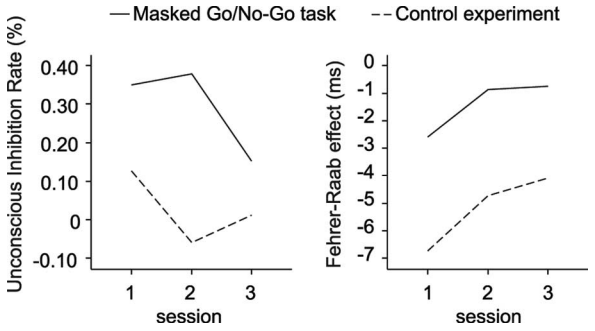

Figure 2. Behavioral measures of unconsciously triggered response inhibition. In the masked Go/No-Go task, participants inhibited their responses more often on unconscious No-Go trials than on Go trials across all sessions (left; effect sizes: percentage of inhibited unconscious No-Go trials minus the percentage of inhibited Go trials). In the control experiment, participants did not inhibit their responses more often on unconscious gray circle trials than on Go trials. Additionally, the Fehrer–Raab effect (right) was significantly smaller in the masked Go/No-Go task (mean RT on unconscious No-Go trials minus mean RT on Go trials) than in the control experiment (mean RT on unconscious gray circle trials minus mean RT on Go trials). This finding supports the notion that unconscious No-Go signals triggered inhibitory control processes in the masked Go/No-Go task, whereas in the control experiment, unconscious gray circles did not (or less so).

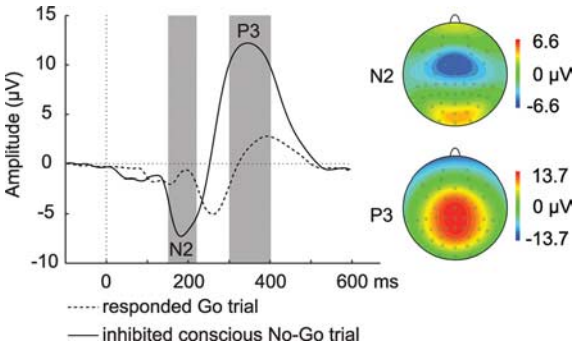

Figure 3. Typical ERP reported in the standard version of the Go/No-Go task. The average ERP at electrode FCz (for the masked Go/No-Go task) is depicted for responded Go trials as well as for conscious No-Go trials that were successfully inhibited (time locked to the onset of the Go signal). Scalp voltage maps show a characteristic frontocentral distribution of the N2 component and a more centroparietal distribution of the P3 component for successfully inhibited (conscious) No-Go trials. The vertical gray bars are an indication of the area that was selected for the computation of the voltage maps.

gray circles should speed up responses, to be counteracted and reversed only by the tendency to implement response inhibition as triggered by the unconscious gray circle (in the masked Go/No-Go task only, not in the control task). Because of the Fehrer–Raab effect, we can only speculate whether RTs on unconscious No-Go trials will eventually become longer than RTs on Go trials in the Go/No-Go task. However, we established a baseline value of the Fehrer–Raab effect in the control experiment (no association between the gray circle and response inhibition) against which we can compare the magnitude of the inhibition effect in the masked Go/No-Go task. We expected to see a clear Fehrer–Raab effect (mean RT on unconscious gray circle trials minus mean RT on Go trials), unconfounded with inhibitory processes in the control experiment, which can be compared with the Fehrer–Raab effect in the masked Go/No-Go task (mean RT on unconscious No-Go trials minus mean RT on Go trials). When the Fehrer–Raab effect in the masked Go/No-Go task is less than in the control experiment, we can conclude that unconscious gray circles, when associated with response inhibition, can trigger inhibitory control and slow down responses that are not inhibited.

In the control experiment, a significant Fehrer–Raab effect was observed ($F_{(1,14)} = 40.19$, $p < 0.001$), whereas in the masked Go/No-Go task this was not the case ($F_{(1,14)} = 2.12$, $p = 0.167$). The Fehrer–Raab effect differed significantly between the masked Go/No-Go task and the control experiment ($F_{(1,28)} = 8.98$, $p = 0.006$) (Fig. 2, right) (same effect for median RTs, $p = 0.014$). These findings mean that the presence of an unconscious gray circle, in itself, speeds up responding. However, the response speed slows down when the unconscious gray circle is strongly associated with response inhibition. We take this finding to support the notion that unconscious gray circles triggered inhibitory control processes in the masked Go/No-Go task, and not (or less so) in the control experiment. Note that this comparison also controls for any general suppressive effect of the gray circle. It could be that unconscious gray circles also trigger response inhibition (and hence slowing) in the control experiment because of a generalized association of prime stimuli with suppressing actions. This should cancel out when looking at the difference in Fehrer–Raab effect between the masked Go/No-Go task and the control experiment, thus rendering our estimation of inhibitory effects conservative, as well as specific.

RTs on Go, conscious No-Go, and unconscious gray circle trials decreased across sessions in both experiments because of practice (largest $p < 0.01$). RTs in the control experiment were slightly, but significantly shorter than RTs in the masked Go/No-Go task ($F_{(1,28)} = 12.29$, $p = 0.002$).

Unconsciously triggered inhibitory control is associated with frontal brain potentials

Figure 3 shows the ERP typically reported in the literature in the standard version of the Go/No-Go task, including the ERP on responded Go trials and the ERP on successfully inhibited conscious No-Go trials. The figure shows a typical sequence of components including, most prominently, a clear N2/P3 complex on conscious No-Go trials. Voltage scalp maps (Fig. 3, right) show the usual scalp distributions for the N2 (peaking at frontocentral electrodes) and the P3 (peaking at centroparietal electrodes) on conscious No-Go trials, replicating characteristic Go/No-Go findings (Eimer, 1993; Kiefer et al., 1998; Falkenstein et al., 1999; Bokura et al., 2001; Nieuwenhuis et al., 2003; Lavric et al., 2004). Note, that the conscious No-Go ERP shows a leftward shift in time compared with Go trials (see, e.g., the N2 latency). This is because ERPs are Go signal-locked and therefore the conscious No-Go signal precedes the Go signal by 83 ms. For this reason, the conscious No-Go ERP starts earlier than the Go ERP.

To determine whether No-Go signals can actively trigger inhibitory mechanisms unconsciously, we compared ERPs on unconscious No-Go trials with ERPs on Go trials. The left panel of Figure 4 shows voltage maps of the difference between Go and unconscious No-Go trials for the interval between 0 and 496 ms after Go signal onset ($t = 0$) for the masked Go/No-Go task. In this task, two neural events can be distinguished: first, an early difference at occipital electrode sites at ~125–164 ms; second, a difference at ~332–414 ms at frontocentral electrode sites. It seems that occipital areas pick up unconscious No-Go signals, before they are processed further downstream, probably in frontal areas. The first neural event is also present in the control experiment, whereas the latter neural event is absent (Fig. 4, right). We report a detailed analysis of these effects for the frontocentral ROI, followed by the analysis of the effects observed for the occipitoparietal ROI (see Materials and Methods).

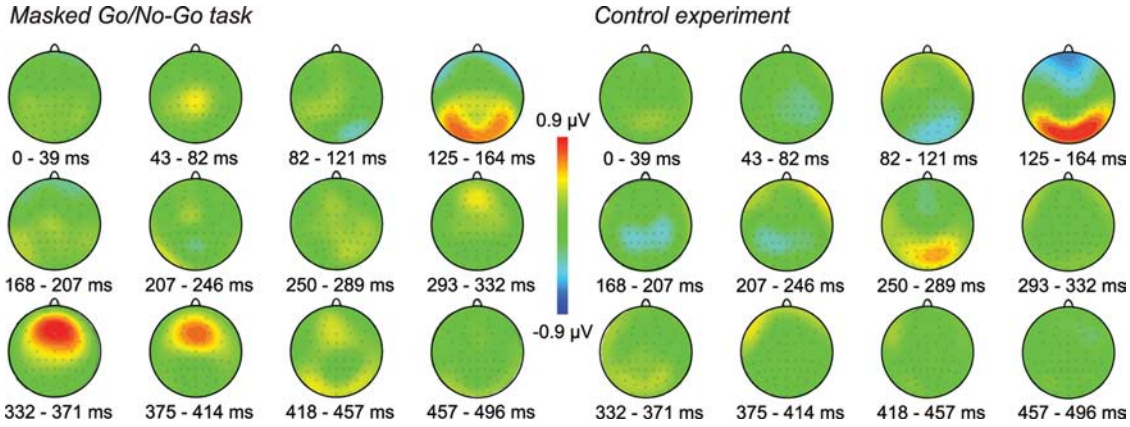

Figure 4. The neural processing of unconscious gray circles. Scalp voltage maps show activations evoked by the unconscious stimulus as a difference between Go trials and unconscious No-Go trials (masked Go/No-Go task) and the difference between Go trials and unconscious gray circle trials (control experiment). The topography of the difference waveform between 0 and 496 ms is shown in 12 steps ($t = 0$ is the onset of the Go signal). In the masked Go/No-Go task, two neural events can be distinguished: (1) an early occipital difference at ~125–164 ms and (2) a frontocentral difference at ~332–414 ms. The first, early occipital event probably represents the visual encoding of the unconscious stimulus and is also present in the control experiment where the masked gray circle has no behavioral relevance. The second, frontal event is unique to the masked Go/No-Go experiment and probably represents the subsequent implementation of inhibitory control in the PFC.

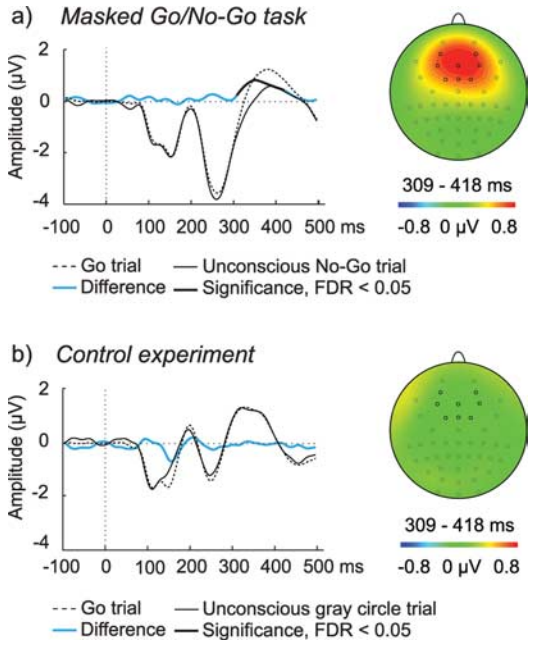

Figure 5. Frontal event-related potentials. ***a***, ERP waveforms for unconscious No-Go trials and Go trials for the frontocentral ROI (pooling of electrodes FCz, FC1, FC2, Fz, F3, F4, AF3, and AF4, time locked to the onset of the Go signal). In the masked Go/No-Go task, unconscious No-Go trials differed significantly from Go trials between 309 and 418 ms. ***b***, In the control experiment, unconscious gray circle trials did not differ from Go trials at any point in time between 0 and 500 ms after Go signal onset. Scalp voltage maps (right, pooled electrodes are shown in black) show the scalp distributions of the differential EEG activity between Go trials and unconscious No-Go trials (masked Go/No-Go task) and Go trials and unconscious gray circle trials (control experiment) between 309 and 418 ms.

Figure 5*a* shows the averaged waveform for Go trials and unconscious No-Go trials for the frontocentral ROI. Unconscious No-Go trials differed from Go trials between 309 and 418 ms ($p < 0.05$, FDR corrected) (see Materials and Methods). This observed differential effect was found at frontocentral electrodes (Fig. 5*a*, voltage maps) close to the cortical sites of activation that have been found in previous functional magnetic resonance imaging (fMRI) studies using standard versions of the Go/No-Go task (Casey et al., 1997; Konishi et al., 1999; Fuster, 2000; Liddle et al., 2001; Kelly et al., 2004; Garavan et al., 2006). To show that this difference at frontocentral electrodes is not a purely stimulus-driven effect (or reflects a neural correlate of the Fehrer–Raab effect per se), we did the same analysis for the control experiment (Fig. 5*b*). In the control experiment, no differences were found for the same frontocentral ROI at any point in time between 0 and 500 ms.

By comparing the evoked frontal ERP activity between the masked Go/No-Go task and the control experiment, we subtract out any perceptual processes related to the gray circle and specifically test whether the observed frontal ERP difference in the masked Go/No-Go task (in which the gray circle is associated with inhibition) is larger than in the control experiment (in which the gray circle is task irrelevant). An ANOVA yielded a significant task effect at the frontocentral ROI between 309 and 418 ms ($F_{(1,29)} = 9.41$, $p = 0.005$). This indicates that our unconsciously triggered inhibitory effect cannot be explained by any stimulus-driven perceptual activity or by the Fehrer–Raab effect per se. More importantly, this finding shows that unconscious gray circles elicit frontal ERP activity when they are associated with response inhibition (masked Go/No-Go task), whereas the same gray circles do not (or less so) when they are irrelevant for the task at hand (control experiment).

Frontal ERP activity determines the magnitude of inhibition

Next, we analyzed individual differences in the extent to which unconscious No-Go signals are capable of triggering inhibitory control processes. In particular, we examined whether individual differences in the behavioral and electrophysiological expressions of inhibition on unconscious No-Go trials covaried. We hypothesized that those participants displaying greater frontal ERP differences between Go trials and unconscious No-Go trials slow down more on unconscious No-Go trials compared with Go trials. Furthermore, the RTs of participants should not increase if they do not show frontal ERP differences. To test this hypothesis, we calculated the mean voltage difference at all 48 electrodes in the significant time window of 309–418 ms. We calculated Spearman's rank correlations between this EEG difference measure and the observed RT slowing effects in the masked Go/No-Go task (RT unconscious No-Go trial minus RT Go trial) across participants at all electrodes. This correlation was highly significant at several specific frontocentral electrodes (Cz, FCz, Fz, FC1, FC2, FC5, all p values < 0.05, two-tailed, all rho values >

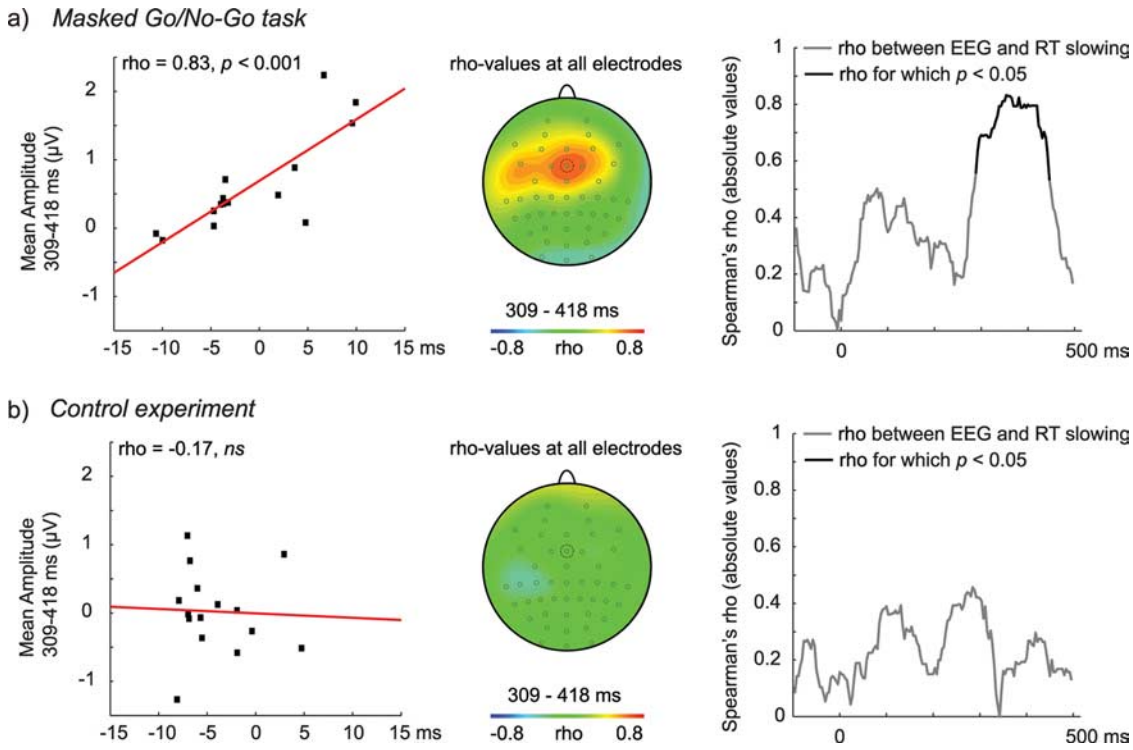

Figure 6. Frontal effects and correlations. ***a***, Left, Correlation between the mean amplitude difference between unconscious No-Go trials and Go trials in the significant time window (309–418 ms) and increase in RT (electrode FCz). The scatter plot shows a strong positive correlation between the size of this frontocentral ERP effect and the increase in RT to subsequent Go signals in the masked Go/No-Go task (each dot is one subject). The map in the middle shows the scalp distribution of rho values for all 48 electrode sites (red, positive correlation; blue, negative correlation). The distribution of the frontal ERP effect (Fig. 5) strongly corresponds to the distribution of correlations in the masked Go/No-Go task. Right, Correlation between a moving average of EEG activity and the increase in RT across time at electrode FCz (the shown rho values are absolute). At the moment in time that frontocentral electrodes differentiate between unconscious No-Go trials and Go trials (309–418 ms), a strong positive correlation appears ($p < 0.05$, between 289 and 445 ms), which is absent at other times, as well as in the control experiment. ***b***, Control experiment.

0.53) as well as the frontocentral ROI (rho = 0.53, $p = 0.043$). The middle panel of Figure 6 shows that the scalp distribution of these correlations closely resembles the observed scalp distribution shown in Figure 5*a*. The left panel of Figure 6*a* shows the scatter plot of this correlation for electrode FCz, which is the electrode usually showing the largest P3 differences in the standard version of the Go/No-Go task (Eimer, 1993; Kiefer et al., 1998; Falkenstein et al., 1999; Bokura et al., 2001). Correlations were completely absent in our control experiment (Fig. 6*b*), demonstrating that they were specific to inhibitory RT slowing and not related to the Fehrer–Raab effect per se. Hence, frontal EEG activity seems to be an indication for the impact unconscious No-Go signals have on subsequent inhibitory control behavior.

To show that the observed correlation between EEG activity and behavior is not only spatially specific but also temporally specific, we computed a moving average of 109 ms (the size of the significant time window between 309 and 418 ms) of the differential activity between unconscious No-Go trials and Go trials and correlated this measure across time with the increase in RT. Figure 6*a* (right) displays this correlation at electrode FCz. At the moment in time that frontocentral electrodes differentiate between unconscious No-Go trials and Go trials (309–418 ms), a strong correlation between increase in RT and EEG activity appears ($p < 0.05$ between 289 and 445 ms), which is absent at other times, as well as in the control experiment (Fig. 6*b*, right).

Thus, the spatial as well as the temporal profile of these correlations suggests that unconscious No-Go signals are able to trigger inhibitory control mechanisms, which are expressed at frontocentral scalp sites at ~300–400 ms poststimulus and result in an attempt to withhold the response. Although often not successful as such, this yields at least a slowing of the response.

Source localization of the frontal ERP activity

To shed some light on the cortical origins of the frontally observed event-related activity in the masked Go/No-Go task, we computed cortical current density maps by using a model consisting of 10,000 current dipoles (see Materials and Methods) (Sergent et al., 2005; Del Cul et al., 2007). Although the spatial resolution of this source modeling method is limited, it was used to yield an approximate estimation of the location and distribution of the neural activity observed on unconscious No-Go trials (compared with Go trials). Figure 7 shows the cortical current maps at 352 ms, which is the moment in time the ERP difference between unconscious No-Go trials and Go trials at the frontocentral ROI is largest. The source imaging revealed that the lateral prefrontal cortex was active at this moment in time. Additionally, the source reconstruction suggests that the cortical origin of our reported inhibitory effects is slightly right lateralized. Although the inferred locations from the source imaging analysis must be taken cautiously, this finding nicely corresponds to results of previous fMRI studies, which consistently reported predominantly the right (dorsal as well as ventral) lateral prefrontal cortex to be associated with No-Go inhibition (Casey et al., 1997; Konishi et al., 1999; Liddle et al., 2001; Kelly et al., 2004; Garavan et al., 2006). Thus, our source imaging results suggest that the frontal ERP component observed in the masked Go/No-Go task reflects the unconscious initiation of an inhibitory control process mediated by the (especially right) lateral prefrontal cortex.

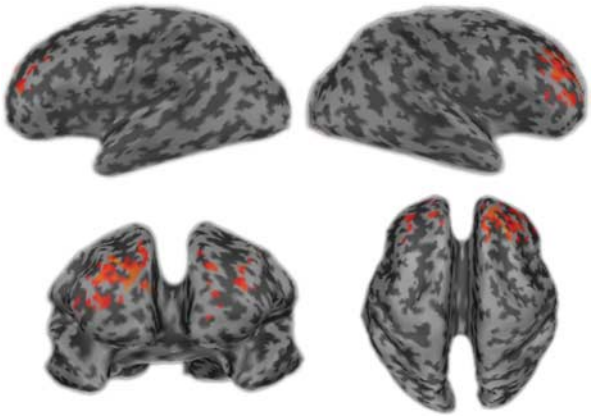

Figure 7. Cortical activity evoked by unconscious No-Go signals. The reconstructed cortical sources at the peak of the differential ERP activity between unconscious No-Go trials and Go trials (352 ms) at the frontocentral ROI (in the masked Go/No-Go task). The source imaging revealed that the (especially right) lateral prefrontal cortex was active at this moment in time. Cortical current maps are represented on smoothed standardized cortex and shown in four different views (left view, right view, anterior view, and superior view). Activity of the reconstructed cortical sources is indicated by color (in current density units, Am), thresholded at 50% of the maximum value (yellow, 6.5×10^{-5} Am).

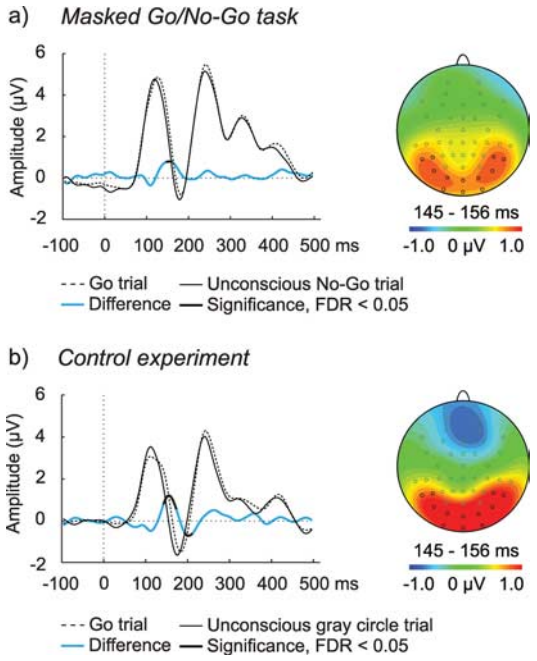

Figure 8. Occipital event-related potentials. ***a***, ERP waveforms for unconscious No-Go trials and Go trials for the occipitoparietal ROI (pooling of electrodes Iz, I1, I2, Oz, O1, O2, PO7, P5, P7, PO8, P6, and P8, time locked to the onset of the Go signal). Unconscious No-Go trials differed significantly from Go trials between 145 and 156 ms in the masked Go/No-Go task. ***b***, In the control experiment, unconscious gray circle trials differed significantly from Go trials between 141 and 172 ms and between 191 and 207 ms. Scalp voltage maps (right) show scalp distributions of the differential activity between 145 and 156 ms for both experiments (pooled electrodes are shown in black). The pattern of activity shows that unconscious gray circles were visually encoded alike in both conditions, but only triggered inhibitory control when they were associated with response inhibition (in the masked Go/No-Go task only).

Visual encoding of unconscious gray circles is similar for both experiments

Before unconscious No-Go signals can influence behavior, they have to be visually encoded (without leading to a conscious percept). To test whether unconscious gray circles are encoded comparably in the masked Go/No-Go task and the control experiment, we calculated the difference between Go trials and unconscious No-Go trials for the occipitoparietal ROI. We found early significant differences between 145 and 156 ms in the masked Go/No-Go task (Fig. 8*a*). Unconscious gray circle trials also differed significantly from Go trials in the control experiment in the same time window (141–172 ms) (Fig. 8*b*), but also slightly later in time (191–207 ms). To test whether there was a task effect at the occipitoparietal ROI, the evoked occipitoparietal ERP activity between the masked Go/No-Go task and the control experiment were directly compared. An ANOVA yielded no significant task effect at the occipitoparietal ROI between 145 and 156 ms ($F_{(1,29)} = 2.37$, $p = 0.135$). Thus, early visual evoked effects were similar across both tasks; if anything, this effect was even slightly larger in the control experiment than in the masked Go/No-Go task. Early occipital activity (145–156 ms) did not correlate with the increase in RT in the masked Go/No-Go task (rho $= -0.03$, $p = 0.93$) or the control experiment (rho $= 0.23$, $p = 0.42$). This pattern of activity shows that unconscious gray circles were visually encoded alike in both experiments, but only triggered inhibitory control when they were associated with response inhibition

Visibility of gray circles

We ran two behavioral detection tasks after the final session of the masked Go/No-Go task to verify that unconscious No-Go trials could not be discriminated from Go trials. The first task was designed to probe the subjective visibility of unconscious No-Go signals, as well as conscious No-Go signals (yes–no detection task, 200 trials). The second, more conservative task (forced-choice discrimination task, 200 trials) tested whether participants could detect subtle differences between unconscious No-Go trials and Go trials. In the yes–no detection task, the hit rate of 2.0% (SD, 1.2) on unconscious No-Go trials did not exceed the false alarm rate of 0.8% (SD, 0.3) observed on Go trials ($t_{(14)} = 1.27$, $p = 0.23$). Participants easily detected conscious No-Go trials, as expressed in an average hit rate of 93.3% (SD, 4.9).

Before starting the forced-choice discrimination task, participants were informed about the presence of No-Go signals appearing very shortly before the Go signal on some trials during the Go/No-Go experiment. None of the participants reported to be aware of these No-Go signals. In the forced-choice discrimination task, subjects were unable to discriminate unconscious No-Go trials from Go trials, yielding a hit rate of 44.9% and a false alarm rate of 43.1% (mean percentage correct, 51.0%; SD, 3.2). The resulting d' score of 0.05 (SD, 0.17) did not differ from the value expected by chance ($t_{(14)} = 1.09$, $p = 0.29$). We chose to analyze detection after the masked Go/No-Go experiment, assuming that any effect of perceptual learning would have revealed itself after the final session.

If incidental No-Go visibility would be responsible for the observed inhibition effects, one would expect a positive correlation between detection scores and behavioral as well as electrophysiological measures of inhibition. To test this, several correlational analyses were done between detection scores and behavioral and electrophysiological effects of the third session. The correlation between each participant's yes–no detection score (hit rate minus false alarm rate) and unconscious RT slowing was not significant (rho $= -0.08$, $p = 0.78$). The correlation between individual d' scores and unconscious RT slowing (rho $= -0.25$, $p = 0.37$) was also not significant. With respect to inhibition rates, there was no correlation between unconscious inhibition rate and yes–no detection (rho $= 0.02$, $p = 0.94$) and a

negative correlation between unconscious inhibition rate and d' scores (rho = −0.52, $p = 0.049$). The overall pattern of these correlations suggests that it is rather unlikely that the reported behavioral effects are attributable to accidental visibility of masked No-Go signals. This conclusion is further supported by the additional finding that there was no correlation between yes–no detection and frontal EEG activity (rho = 0.05, $p = 0.86$) and a trend toward a negative correlation between this EEG effect and d' scores (rho = −0.50, $p = 0.055$). The pattern of these correlations further support the notion that it is unlikely that No-Go visibility can account for the observed frontal EEG effects; if anything, individuals who were better at detecting the masked No-Go signal used it less often to inhibit their response and showed less frontal EEG activity. Therefore, our behavioral and ERP results cannot be explained by assuming accidental visibility of masked No-Go signals.

Discussion

We developed a new version of the Go/No-Go paradigm in which we masked No-Go signals to study the effect of unconscious No-Go signals on behavior and brain activity using ERPs. Previous brain imaging studies revealed that a predominantly right-hemispheric network of regions, including the PFC, is involved in response inhibition in the Go/No-Go paradigm (Casey et al., 1997; Konishi et al., 1999; Fuster, 2000; Liddle et al., 2001; Kelly et al., 2004; Ridderinkhof et al., 2004; Garavan et al., 2006). Behaviorally, we found that unconscious No-Go signals triggered full-blown response inhibition on some occasions and slowed down those responses that were not withheld. Our EEG results revealed a sequence of ERP deflections typically seen in the Go/No-Go paradigm (Eimer, 1993; Kiefer et al., 1998; Falkenstein et al., 1999; Bokura et al., 2001; Nieuwenhuis et al., 2003; Lavric et al., 2004). Specifically, we found differential activity between Go and unconscious No-Go trials at frontocentral electrode sites in the P3 latency range (309–418 ms). Although the spatial resolution of EEG is rather limited, source modeling of this frontal component suggests that it originates from (especially right) lateral prefrontal cortex. Therefore, it seems plausible that this unconsciously triggered No-Go activity corresponds to prefrontal activity seen in fMRI (Casey et al., 1997; Konishi et al., 1999; Liddle et al., 2001; Kelly et al., 2004; Garavan et al., 2006). However, the frontal origins of our effects should be explored more precisely with anatomically more accurate methods in the future.

Electrophysiological activity evoked by unconscious No-Go signals correlated strongly with the increase in RT to subsequent Go signals. Maximum correlations were specifically centered on frontocentral electrodes where previous No-Go studies (using conscious trials only) found the largest electrophysiological effects. The spatial profile and the onset and offset latency of these correlations suggests that unconscious No-Go signals make their way to frontal cortices, triggering response inhibition in the absence of awareness. This frontal ERP effect is unique to the masked Go/No-Go experiment, because the same masked signals do not trigger inhibitory control behavior and do elicit frontal ERP activity when they have no behavioral relevance. Importantly, early occipital activity (145–156 ms), representing the visual encoding of the unconscious stimulus, did not differentiate between the masked Go/No-Go task and the control experiment. This suggests that early (probably feedforward) (Fahrenfort et al., 2007) visual activity is not influenced by the task relevance of gray circles.

In the present experiments, we show that unconscious signals are processed more elaborately (probably activating brain areas further downstream) when the current task demands it than when they are irrelevant for the task at hand. This is in line with the idea that the depth and scope of neural processing of masked stimuli is modulated by top-down settings of the cognitive system (Dehaene et al., 2006; Nakamura et al., 2006, 2007). However, more importantly, our results go one step further by showing that cognitive control processes itself can be triggered unconsciously. So, a top-down task set does not only seem to influence the extent of neural processing of unseen events (Dehaene et al., 2006; Nakamura et al., 2006, 2007), but also the opposite seems to be case: unconscious stimuli seem able to exert a form of cognitive control (influence whether a task will be performed or interrupted).

These results nicely converge with data from a recent and elegant fMRI study of Lau and Passingham (2007) in which participants had to prepare to perform either a phonological or a semantic judgment on a visually presented word. On a single-trial basis, a visible metacontrast cue instructed participants which task to perform. A prime resembling the instruction cue, presented just before the metacontrast instruction cue, could trigger the alternative or the same task. Masked primes triggered task-related neural activity and interfered with visibly instructed task performance. So task set (Mattler, 2003; Lau and Passingham, 2007) as well as task interruption (the present study) can be triggered unconsciously, which questions the assumption that all cognitive control functions require consciousness.

Before further interpretation, it is important to discuss one theoretical issue about our data. Typically, No-Go trials elicit larger N2 and P3 components than Go trials (Eimer, 1993; Kiefer et al., 1998; Falkenstein et al., 1999; Bokura et al., 2001; Nieuwenhuis et al., 2003; Lavric et al., 2004). We replicated this effect when we compared Go trials with conscious No-Go trials (Fig. 3). In contradiction to this result, we found a consistent P3 reduction on unconscious No-Go trials compared with Go trials at the expected scalp sites and in the expected time-window (Fig. 5). Although such qualitative differences between unconscious and conscious processes are usually difficult to find in the brain, a recent study did find these kinds of effects in a task-switching paradigm (Lau and Passingham, 2007). An advantage of the observed differences in ERP amplitude between conscious and unconscious No-Go trials is that such qualitative differences rule out the possibility that our results can be explained by assuming accidental perception of masked No-Go signals, because this would lead to similar (perhaps slightly smaller, but not opposite) results in the unconscious compared with the conscious No-Go condition.

What then is the difference between conscious and unconscious No-Go processing? To answer this question, it is important to note that we manipulated awareness of No-Go signals by means of masking. Monkey and human studies have shown that initial neural responses (the feedforward sweep) are probably partly preserved during masking, whereas feedback signals seem mostly interrupted (Lamme et al., 2002; Del Cul et al., 2007; Fahrenfort et al., 2007). These results indicate that the feedforward sweep alone is not sufficient to produce a conscious percept (no matter what area in the brain is activated) (Thompson and Schall, 1999; Lamme and Roelfsema, 2000; Lau and Passingham, 2007). On the contrary, recurrent interactions between high- and low-level areas seem to be crucial for (visual) awareness (Di Lollo et al., 2000; Pascual-Leone and Walsh, 2001; Ro et al., 2003; Dehaene et al., 2006; Lamme, 2006; Fahrenfort et al., 2007). Recurrent interactions initiate a long-lasting pattern of widespread neural activity, whereas the strength of the feedforward sweep decays rapidly with depth because it is not boosted by recurrent

processes (Lamme and Roelfsema, 2000; Dehaene and Naccache, 2001; Dehaene et al., 2006). This could explain why the effect of unconscious No-Go signals on subsequent behavior is relatively small compared with the effect of unconscious stimuli on lower-level perceptual or response-related processes (usually in the order of 10–100 ms), associated with more posterior brain areas (for a similar argument see Dehaene, 2008). Thus, because the strength, duration, and scope of neural activity differs substantially between conscious and unconscious No-Go signal processing, it seems plausible that conscious No-Go signals can initiate full-blown inhibitory control on the majority of trials, whereas unconscious No-Go signals are able to trigger (probably prefrontal) inhibitory processes; however, generally this process does not run to completion.

Another important question is why a small number of unconscious No-Go signals are capable of triggering complete response termination, whereas the majority of unconscious No-Go trials do not (and trigger an increase in RT). As we have shown, the impact of unconscious No-Go signals on RTs differs substantially across subjects. It is likely that the strength of unconscious No-Go signal processing also differs substantially across trials (e.g., because certain prestimulus conditions fluctuate), which might cause the behavioral (and neural) effects to differ across subsequent trials. In such a scheme, the strength of single-trial evoked neural activity caused by the Go signal might have to compete with the strength of neural activity caused by the unconscious No-Go signal. The outcome of this dynamic interaction between both processes might determine the consequence of No-Go signals on subsequent behavior. On the majority of conscious No-Go trials, the evoked No-Go activity is strong enough to override the Go activity, which causes complete response termination. However, activity evoked by unconscious No-Go signals is weaker, often too weak to win the race against the Go process. Therefore, in general, unconscious No-Go signals cause an increase in RT, but not result in actual stopping. However, on some unconscious No-Go trials, the evoked No-Go activity may have been sufficiently strong, or the evoked Go activity sufficiently weak (or both), such that the relative strength of the unconscious No-Go activation is sufficient to trigger inhibition. Whether this is indeed the case is an important avenue for future research.

Traditionally, the neural processing of unconscious information has been thought to be limited in scope and depth (for review, see Kouider and Dehaene, 2007). Among all cognitive functions, high-level cognitive control functions of the PFC seem the ones most likely to require conscious experience (for review, see Dehaene and Naccache, 2001; Hommel, 2007). Our results show that inhibitory control functions can be influenced by unconscious events. Although never demonstrated before, these findings are in line with recent theoretical models concerning the neural correlates of consciousness and the potential depth of processing of unconscious information (Dehaene et al., 2006; Lamme, 2006).

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