tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Journal of Experimental Psychology:
Human Perception and Performance

0096-1523/15/$12.00 http://dx.doi.org/10.1037/a0038654

AQ: 1

The Temporal Dynamic of Automatic Inhibition of Irrelevant Actions

AQ: au

Anne Atas and Axel Cleeremans
Université Libre de Bruxelles and Consciousness, Cognition
AQ: 2 and Computation Group (CO3), Bruxelles, Belgium

Motor inhibition can occur even without conscious perception and any voluntary effort. Although it is now clear that such an inhibitory process needs time to unfold, its exact temporal dynamic remains to be elucidated. Therefore, the present study aims to examine the impact of various temporal factors on automatic motor inhibition using the masked priming task. Results shows that this process can be modulated by any factor that introduces time between the mask onset and the execution of target
AQ: 3 response, whether it stems from a purely external origin (mask-target SOA), a purely internal origin (spontaneous reaction time [RT] fluctuations), or a mix of both (RT fluctuations from the target sequence). Moreover, when the external temporal factor could not determine the direction of prime influence, the RT fluctuations had the strongest impact on the priming effect. These RT fluctuations are plausibly because of spontaneous trial-to-trial changes from more impulsive and error-prone decisions to more cautious and accurate decisions to the target. Indeed, both accuracy and speed were equally required during the task, but both requirements are impossible to achieve perfectly in every trial. This suggests that fluctuations in the level of caution in voluntary decisions can modulate unconscious and involuntary motor inhibition.

Keywords: automatic motor inhibition, fluctuations of RT, mask-target SOA, random line masking, positive and negative compatibility effects

Supplemental materials: http://dx.doi.org/10.1037/a0038654.supp

Perceiving stimuli from our environment can activate specific actions that we have associated with them, even if we have no intention to respond to these stimuli (Gibson, 1979; Grèze & Decety, 2002; Tucker & Ellis, 1998). Such action affordances allow us to quickly react to our environment and perform routines of everyday life. However, automatic actions might sometimes interfere with our voluntary action selection, to such an extent that premature and irrelevant responses can appear. Inhibition of these unwanted actions is thus crucial to behave in accord with our intentions (Miller, 2000; Ridderinkhof, 2002a, 2002b; Ridderinkhof, van den Wildenberg, Wijnen, & Burle, 2004; Sumner et al., 2007). Note that such selective inhibitory mechanisms might sometimes appear maladaptive, because they suppresses actions that might subsequently be required. Nonetheless, it is a small cost relative to risking the execution of an irrelevant action (Sumner et al., 2007). It is important that although selective inhibitory mechanisms are needed to improve voluntary action selections, they are not necessarily under conscious, voluntary control.

Indeed, several studies have suggested that selective motor inhibition can take place automatically and without any voluntary effort (e.g., Eimer & Schlaghecken, 2003; Sumner et al., 2007). This automatic form of motor inhibition was typically observed in the context of the masked arrow priming task, during which participants are instructed to respond as fast and accurately as possible to the direction of a target arrow, which could either point to the left or right (≪ or ≫). Crucially, the target is preceded by a masked arrow that could either point in the same direction as the target (compatible condition) or in the opposite direction (incompatible condition). This masked arrow is generally not consciously perceived, and participants are not explicitly instructed to inhibit some responses during the task (in opposition to van Gaal, Ridderinkhof, Scholte, & Lamme, 2010), suggesting that inhibition cannot be under conscious, voluntary control. Supporting a low-level locus of the inhibitory mechanism, several studies suggested that the supplementary motor area (SMA; a posterior area of the frontal cortex interconnected with the primary motor area) is the brain area that causes the inhibitory process in the masked priming task (Boy, Evans, et al., 2010; Sumner et al., 2007).

It is now well accepted that two important experimental manipulations are necessary to observe an automatic motor inhibitory effect (for a review, see McBride, Boy, Husain, & Sumner, 2012).

First, the delay between the backward mask and the target arrow plays a major role in the occurrence of automatic motor inhibition. It has been systematically found that when the mask-target SOA is very short (about 0–40 ms), responses are faster and more accu-

Anne Atas and Axel Cleeremans, Center for Research in Cognition and Neurosciences (CRCN) and ULB Neuroscience Institute (UNI), Université Libre de Bruxelles, Bruxelles, Belgium; Consciousness, Cognition and Computation Group (CO3), Bruxelles, Belgium.

This research was supported by the Fund for Scientific Research (F.R.S.-FNRS, Belgium) and by the Interuniversity Attraction Poles Grant P7/33 from the Belgian Science Policy Office to Anne Atas and Axel Cleeremans. The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Correspondence concerning this article should be addressed to Anne Atas, Consciousness, Cognition and Computation Group, Université libre de Bruxelles—Av. F. Roosevelt 50/122–1050 Bruxelles, Belgium. E-mail: aatas@ulb.ac.be

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

rate in compatible than in incompatible trials (positive compatibility effect, PCE). This PCE reflects automatic motor activation of the prime influencing the target response. In contrast, when the mask-target SOA is long (about 100–200 ms), responses are slower and less accurate in compatible than in incompatible trials. This Negative Compatibility Effect (NCE) is thought to reflect the subsequent inhibition of the prime response activation (Aron et al., 2003; Boy et al., 2010; Eimer & Schlaghecken, 2003; Sumner et al., 2007).

Second, the mask must share no relevant stimulus features with the prime, otherwise the NCE may reflect object-updating instead of motor inhibition. Object-updating is thought to account for the NCE observed in studies that use pattern masks constructed by superimposing the two alternative prime stimuli (Eimer & Schlaghecken, 1998; Schlaghecken & Eimer, 2000), or metacontrast masks, in which the internal contours correspond to the contours from the superimposition of the two alternative primes (Eimer, 1999; Lingnau & Vorberg, 2005; Mattler, 2006). According to the object-updating theory (Lleras & Enns, 2004, 2006; Verleger, Jaśkowski, Aydemir, van der Lubbe, & Groen, 2004), each new object presented to an observer becomes integrated into an already existing percept, so that if the percept changes, the existing percept is replaced by the most recent update. Therefore, when the prime (e.g., a right-pointing arrow) is rapidly followed by the mask (i.e., composed of both right-pointing and left-pointing arrow features), the updated object (the novel element added to the percept) consists of those features of the mask that are not present in the prime; that is, a left-pointing arrow. In this context, the observation of an NCE simply reflects that the updated arrow induces priming by itself and always points in the opposite direction to the prime arrow. It is important that several studies have demonstrated an NCE, even with masks sharing no relevant stimulus features with the prime (Boy & Sumner, 2010; Klapp, 2005; Schlaghecken & Eimer, 2006; Sumner, 2008). For instance, a significant NCE was found with masks that contained vertical and horizontal random lines when the primes and targets were left-pointing and right-pointing double arrows (Schlaghecken, Bowman, & Eimer, 2006). In such a case, the NCE is likely to be the result of automatic inhibitory mechanisms (for a similar instance, see Boy & Sumner, 2010). For random-line masks (i.e., masks constructed by combining all the possible line orientations), an NCE was also observed when the primes and targets were left-pointing and right-pointing double arrows (used in e.g., Eimer & Schlaghecken, 2002; Praamstra & Seiss, 2005; Schlaghecken & Eimer, 2002, 2004; Seiss & Praamstra, 2004). This random-line mask is not fully irrelevant because it might be composed of oblique lines of the same orientation as that of the prime stimuli. However, several findings suggest that object-updating might not play a major role in NCE with random line masks. For instance, Sumner (2008) found that the NCE was not different between classical random-line masks and fully irrelevant masks. More recently, Atas, San Anton, and Cleeremans (2014) showed that the origin of the NCE obtained with random line masks was purely motor, which is consistent with the motor inhibitory account. This NCE was not correlated with the NCE obtained with metacontrast masks, which elicited strong perceptual object updating effects. Taken together, these studies suggest that NCEs obtained with random line masks are very similar to those obtained with fully irrelevant masks and are likely to be the result of motor inhibitory mechanisms.

The fact that participants are not aware of the brief task-irrelevant stimulus and that they are not instructed to employ response inhibition during the task suggest that the observed motor inhibition (i.e., the NCE) takes place automatically and without any conscious intention to suppress the prime response. Note, however, that unawareness of the stimuli is not necessary to observe this effortless and automatic inhibitory process: NCEs have been observed when random-line mask stimuli were presented after the prime but not in the same location, so that the prime was perfectly visible (e.g., Jaśkowski, 2008). This latter study also suggests that the automatic motor inhibitory process is triggered by the occurrence of the mask after the prime. This mask is thought to indicate that the ongoing action elicited by the prime is possibly premature and wrongly prepared (Jaśkowski, 2008; Jaśkowski, Białuńska, Tomanek, & Verleger, 2008; Jaśkowski & Przekoracka-Krawczyk, 2005; Jaśkowski & Verleger, 2007). Thus, after the occurrence of the mask, inhibition can be initiated and then can be completed when the delay between the mask onset and the target onset is sufficiently long.

However, besides the mask-target SOA and the type of mask, a third factor that could also play a major role on automatic motor inhibition is fluctuations of reaction time (RT) to the target. If motor inhibitory mechanisms need time to become effective, then more time between the mask onset and the target response should produce more inhibition no matter of the source of time examined. The response latency to the target can thus be seen as another temporal factor that could modulate automatic inhibition in the same direction as the Prime-Target SOA factor. Relevant with this hypothesis, previous studies using other types of conflict tasks showed a decrease or reversal of the compatibility effect (CEs) across the levels of response latency (De Jong, Liang, & Lauber, 1994; Hommel, 1997; Ridderinkhof, 2002a; Valle-Inclán & Redondo, 1998; Wascher, Schatz, Kuder, & Verleger, 2001). This suggests that motor inhibitory processes can be revealed through the RT distributional analysis (see Ridderinkhof, 2002a, 2002b; Ridderinkhof et al., 2004). These studies used mainly the Simon task, which consists of choosing between a left- and right-button press according to the color of a stimulus presented either on the left or on the right of a fixation point (Craft & Simon, 1970). Noteworthy, selective motor inhibition processes in this task are thought to be effortful and under voluntary control. Consistent with this view, a functional MRI (fMRI) study showed that individual differences in the efficiency to implement inhibitory control in the Simon task were associated with differences in the right inferior frontal cortex (Forstmann, van den Wildenberg, & Ridderinkhof, 2008), a prefrontal area typically involved in effortful and voluntary suppression of actions (Aron & Poldrack, 2006; Chevrier, Noseworthy, & Schachar, 2007; Konishi et al., 1999; Leung & Cai, 2007; Li, Huang, Constable, & Sinha, 2006).

Slower decisions to the target might reveal more motor inhibition of irrelevant actions not only in the Simon task, but also in the masked priming task. This will extend the role of RT fluctuations to the case of automatic motor inhibition. However, at present, the influence of RT fluctuations on the NCE has only been investigated in few studies, all of which have produced contradictory results. Both Eimer (1999) and Schlaghecken and Eimer (2000) found that the NCE increases with response latency, whereas

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Verleger, Jaśkowski, Aydemir, van der Lubbe, and Groen (2004) observed that its influence was minimal. Noteworthy, Eimer (1999), Schlaghecken and Eimer (2000), and Verleger et al. (2004) all used masks consisting of superimposed left and right arrows. Therefore, the NCE obtained with these masks more likely reflects a CE stemming from objet-updating rather than from automatic inhibition of the prime. More recently, using random-line masks and a mask-target SOA of 150 ms, Maylor, Birak, and Schlaghecken (2011) have shown that the NCE had the same magnitude over the different bins of RT in a group of young adults. Based on these results, it seems that response latency does not modulate automatic motor inhibition in the young population. In contrast, the CE was positive or null for fast responses and negative for slow responses in a group of older adults. These results might suggest that the NCE is delayed in older adults compared with younger adults. If this hypothesis is correct, then the delay of 150 ms between the mask onset and the target onset might already be sufficient in young adults to completely inhibit the prime motor activation even at the fastest responses, suggesting that response time might lose its power to modulate motor inhibition at this long SOA. Thus, it remains possible that response latency to the target might also modulate the direction of the CE in young adults at shorter mask-target SOAs.

In the present study, we investigated whether RT fluctuations could modulate automatic motor inhibition in young adults across different mask-target SOA conditions. To address this issue and ensure that the NCE indeed reflects prime-induced inhibition, we used masks consisting of random lines (see Sumner, 2008). The first goal of the present study was to explore the role that response latency to the target plays in the onset of automatic motor inhibition. We hypothesized that longer target RTs would be associated with more negative values of the CE (i.e., more automatic motor inhibition). The second goal was to examine the relationship between time from mask-target SOA and time from response latency on inhibition. If the impact of response latency on the NCE was confirmed, how is this impact modulated by the mask-target SOA? There are at least two (not incompatible) reasons to hypothesize that both temporal factors should interact. The first reason is related to the fact that mask-target SOA and response latency represent two qualitatively different variations of time. The mask-target SOA is an external manipulation of the temporal factors taking place within the stimulation that strongly constrains the sequence of prime-driven activation and target-driven activation (Aron et al., 2003; Eimer & Schlaghecken, 2003). In contrast, RT fluctuations represent an internal variation of time that occurs independently from the stimulation and are more related to internal fluctuations in decision-making processes (Bogacz et al., 2010). These two sources of time (external and internal) are opposite and might modulate the influence of each other during the dynamic of activation-suppression of the prime motor representation. This hypothesis is based on previous studies showing that stimulus factors (i.e., stimulus strength) modulate the weight of the influence of internal fluctuations (i.e., prestimulus neuronal activity) on perceptual decisions (Bode et al., 2012; Shadlen & Newsome, 2001): When the stimulus strength was weak, the influence of internal fluctuations was stronger on perceptual decisions than when the stimulus strength was strong. In a similar way, because intermediate Masked-Target SOAs of 60–80 ms are external stimulus factors that fail to determine the direction of the CE (the CE is typically null at these SOAs), RT fluctuations might have a greater impact on the CE at these SOAs than at short or long SOAs (i.e., an SOA of 0 ms with a strong PCE or of 100–150 ms with a strong NCE). Second, previous research showed that the magnitude of motor inhibition (NCE) depends on the magnitude of the motor activation (PCE) elicited by the prime (Boy et al., 2010; Boy & Sumner, 2010). We thus expect that once sufficient time is provided by the mask-target SOA to completely inhibit the prime motor activation (i.e., SOAs of 100–150 ms), response time will lose its power to modulate motor inhibition, simply because there is nothing else (no more positive motor activation) to inhibit.

Finally, because fluctuations of RTs across trials might partly stem from systematic sources originating in the external environment, we also studied the impact of a systematic source of RT fluctuations on the CE; that is, the target sequence. In two-alternative forced-choice tasks, previous targets modulate the RT on the current target in a systematic manner, even though target sequences are randomly ordered (Gao, Wong-Lin, Holmes, Simen, & Cohen, 2009; Soetens, Boer, & Hueting, 1985). When the response-to-stimulus interval (RSI) is sufficiently long (e.g., 500–1,000 ms), previous targets modulate strategic expectancies about the actual target: RT tends to be shorter if the current target confirms the expectation (Gao et al., 2009). In the present study, we therefore used a long RSI of about 1,600 ms to track such expectancy-driven RT fluctuations. Crucially, at first glance, the target sequence variable has no link with automatic motor inhibition. Then, the demonstration that such a variable can modulate motor inhibition simply because this variable modulates RTs has an important implication in the field. Indeed, this would suggest that automatic motor inhibition is potentially modulated by many factors that were previously considered as no relevant (i.e., any factor that modulates RT).

To summarize, the purposes of the present study were (a) to investigate the impact of RT fluctuations on the time-course of automatic inhibition of a masked prime; (b) to test whether its impact interacts with the impact of mask-target SOA on inhibition; and (c) to track whether a systematic source of RT fluctuations stemming from the situation's context might account for the inhibition observed: RT fluctuations because of target sequence.

Method

Experiments 1a and 1b were designed to examine precisely the evolution of the CE through response latency: The CE was examined across 10 levels of response latency (i.e., at the level of RT deciles). To have a sufficient number of trials in each subcondition (crossing the levels of the compatibility, mask-target SOA, and response latency variables), we only used 4 mask-target SOAs in each of these experiments. The mask-target SOAs of 0, 50, 100, and 150 ms were examined in the Experiment 1a. These SOAs are known to produce either strong PCEs (SOAs of 0 and 50 ms) or strong NCEs (SOAs of 100 and 150 ms), and are thus used in the majority of the masked priming studies with arrow primes. In contrast, the mask-target SOAs of 60, 70, 80, and 90 ms were examined in the Experiment 1b. These SOAs are known to produce no sizable CEs (typically null or small effects), and for this reason, they are not frequently used in masked priming studies with arrow primes.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Experiment 2 was designed to examine precisely how the impact of response latency on CE differs across the levels of mask-target SOAs. For this experiment, we used the 8 mask-target SOAs (0, 50, 60, 70, 80, 90, 100, and 150 ms), but the CE was examined only across 5 levels of response latency (i.e., at the level of RT quintiles). Therefore, the same number of trials was used in each subcondition (crossing the levels of the compatibility, mask-target SOA, and response latency variables) of each experiment. Except the specificities mentioned earlier, all apparatus, stimuli, and procedures were identical in the three experiments.

Participants

Fifty students (42 women; mean age = 21.5) from the Université Libre de Bruxelles were tested and included in the participant samples: Sixteen, 15, and 19 students participated in Experiments 1, 2, and 3, respectively. Six more participants were tested but discarded from the analyses because they were able to discriminate the prime stimulus, as evidenced by above-chance performance during a discrimination test on the arrow prime ($p < .05$, χ^2 test, see below for further details on this visibility test). Participants all reported normal or corrected-to-normal vision and were naive to the purpose of the experiment. They were paid €8 for their participation.

Apparatus and Stimuli

Stimuli were displayed on a CRT monitor (Philips 107T, resolution 800 × 600) at a refresh rate of 100 Hz. Subjects viewed the screen from a distance of 70 cm. Responses were executed with the index fingers of both hands and collected through the two extreme keys of a button box (E-prime 1.1, PST software, Pittsburgh, PA). All stimuli were black and displayed at the center of the screen on a white background. Both prime and target stimuli were left- and right-pointing double arrows (≪ and ≫) subtending a visual angle of approximately 1.6° × 0.7°. Masks were made up of 35 randomly orientated lines of different length positioned on a virtual grid of 3.3° × 2.7° (Figure 1). Fifty different masks were created, and one was randomly selected on each trial. The fixation cross subtended a visual angle of approximately 0.6° × 0.6°. The fixation cross, the prime, and the mask stimuli appeared at the center of the screen, whereas the target was randomly presented just above or below the mask (the distance from the center of the screen to the center of the target arrow subtended a visual angle of 1.8°).

Design and procedure. In the priming task, participants had to make quick and accurate responses with a left- or right-hand key press to the orientation of the target arrow. The prime was either identical to (compatible trials) or opposite of (incompatible trials) the target arrow stimulus. Each trial started with a fixation cross that was presented for 500 ms, followed by a blank of 300 ms. The prime was then presented at the center of the screen for 30 ms and was immediately followed by a mask (100 ms). The target was presented for 100 ms, either above or below the mask. The interval between the onset of the mask and the onset of the target (the mask-target SOA) was systematically manipulated. Depending on the experiment, 4 or 8 of the following mask-target SOAs were used: 0, 50, 60, 70, 80, 90, 100, and 150 ms (Figure 1). After the offset of the target, a blank screen was displayed until participants had responded. The intertrial interval consisted of an additional blank screen presented for 700 ms. Considering the target as the stimulus, the RSI ranged from 1,520 ms to 1,670 ms (Figure 1). The priming task consisted of 16 experimental blocks of 80 trials each, separated by shorts breaks of minimum 30 s. Each block contained only trials with one specific mask-target SOA condition. The order of the 16 blocks was counterbalanced between participants.[1] Each block contained an equal number of compatible and incompatible trials, presented in a randomized order. Performance feedback was presented after each block (mean RTs and percentage of correct responses to the target). A practice block of 20 trials preceded the 16 experimental blocks. At the end of the experiment, visibility of the prime was evaluated by a discrimination test on the arrow prime. Prime and mask stimuli were presented in the same way as in the main experiment, but the target was removed. After the offset of the mask, either the question "left?" or "right?" was displayed until participants had responded, which could be done without time pressure. Participants had to respond "yes" with the left index if the direction of the prime arrow corresponded to the question, and "no" with the right index if it did not. The visibility task consisted of 48 trials. The different prime-target-question relations were all represented equally and randomly presented.

Data analysis. The first trial of each block, as well as any trial in which RT <100 or >1,000 were excluded from the analyses (<1%).[2] For any analysis on RTs, errors were also excluded.

For the analysis of "*CE independent of response latency*," mean error percentages and correct RTs were each submitted to a repeated measure analysis of variance (Greenhouse-Geisser correction) with mask-target SOA (for the specific SOAs used in each experiment, see above) and compatibility (compatible or incompatible) as within-subject factors.

For the analysis of "*CE on RTs across different levels of response latency*," we used the RT distributional analysis procedure (De Jong et al., 1994; Hommel, 1997; Ridderinkhof, 2002a; Valle-Inclán & Redondo, 1998; Wascher et al., 2001). RTs were rank-ordered separately for each participant and for each of the 8 (or the 16) conditions created by crossing the 2 levels of compatibility and the 4 (or the 8) levels of mask-target SOA. Then, for each subject and for each of these conditions, RTs were separated into 10 (or 5) speed bins, which were determined by the specific decile (or quintile) values. Therefore, each speed bin contained almost the

[1] In the Experiments 1a and 1b, each participant performed one of the two predetermined orders of SOA blocks. The SOA of the successive blocks in Experiment 1a were either (1) 0, 0, 0, 0, 50, 50, 50, 50, 100, 100, 100, 100, 150, 150, 150, and 150 ms, or (2) 150, 150, 150, 150, 100, 100, 100, 100, 50, 50, 50, 50, 0, 0, 0, and 0 ms. The SOA of the successive blocks in Experiment 1b were either (1) 60, 60, 60, 60, 70, 70, 70, 70, 80, 80, 80, 80, 90, 90, 90, and 90ms, or (2) 90, 90, 90, 90, 80, 80, 80, 80, 70, 70, 70, 70, 60, 60, 60, and 60 ms. In the Experiment 2, each participant performed one of the four predetermined orders of SOA blocks. The SOA of the successive blocks were either (1) 0, 0, 50, 50, 60, 60, 70, 70, 80, 80, 90, 90, 100, 100, 150, and 150 ms, (2) 150, 150, 100, 100, 90, 90, 80, 80, 70, 70, 60, 60, 50, 50, 0, and 0 ms, (3) 80, 80, 90, 90, 100, 100, 150, 150, 70, 70, 60, 60, 50, 50, 0, and 0 ms, or (4) 70, 70, 60, 60, 50, 50, 0, 0, 80, 80, 90, 90, 100, 100, 150, and 150 ms.

[2] Because participants started each block by pressing the spacebar of the keyboard and then responded to the target by choosing between the two extreme keys of the button box, the RT of the very first trial was affected by the changing of devices. For this reason, it was systematically not analyzed.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

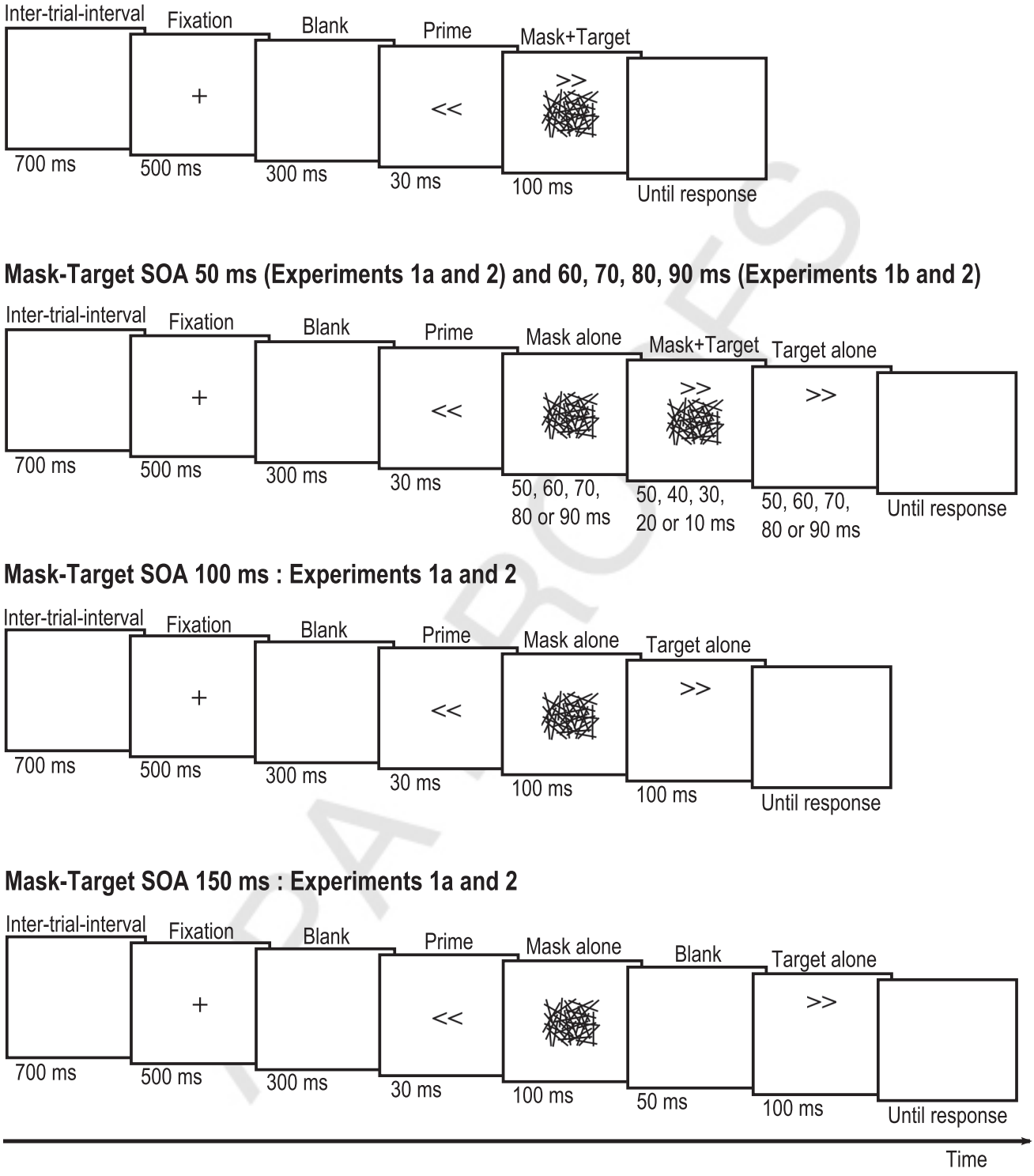


Figure 1. Trial procedure of the priming task. Trials were compatible when prime and target arrows pointed in the same direction, incompatible when they pointed in opposite directions. For mask-target SOA 0 blocks, targets appeared together with (above or below) the mask. For mask-target SOA 50, 60, 70, 80, or 90 blocks, the mask was first presented alone and then together with the target, which remained alone on the screen after the offset of the mask. In mask-target SOA 100 blocks, targets appeared immediately after the offset of the mask. In mask-target SOA 150 blocks, targets appeared 50 ms after the offset of the mask.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

same number of trials (about 16 trials). Finally, the mean RT was calculated for each speed bin in each condition for each participant. As a consequence, the global mean of RTs was similar to the average of the mean RT of the 10 (or 5) speed bins. This procedure was carried out so as to make it possible to create a new variable, "response latency," with 10 (or 5) levels, ranging from decile 1 (or quintile 1; i.e., the first speed bin with very fast trials) to decile 10 (or quintile 5; i.e., the last speed bin with very slow trials). The same analysis of variance (ANOVA) design as in the previous analysis of RTs was used, but included the new variable response latency with 10 levels (or 5 levels).

The purpose of the Experiment 2 was to examine how response latency exerts a different modulatory influence on the CE dependent upon the level of mask-target SOA. To do so, one method would be to compare the CEs from each sublevel of mask-target SOA × Response Latency. However, this is not efficient enough to get a clear picture of the results, because multiple analyses can detect even subtle differences that are not relevant. Instead, we used another method that consisted of *computing the slope of the 5 points corresponding to the CEs of the 5 quintiles* for each mask-target SOA and for each participant. A negative value of the slope for a particular mask-target SOA level means that the CE decreases or becomes more negative across the 5 quintiles for this SOA. The higher the negative value of the slope, the stronger the impact of response latency on the CE for the particular mask-target SOA level. Comparing the slope values of the different SOAs should indicate in which of the SOA conditions the impact of the response latency is the strongest. Slope values were submitted to a repeated measures analysis of variance with the mask-target SOA (0, 50, 60, 70, 80, 90, 100, and 150 ms) as a within-subject factor.

For the analysis of "*Prime visibility*," the accuracy performance in the visibility test and the d' (an index of signal detection) were each submitted to a one-sample *t*-test against chance level (50% for the accuracy performance, and 0 for the d'). Moreover, to examine whether the CEs (the CE for each mask-target SOA, both on RTs and on errors) and the prime visibility were correlated, the d' performance as it relates to each of the 8 (or 16) overall CEs was examined in 8 (or 16) correlation analyses.

Finally, for the analysis of "*Correlation with intersubject variability in response latency*," we examined whether the CEs and the intersubject variability in response speed (i.e., the fact that participants differ regarding their overall response speed to the target) were correlated. The overall response speed as it relates to each of the 4 (or 8) overall CEs on RTs was examined in 4 (or 8) correlation analyses. We did not perform the same analysis with the CEs on errors because of the strong speed–accuracy trade-off (i.e., faster participants made more errors than slower participants); $r(1,16) = -.74$, $p = .001$ in Experiment 1a; $r(1,15) = -.72$, $p = .003$ in Experiment 1b; and $r(1,19) = -.50$, $p = .03$ in Experiment 2.

Results of the Experiment 1a

CE (on errors and RTs) independent of response latency. The CE decreased (became more negative) with the levels of mask-target SOA, both on RTs, $F(1.9, 28.3) = 74.63$, $p < .001$, and on errors, $F(1.4, 21.5) = 10.91$, $p = .001$; mean error = 4% (Figure 2). Planned comparisons indicated a significant PCE on both measures at the SOA 0 ms, $t(15) = 3.54$, $p = .003$, PCE = 3.8%; $t(15 = 10.42$, $p < .001$, PCE = 48 ms), and at the SOA 50 ms, $t(15) = 3.37$, $p = .004$, PCE = 3.9%; $t(15 = 4.21$, $p = .001$, PCE = 21 ms. Thus, PCEs were found at the short SOAs, which reflect the initial motor activation of the prime. In contrast, an NCE was found both at the SOA 100 ms, $t(15) = -1.87$, $p = .081$, NCE = −1.4%, $t(15 = -2.37$, $p = .032$, NCE = −11 ms, and at the SOA 150 ms, $t(15) = -2.18$, $p = .046$, NCE = −1.9%; $t(15) = -2.89$, $p = .011$, NCE = −15 ms. Thus, NCEs were found at the long SOAs, which reflect the subsequent inhibition of the prime motor activation.

CE (on RTs) across different levels of response latency. The CE decreased with the levels of response latency, $F(1.5, 22.9) = 26.79$, $p < .001$. Large PCEs were found at fast RTs, whereas no CE or small NCEs were found at slow RTs (Table 1). Moreover, this CE decrease with response latency was modulated by the mask-target SOA, $F(3.8, 57.6) = 3.18$, $p = .021$ (Figure 2 and Table S1a [available online as supplemental material]).

Prime visibility. Participants were unable to perceive the prime arrows, mean accuracy = 48.9%, $t(15) = -0.75$, $p = .463$; mean $d' = -0.06$, $t(15) = -0.77$, $p = .454$. Moreover, the CEs and the prime visibility seem to be independent (none of the correlations were significant, all *p*s > .096).

Correlation with intersubject variability in response latency. The CEs and the intersubject response speed also appear to be independent (none of the correlations were significant, all *p*s > .669). Therefore, while intrasubject variability in response latency modulated the CE, the intersubject variability had no impact on this factor.

Results of the Experiment 1b

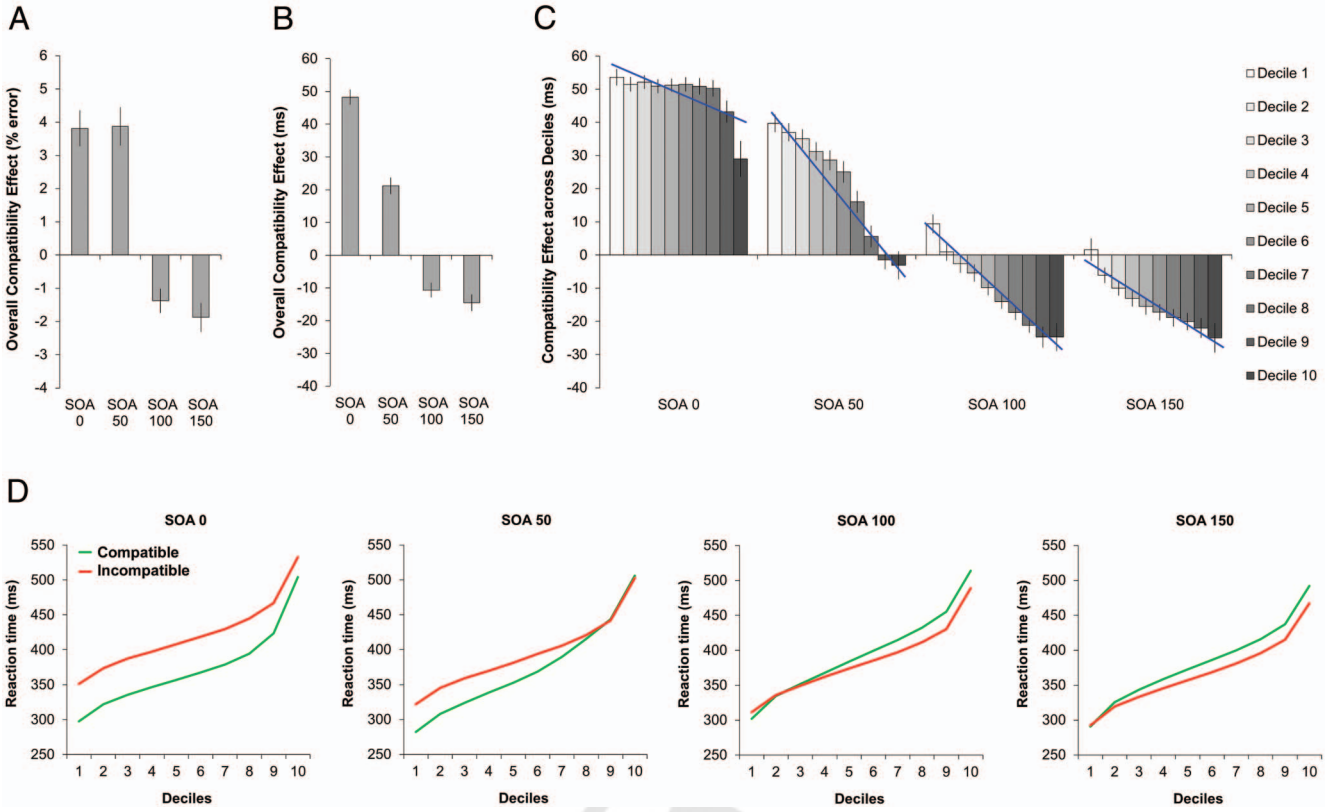
CE (on errors and RTs) independent of response latency. For errors (2.7% of trials), the interaction between mask-target SOA and compatibility was not significant, $F(2.3, 31.6) = 1.07$, $p = .371$. For correct RTs, the CE decreased with the levels of mask-target SOA, $F(2, 27.3) = 19.08$, $p < .001$ (Figure 3). Results showed a small and just significant PCE at the SOA 60 ms, $t(14) = 2.15$, $p = .049$, PCE = 10 ms; a nonsignificant effect at the SOA 70 ms, $t(14) = 0.366$, $p = .720$, CE = 2 ms; a small NCE at the SOA 80 ms, $t(14) = -2.79$, $p = .015$, NCE = −7 ms; and a larger and more robust NCE at the SOA 90 ms, $t(14) = -3.30$, $p = .005$, NCE = −12 ms.

CE (on RTs) across different levels of response latency. The CE decreased with the levels of response latency, $F(2.1, 29.7) = 33.38$, $p < .001$. Large PCEs were found at fast RTs, whereas large NCEs were found at slow RTs (Table 2). The CE decrease with response latency was not significantly modulated by the mask-target SOA, $F(4.6, 64.4) = 2.15$, $p = .075$ (Figure 3 and Table S1b). This is not surprising because these mask-target SOAs have very close values.

Prime visibility. Participants were unable to perceive the prime arrows, mean accuracy = 48.1%, $t(14) = -1.17$, $p = .260$; mean $d' = -0.10$, $t(14) = -1.14$, $p = .272$. The CEs and the prime visibility appear also to be independent (none of the correlations were significant, all *p*s > .086).

Correlation with intersubject variability in response latency. The CEs and the intersubject response speed were independent (none of the correlations were significant, all *p*s > .500).

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484



ONLINE COLOR

Figure 2. Results of Experiment 1a. (A) The overall compatibility effect (CE) on errors (incompatible-compatible trials) significantly decreases (i.e., becomes more negative) with the levels of mask-target SOA; (B) The overall CE on reaction times (RTs; incompatible-compatible trials) significantly decreases (i.e., becomes more negative) with the levels of mask-target SOA; (C) The CE on RTs decreases with the levels of response latency, and this decrease is significantly modulated by the mask-target SOA. The slopes of the CE across the 10 deciles are represented in blue for each of the mask-target SOA conditions; (D) Mean RTs for Compatible (green) and Incompatible (red) trials across the 10 deciles for each of the mask-target SOA conditions. Error bars represent 1 *SEM*. See the online article for the color version of this article.

AQ: 10

In sum, Experiments 1a and 1b replicated the previous findings: Large PCEs occurred at short SOAs, no or small CEs occurred at intermediate SOAs, and large NCEs occurred at long SOAs. More important, by using masks known to elicit inhibition of the prime motor activation, these experiments also demonstrated that faster response times were associated with more positive values of the CE, whereas slower response times were associated with more negative values of the CE. This suggests a major role of RT fluctuations on the time-course of unconscious and automatic motor inhibition.

Table 1
The Compatibility Effect Observed for Each Level of Response Latency in Experiment 1a Was Submitted to a One-Sample t-Test Against Zero

Decile	*t*	*p*	Compatibility effect
1	5.44	.000	26
2	5.15	.000	21
3	4.56	.000	19
4	4.02	.001	16
5	3.58	.003	14
6	2.98	.009	11
7	2.00	.064	8
8	0.97	.347	4
9	−0.33	.748	−1
10	−1.05	.308	−6

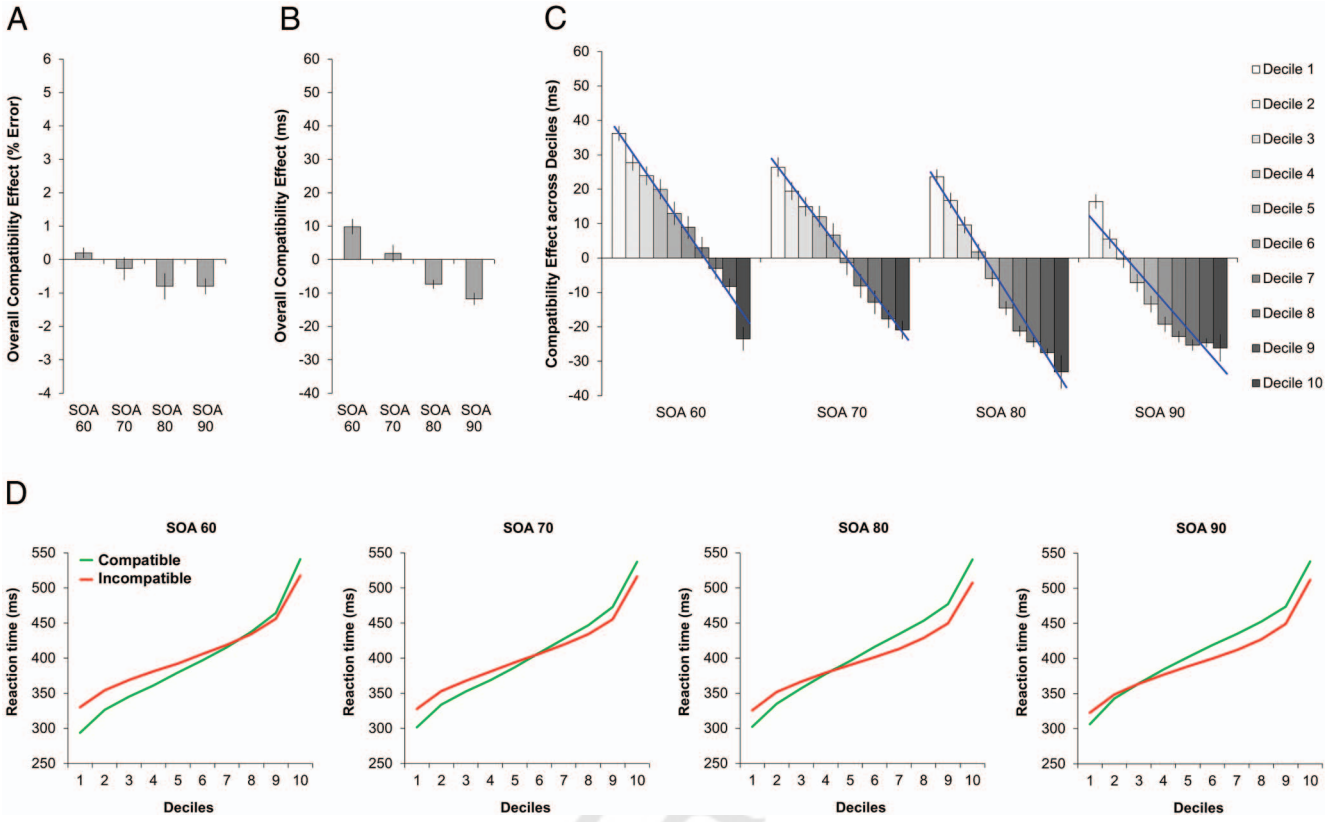
Note. Based on the Bonferroni alpha correction for the 10 tests, statistical significance was assumed at a value of $p < .005$ (alpha of .05 divided by 10 = .005).

Results of the Experiment 2

CE (on errors and RTs) independent of response latency. The CE decreased with the levels of mask-target SOA, both on RTs, $F(3.8, 67.7) = 41.33$, $p < .001$, and on errors, $F(4.2, 76) = 8.14$, $p < .001$ (Figure 4). On errors, a PCE was found at the SOA 0 ms, $t(18) = 3.93$, $p = .001$, PCE = 4.3%; no significant effect was found at the SOAs 50, 60 and 70 ms (all $ps > .204$); a marginal effect was observed at the SOA 80, $t(18) = -2.03$, $p = .057$, NCE = −1.5%; and a NCE was found at the SOAs 90, 100 and 150 ms, all $ts(18) < -2.97$ and $ps < .01$, with an NCE of −2.0%, −2.3% and −2.6%, respectively. Similarly on RTs, a PCE was found at the SOA 0 ms, $t(18) = 8.64$, $p < .001$, PCE = 36 ms; no significant effect was found at the SOAs 50 and 60 ms ($ps > .232$); and a significant NCE was observed at the SOAs 70,

F4

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484



ONLINE COLOR

Figure 3. Results of Experiment 1b. (A) The overall compatibility effect (CE) on errors does not significantly decrease with the levels of mask-target SOA; (B) The overall CE on reaction times (RTs) significantly decreases (i.e., becomes more negative) with the levels of mask-target SOA; (C) The CE on RTs decreases with the levels of response latency, but this decrease is not significantly modulated by the mask-target SOA. The slopes of the CE across the 10 deciles are represented in blue for each of the mask-target SOA conditions; (D) Mean RTs for compatible (green) and incompatible (red) trials across the 10 deciles for each of the mask-target SOA conditions. Error bars represent 1 *SEM*. See the online article for the color version of this article.

AQ: 11

80, 90, 100, and 150 ms, all $ts(18) < -3.65$ and $ps < .003$, with an NCE of −16 ms, −15 ms, −21 ms, −20 ms, and −19 ms, respectively). Note that a large PCE was found at the SOA 50 ms in Experiment 1a, while the effect was already null here, suggesting that the reversal (inhibition) occurred earlier in this Experiment 2.

CE on RTs across different levels of response latency. The CE decreased with the levels of response latency, $F(1.8, 32) = 102.36$, $p < .001$ (Table 3). Moreover, this decrease of the CE with response latency was modulated by the mask-target SOA, $F(5.1, 91) = 4.81$, $p = .001$ (Figure 4 and Table S2).

T3, AQ:4

Slopes analysis. Consistent with the significant 3-way interaction obtained in the previous analysis, the effect of mask-target SOA was significant, $F(3.1, 56.5) = 6.29$, $p = .001$, confirming that the CE decrease with response latency (the negativity of the slope value) was modulated by the mask-target SOA. The slope values of the SOAs 0, 50, 60, 70, 80, 90, and 100, each tested against zero (one-sample *t* test), were all significant and negative (all $ps <$ .006 with slope values of −7.18, −13.07, −14.38, −12.47, −10.11, −7.69, and −8, respectively). The slope of SOA 150 ms was marginally significant, $t(18) = -2.06$, $p = .054$, slope = −2.02. Thus, except for the longest SOA condition, the CE significantly decreased with response latency. The slope of the SOA 150 ms may have failed to reach significance because of the more important decay associated with the unconscious neural representation of the prime at this longer SOA (Gaillard et al., 2009; Greenwald, Draine, & Abrams, 1996). Indeed, for the quintile 5 of the SOA 150 ms, the Mask-Response SOA (the delay between the mask onset and the overt response) was longer than in all other Mask-Response conditions (Figure 5). For this reason, the slope of this SOA might not be fully comparable with those of the other SOAs. It is important that the effect of the mask-target SOA on the slope measure was still significant when the SOA 150-ms condition was not included in the analysis, $F(2.9, 51.3) = 3.049$, $p = .039$.

F5

Regardless of the SOA 150 ms condition, results of Experiment 2 suggest that although the decrease of the CE with response latency is observed for each mask-target SOA, the decrease seems to be stronger for the more intermediate SOAs (50, 60, 70, and 80 ms with slope values of −14.5 and −13 and −12.5, and −10, respectively) than for the more extreme SOAs (0, 90, and 100 ms, with slopes values of −7, −8, and −8, respectively). A contrast test showed that the slopes of these four intermediate mask-target SOAs (50, 60, 70, and 80 ms) were significantly more negative than were the slopes of the three extreme SOAs (0, 90, and 100 ms), $F(1, 18) = 11.82$, $p = .003$ (the contrast values of this test

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Table 2
The Compatibility Effect Observed for Each Level of Response Latency in Experiment 1b Was Submitted to a One-Sample t-Test Against Zero

Decile	*t*	*p*	Compatibility effect
1	6.41	.000	26
2	3.76	.002	17
3	2.44	.029	12
4	1.27	.226	7
5	0.01	.990	0
6	−1.27	.224	−7
7	−2.74	.016	−12
8	−4.23	.001	−16
9	−6.87	.000	−20
10	−5.66	.000	−26

Note. Based on the Bonferroni alpha correction for the 10 tests, statistical significance was assumed at a value of $p < .005$ (alpha of .05 divided by 10 = .005).

were −1/3, +1/4, +1/4, +1/4, +1/4, −1/3, −1/3 for the SOAs 0, 50, 60, 70, 80, 90, and 100, respectively). Follow-up analyses indicated that the slope values of SOAs 50, 60, 70, and 80 ms did not significantly differ from each other, $F(2.2, 39.8) = 1.50$, $p = .234$. Similarly, the slope values of SOAs 0, 90, and 100 ms did not significantly differ from each other, $F(1.4, 25) = 0.052$, $p = .893$.

In sum, the slope analysis showed that the decrease of the CE with response latency was stronger for the more intermediate mask-target SOAs than for more extreme ones (short or long).

The impact of mask-response delay. The variable Mask-Response SOA was created by adding the mask-target SOA value to the mean RT value of each quintile (Figure 5). Although the CE decreased over the levels of the Mask-Response SOA in a systematic manner, this SOA variable could not account for all the variations observed in the CE. The most striking instance concerns the CE at the mask-target SOA 0 ms with a response latency of 486 ms (Quintile 5) and for the mask-target SOA 70 ms with a response latency of 412 ms (Quintile 4): While both Mask-Response SOAs were about 485 ms, the former exhibited a PCE of 21 ms and the latter an NCE of 30 ms.

Prime visibility. Participants were unable to perceive the prime arrows, mean accuracy = 51.4%, $t(18) = 1.10$, $p = .284$; mean $d' = -0.06$, $t(18) = 0.86$, $p = .402$. The CEs and the prime visibility seem also to be independent (none of the correlations were significant, all ps $> .088$).

Correlation with intersubject variability in response latency. The CEs and the intersubject response speed appear to be independent (none of the correlations were significant, all ps $> .184$).

To sum up, replicating Experiment 1a and 1b, results of Experiment 2 showed that RT fluctuations play a major role on the time-course of automatic motor inhibition. Moreover, the impact of RT fluctuations on the CE varies depending on the specific mask-target SOA. That is to say, when the mask-target SOA clearly determines the direction of prime influence, as was the case for short and long SOAs (strong positive CE or strong negative CE), response latency had only a weak impact on the CE. Conversely, when the SOA was intermediate and was less efficient to determine the direction of the influence of the prime (null or smaller overall CE), response latency more strongly influenced the evolution of activation-inhibition.

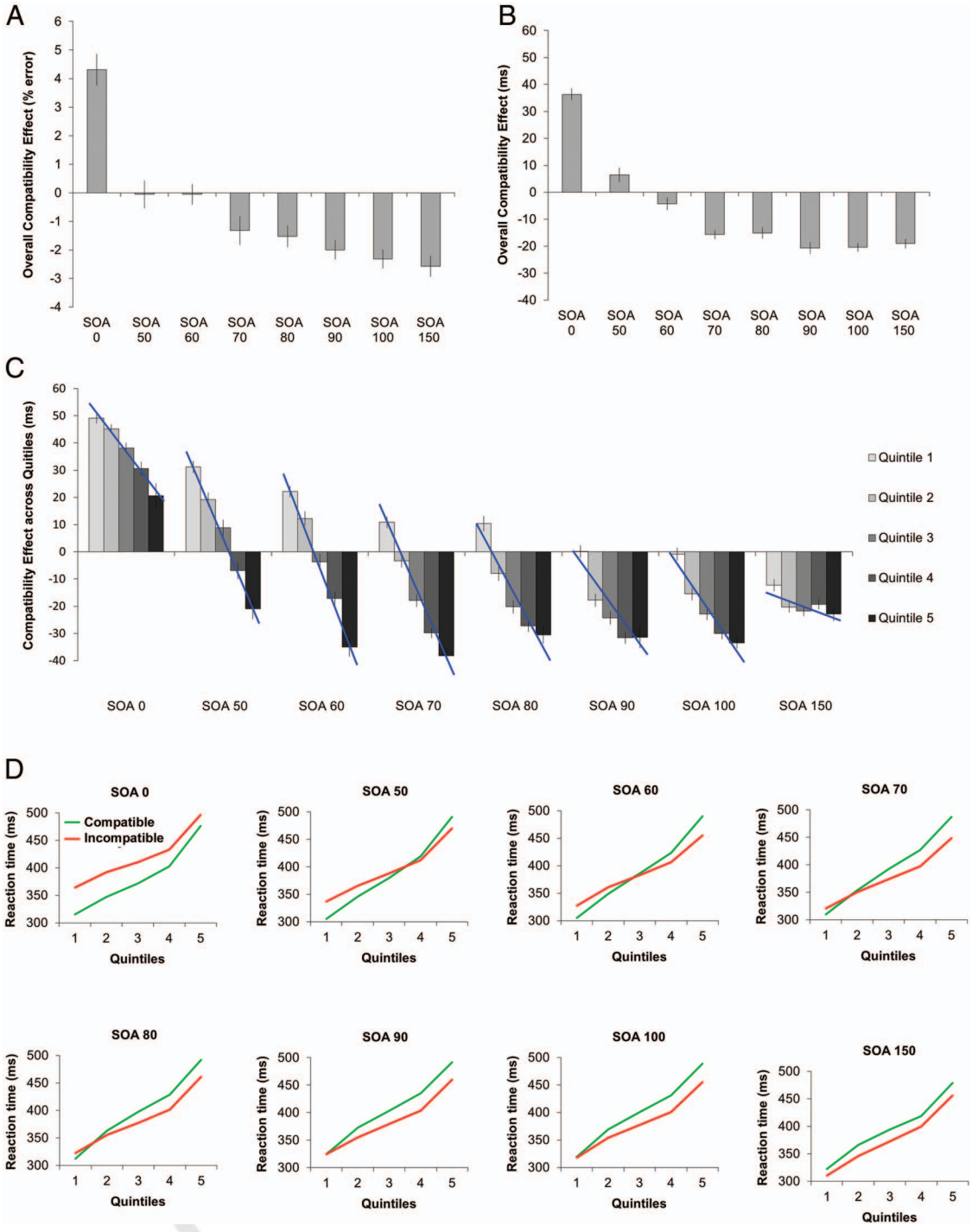
RT Variations From Target Sequence in All Experiments

RT fluctuations might partly stem from systematic sources originating in the external environment. Here, we examined whether RT fluctuations because of target sequence will modulate the CE in a relevant manner. That is to say, faster RTs because of target sequence will be associated with higher positive values of the CE, and vice versa. According to Soetens, Boer, and Hueting (1985), there are two types of RT fluctuations because of target sequence: (1) the influence of the immediately preceding target on the RT of the current trial (*first-order target sequence*), and (2) the influence of a sequence of targets on the RT of the current trial (*higher-order target sequence*). With our relatively long RSI (about 1,500 ms), it is not clear that RTs will be faster for immediate target repetitions than for immediate target alternations (Gao et al., 2009; Soetens et al., 1985). However, if this is the case, we expect that RT modulations because of first-order target sequence will modulate the CE in a relevant manner. With respect to higher-order target sequence, when the RSI is sufficiently long (e.g., 500–1,000 ms), the sequence of previous targets modulates strategic expectancies about the actual target: RTs tend to be shorter if the current target confirms the expectation (Gao et al., 2009). We expect that such expectancy-driven RT fluctuations will also modulate the CE in a relevant manner.

First-order target sequence. RT on the current trial *n* is examined depending on the type of target in the immediately preceding trial *n-1* (i.e., left or right arrow). The variable first-order sequence has two levels: *Repetition* for which the current target *n* is a repetition of the immediately preceding target *n-1* (i.e., [$\text{Left}_{\text{trial}\_n\text{-}1}$ $\text{Left}_{\text{trial}\_n}$] or [$\text{Right}_{\text{trial}\_n\text{-}1}$ $\text{Right}_{\text{trial}\_n}$]), and *Alternation* for which the current target is an alternation of the immediately preceding one (i.e., [$\text{Left}_{\text{trial}\_n\text{-}1}$ $\text{Right}_{\text{trial}\_n}$] or [$\text{Right}_{\text{trial}\_n\text{-}1}$ $\text{Left}_{\text{trial}\_n}$]). The first trial of each block, incorrect responses, trials following incorrect responses, and RTs <100 or >1000 were excluded from the analysis. For each experiment, mean RTs were submitted to a repeated measures analysis with first-order sequence (repetition and alternation), compatibility (compatible or incompatible), and mask-target SOA (4 or 8 levels depending on the experiment) as within-subject factors. The following paired-sample *t* tests were carried out with Bonferroni corrections (post hoc analyses).

In Experiment 1a, RTs were not significantly different between repetitions and alternations, $F(1, 15) = 1.07$, $p = .318$. However, the interaction between first-order sequence and compatibility was highly significant, $F(1, 15) = 28.18$, $p < .001$. The difference between compatible and incompatible trials for target repetitions was significant and corresponded to a PCE, $t(15) = 4.19$, $p = .003$; PCE = 16 ms; the other differences were not significant (all ps $> .08$). Finally, the impact of first-order sequence on the CE was similar over the different mask-target SOA conditions, 3-way interaction: $F(3, 45) = 0.94$, $p = .429$.

In Experiment 1b, RTs were not significantly different between repetitions and alternations, $F(1, 14) = 1.56$, $p = .233$. The interaction between first-order sequence and compatibility was significant, $F(1, 14) = 5.52$, $p = .034$, indicating that the CE had a higher positive value for repetitions (CE = 2 ms) than for alternations (CE = −6 ms). However, none of the four paired *t* tests reached significance (all ps $> .366$). As for the previous



ONLINE COLOR

Figure 4. Results of Experiment 2 (Part 1). (A) The overall compatibility effect (CE) on errors significantly decreases (i.e., becomes more negative) with the levels of mask-target SOA; (B) The overall CE on reaction times (RTs) significantly decreases (i.e., becomes more negative) with the levels of mask-target SOA; (C) The CE on RTs decreases with the levels of response latency, and this decrease is significantly modulated by the mask-target SOA. The slopes of the CE across the 5 quintiles (in blue) are more negative (the CE decreases with response latency was stronger) for the intermediate SOAs (50, 60, 70, and 80 ms) than for the more extreme SOAs (0, 90, 100, and 150 ms); (D) Mean RTs for compatible (green) and incompatible (red) trials across the 5 quintiles for each of the mask-target SOA conditions. Error bars represent 1 *SEM*. See the online article for the color version of this article.

AQ: 12

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Table 3
The Compatibility Effect Observed for Each Level of Response Latency in Experiment 2 Was Submitted to a One-Sample t-Test Against Zero

Quintile	*t*	*p*	Compatibility effect
1	4.06	.001	14
2	0.43	.670	1
3	−2.36	.030	−8
4	−5.86	.000	−16
5	−7.64	.000	−24

Note. Based on the Bonferroni alpha correction for the 5 tests, statistical significance was assumed at a value of $p < .01$ (alpha of .05 divided by 5 = .01).

experiment, the impact of first-order sequence on the CE was similar over the different mask-target SOA conditions, $F(3, 42) = 0.23$, $p = .874$.

In Experiment 2, RTs were significantly faster for repetitions than for alternations, $F(1, 18) = 4.92$, $p = .04$. Moreover, the interaction between first-order sequence and compatibility was significant, $F(1, 18) = 32.16$, $p < .001$. The CE was not significant for target repetition ($p > .990$), whereas RTs were significantly slower in compatible than incompatible trials for target alternations, $t(18) = 3.88$, $p = .004$; NCE = −13 ms. RTs were also significantly faster for compatible target repetitions than for compatible target alternations, $t(18) = 3.37$, $p = .014$; difference = 15 ms, whereas no significant difference was observed between incompatible target repetitions and alternations ($p > .890$). Again, the 3-way interaction between first-order sequence, SOA, and compatibility was not significant, $F(7, 126) = 1.13$, $p = .346$.

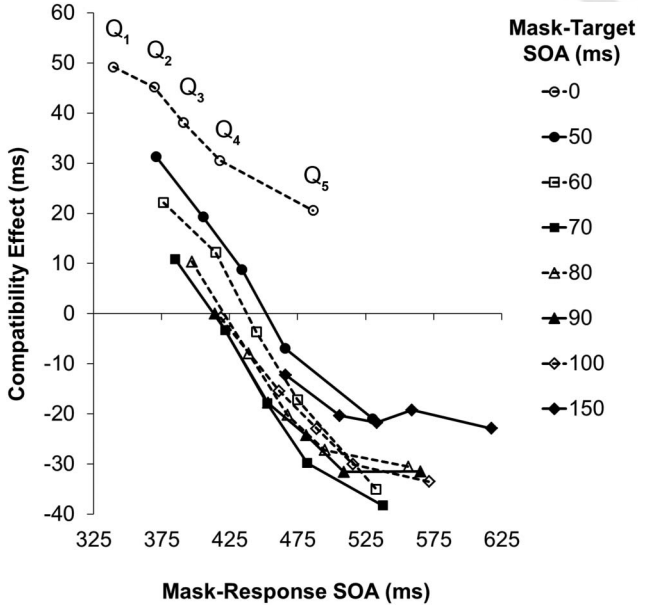


Figure 5. Results of Experiment 2 (part 2) compatibility effect (ms) as a function of mask-response SOA, computed by adding the mask-target SOA value to the mean reaction time (RT) value of each quintile. Q_n = Quintile n.

Because the mask-target SOA in each experiment did not modulate the interaction between first-order target sequence and compatibility, we grouped the data from the three experiments to obtain a more general perspective on the interaction between first-order sequence and compatibility (Figure 6). RTs were not significantly different between target repetitions and alternations, $F(1, 49) = 3.11$, $p = .084$, whereas the interaction between first-order target sequence and compatibility was highly significant, $F(1, 49) = 48.92$, $p < .001$. RTs were significantly faster for compatible target repetitions than for compatible target alternations, $t(49) = 3.31$, $p = .007$; difference = 9 ms, whereas RTs were not different between incompatible target repetitions and alternations, $t(49) = 0.19$, $p = .999$; difference = 0 ms. RTs were marginally faster in compatible than in incompatible trials for target repetitions, $t(49) = 2.43$, $p = .07$; PCE = 5 ms, whereas the CE was negative and not significant for target alternations, $t(49) = 1.78$, $p = .24$; NCE = −4.5 ms. F6

Therefore, the relation between the target on trial *n*-1 and prime and target on trial *n* appears to have an influence on the direction of the CE (PCE vs. NCE). The results suggests that complete repetitions between the previous target (e.g., LEFT) and the cur-

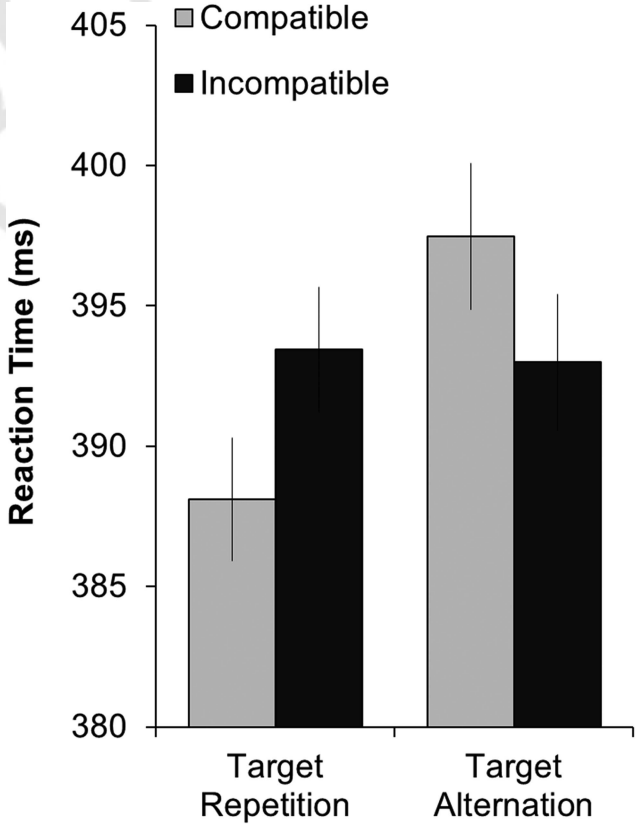


Figure 6. Results from all experiments. The compatibility effect is significantly modulated by the first-order sequence. "Target repetition" means that the current target *n* (e.g., a left arrow) is a repetition of the immediately preceding target *n-1* (e.g., a left arrow as well), and "target alternation" means that the current target *n* (e.g., a left arrow) is an alternation of the immediately preceding one *n-1* (e.g., a right arrow). Error bars represent 1 *SEM*.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

rent prime and target (e.g., left-LEFT) yielded the fastest responses, whereas complete alternation between the previous target (e.g., LEFT) and the current prime and target (e.g., right-RIGHT) led to the slowest responses. RTs were intermediate and equivalent for partial repetitions (e.g., [$LEFT_{trial\_n-1}$ with right-$LEFT_{trial\_n}$], [$LEFT_{trial\_n-1}$ with left-$RIGHT_{trial\_n}$]).

Higher-order target sequence. RT in the current target *n* was examined depending on the type of succession (alternation or repetition) of each couple of targets in a sequence of 4 targets (see Gao et al., 2009; Soetens et al., 1985). For instance, $Left_{trial\_n-3}$ $Left_{trial\_n-2}$ $Right_{trial\_n-1}$ $Left_{trial\_n}$ (LLRL) is described as a repetition (between $Left_{trial\_n-3}$ and $Left_{trial\_n-2}$) followed by an alternation (between $Left_{trial\_n-2}$ and $Right_{trial\_n-1}$) followed by another alternation (between $Right_{trial\_n-1}$ and $Left_{trial\_n}$), and thus consists in a $Repetition_{n-3\&n-2}$ $Alternation_{n-2\&n-1}$ $Alternation_{n-1\&n}$ sequence. In this RA(A) example, the A presented in brackets is the first-order sequence, whereas the RA outside the brackets is the higher-order sequence. The level 1 of the higher-order sequence variable is RR(R) or RR(A), the level 2 is AR(R) or AR(A), the level 3 is RA(R) or RA(A), and the level 4 is AA(R) or AA(A). Thus, higher-order repetitions decrease over the levels of the variable, whereas higher-order alternations increase. Soetens et al. (1985) showed that RTs increased with the increase of higher-order alternations when the current target was a repetition of the immediately preceding one, while the reverse pattern was observed when the current target was an alternation of the immediately preceding one. We expected to replicate this interaction between first-order and higher-order sequences on RTs, and to show that the CE will mirror these RT modulations because of target sequence (i.e., faster RTs because of target sequence will be associated with higher positive values of the CE, and vice versa). Thus, we were particularly interested in examining the relation between compatibility, first-order and higher-order target sequences. The role of the mask-target SOA in this relation was not of direct importance.

To increase the sensitivity of the results (by maximizing the number of trials and participants per subcondition) and to avoid possible bias in the results by the mask-target SOA variable, we separated data between groups of similar SOAs and assembled results from experiments containing these SOAs. In addition to the
AQ: 5 trial exclusion process we applied previously (see above), the three trials that followed incorrect responses, as well as RTs <100 or $>1{,}000$ were also excluded. The mean RTs were submitted to a repeated measures analysis of variance with first-order sequence (two levels), higher-order sequence (four levels), and compatibility (two levels) as within-subject factors.

The first analysis was restricted to the data from SOAs 60, 70, and 80 ms: These three mask-target SOAs have been unambiguFn3 ously considered as "intermediate" through all the experiments.[3] Both Experiments 1b and 2 shared these SOAs and were therefore grouped together (34 participants). The interaction between first-order and higher-order sequences was highly significant, $F(3, 99) = 57.87$, $p < .001$ (Figure 7A). For the first-order repetitions, RTs gradually increased with alternations in the higher-order sequence, linear contrast: $F(1, 33) = 47.09$, $p < .001$) In contrast, for the first-order alternations, RTs gradually decreased with alternations in the higher-order sequence, linear contrast: $F(1, 33) = 86.29$, $p < .001$. This replicates the exact results of Soetens et al. (1985). The 3-way interaction between first-order sequences, higher-order sequences, and compatibility was also significant, $F(3, 99) = 4.49$, $p = .005$ (Figure 7C). For the first-order repetitions, the CE gradually decreased (from positive to negative values) with alternations in the higher-order sequence, linear contrast: $F(1, 33) = 5.51$, $p = .025$. In contrast, for the first-order alternations, the CE gradually increased (from large negative to small negative values) with alternations in the higher-order sequence, linear contrast: $F(1, 33) = 10.46$, $p = .003$.

The second analysis was restricted to the data from SOAs 100 and 150 ms: These two SOAs have been considered to be "long" SOAs eliciting NCEs. This is reflected in the literature, as well as in the present experiments. Both Experiments 1a and 2 shared these SOAs and were thus assembled (35 participants). The interaction between first-order and higher-order sequences was highly significant, $F(3, 102) = 77.36$, $p < .001$ (Figure 7B). For the first-order repetitions, RTs gradually increased with alternations in the higher-order sequence, linear contrast: $F(1, 34) = 56.03$, $p < .001$. In contrast, for the first-order alternations, RTs gradually decreased with alternations in the higher-order sequence, linear contrast: $F(1, 34) = 53.95$, $p < .001$. Moreover, the 3-way interaction between first-order, higher-order sequences, and compatibility was significant, $F(3, 102) = 2.74$, $p = .047$ (Figure 7D). For the first-order repetitions, the CE did not significantly decrease with alternations in the higher-order sequence, linear contrast: $F(1, 34) = 1.42$, $p = .241$. Finally, for the first-order alternations, the CE gradually increased (from large negative to small negative values) with alternations in the higher-order sequence, linear contrast: $F(1, 34) = 5.85$, $p = .021$.

We did not perform an analysis for the short SOA condition, because only SOA 0 ms can be unambiguously considered as short (eliciting a strong overall PCE) through all the experiments. This SOA alone does not contain a sufficient number of trials (8 trials per subcondition) for reliable analysis.

To conclude, the CE modulations followed the RT modulations stemming from target sequences for intermediate SOAs: When RT increased with higher-order sequence, CE values were more negative, whereas when RT decreased with higher-order sequence, CE values were less negative. A similar pattern of results was found for long SOAs, but the effects were weaker.

Discussion

When we make deliberate actions in response to target stimuli, our response time varies considerably even though the perceived information is exactly the same. In three masked priming experiments, we demonstrated that these RT fluctuations in voluntary decision could modulate the magnitude and the direction of unconscious priming effects. During these experiments, participants had to make quick and accurate responses with a left- or right-hand key press to the orientation of a target arrow. Participants were kept unaware of the fact that the arrow to which they were responding (the target) was preceded by another arrow (the prime) presented very briefly and immediately before a mask. The prime and the target arrows could either point in the same direction (compatible trials) or in opposite directions (incompatible trials). Unlike previous studies examining the role of RT fluctuations on

[3] That is, SOAs eliciting a small overall CE and both PCE and NCE when the CE was examined in each speed bin.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

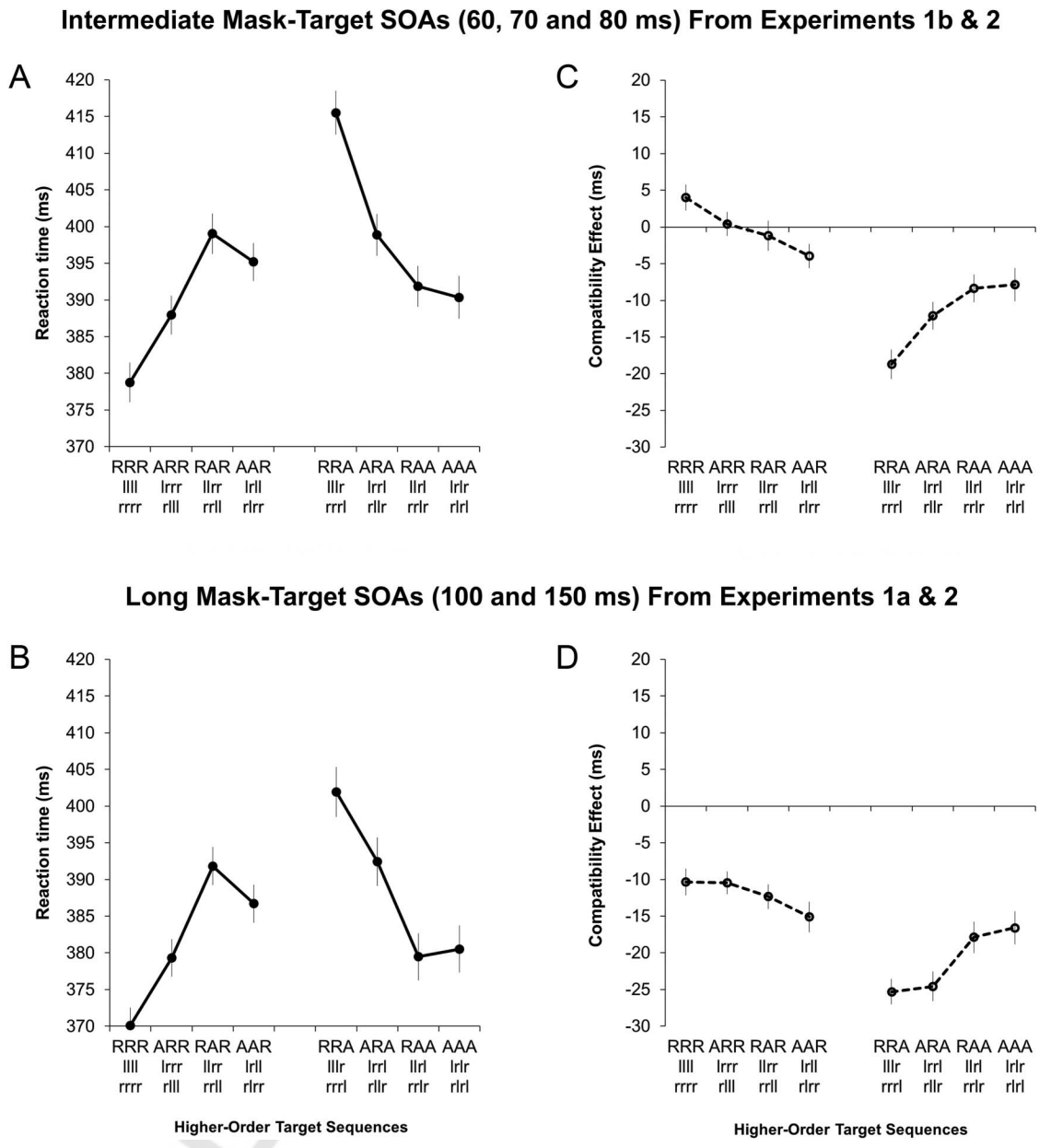


Figure 7. (A and B) Reaction time (RT) change as a function of the higher-order target sequence is the same for the intermediate mask-target SOAs (plot A) and for the long mask-target SOAs (plot B). Left side of each plot: When the current target *n* was a repetition of the immediately preceding one *n-1* (first-order target repetition, in bold in the sequences RR**R**, A**R**R, RA**R**, AA**R**), RTs increase with the increasing repetitions of higher-order alternations (i.e., the alternations underlined in the sequences RR**R**, A**R**R, RA**R**, AA**R**). Right side of each plot: When the current target *n* was an alternation of the immediately preceding one *n-1* (first-order target alternation, in bold in the sequences RR**A**, A**R**A, RA**A**, AA**A**), RTs decreased with the increase of higher-order alternations (the alternations underlined in the sequences RR**A**, A**R**A, RA**A**, AA**A**). (C) The CE evolution as function of the higher-order target sequence for intermediate mask-target SOAs. Left side of the plot: When the current target was a repetition of the immediately preceding one, the CE gradually decreased (from positive to negative values) with the increase of higher-order alternations. Right side of the plot: When the current target was an alternation of the immediately preceding one, the CE gradually increased (from large negative to small negative values) with the increase of higher-order alternations. (D) A similar pattern of results was observed for the long mask-target SOAs, but the effects were less significant. r = right target, l = left target; A = Alternation between two successive targets (e.g., $r_{n-1}l_n$; $l_{n-3}r_{n-2}$); R = repetition between two successive targets (e.g., $r_{n-1}r_n$; $l_{n-2}l_{n-1}$). For instance, *AA***R** (e.g., $r_{n-3}l_{n-2}r_{n-1}r_n$) is a higher-order alternation ($r_{n-3}l_{n-2}$) followed by another higher-order alternation ($l_{n-2}r_{n-1}$), followed by a first-order repetition ($r_{n-1}r_n$). Error bars represent 1 *SEM*.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

the negative CE (Eimer, 1999; Schlaghecken & Eimer, 2000; Verleger et al., 2004), we used masks composed by random lines to ensure that the NCE reflects automatic motor inhibition and not object-updating (Lleras & Enns, 2004).

Prior studies using random line masks have already shown that the mask-target SOA modulates automatic inhibition of the irrelevant prime activation (Aron et al., 2003; Boy, Evans, et al., 2010; Boy, Husain, & Sumner, 2010; Boy & Sumner, 2010; Eimer & Schlaghecken, 2003; Seiss & Praamstra, 2006). More specifically, these studies showed that short SOAs (0–40 ms) were associated with a PCE, while long SOAs (100–150 ms) were associated with an NCE. We replicated these results and showed that this SOA variable was not the only temporal factor contributing to this inhibition, but that RT fluctuations also play a determinant role. Indeed, the RT distributional analysis showed that PCEs occurred at shorter RTs to the target, reflecting the initial motor activation of the prime. In contrast, NCEs were observed at longer RTs to the target, reflecting the subsequent inhibition of this irrelevant motor activation. Thus, RT fluctuations in the voluntary decision modulated the strength of automatic motor inhibition.

Moreover, we demonstrated that RT influences on the CE was not the same for all mask-target SOA conditions. Indeed, when the mask-target SOA could not determine the prime's influence on the response to the target (i.e., null or small CE at intermediate SOAs), internal RT fluctuation had a stronger impact on the CE. In contrast, when the mask-target SOA strongly determines the influence of the prime on the response to the target (i.e., large PCEs at the short SOA or large NCEs at the long SOAs), internal RT fluctuations had a weaker impact on the CE. It is interesting that at the SOA of 150 ms, RT fluctuations did not significantly modulate the CE anymore. Our results are thus consistent with the recent study of Maylor, Birak, and Schlaghecken (2011), who found exactly the same results in young adults using random line masks and an SOA of 150 ms. Why is the impact of RT fluctuations on CE so different between the different levels of mask-target SOA? Four different reasons, not altogether incompatible with each other, might account for the observed interaction.

According to a first explanation, the dependency between the magnitude of motor activation (PCE) and the magnitude of motor inhibition (NCE) observed in previous studies (Boy et al., 2010; Boy & Sumner, 2010) might explain the fact that RT fluctuations had a weaker impact on the CE at long SOAs compared with intermediate SOAs. Indeed, once sufficient time is provided by the mask-target SOA to inhibit completely the prime motor influence (i.e., at SOAs of 100–150 ms), the longer RTs do not allow more inhibition because there is nothing else to inhibit. In contrast, for intermediate SOAs, the time provided by the mask-target SOA is insufficient to completely inhibit the prime's motor influence (i.e., at SOAs of 60–80 ms), allowing the slower RTs to complete the motor inhibitory process.

According to a second explanation, the different results for the long SOAs (especially, the SOA of 150 ms) compared with the intermediate SOAs can also be explained by the short-lived nature of the unconscious representation, which disappears after a few hundreds of milliseconds (Gaillard et al., 2009; Greenwald et al., 1996). Therefore, the NCE increase with response latency at the long SOAs might be smaller because of the more important decay associated with the unconscious neural representation at these long SOAs. That is to say, there is less accumulation of inhibition at the long SOAs because the neural representation is starting to disappear.

According to a third explanation, these results could also be explained by a possible reversal of the NCE across time (i.e., the reoccurrence of a PCE with extremely long delays). Indeed, Sumner and Brandwood (2008) found an NCE at an SOA of 150 ms that was followed by a PCE at SOAs of 400–600 ms, suggesting that once the motor inhibitory effect has accumulated sufficiently, it is again inhibited, resulting in a small PCE at longer delays. Thus, the weaker increase of the NCE with slow responses at longer SOAs compared with intermediate SOAs might be because of the initiation of the inhibition of the NCE at longer RTs of the longer SOAs (i.e., the reoccurrence of a PCE).

Finally, the fourth explanation is more related to the fact that the mask-target SOA is an external stimulus factor that strongly determines the sequence of prime-driven activation and target-driven activation, while RT fluctuations represent internal fluctuations in decision-making processes. It is possible that the level of this external stimulus factor constrains the weight of the influences of the internal fluctuations on the CE. This interpretation is based on the study of Bode et al. (2012), who showed that the level of stimulus discriminability (weak vs. strong) modulates the weight of the influence of internal fluctuations on the perceptual decision (for similar results, see Shadlen & Newsome, 2001). More specifically, they showed that when stimuli provide sufficient discriminative information (noise-masked images of chairs or pianos), decisions were better predicted by poststimulus neuronal activity; that is, by the stimulation itself. In contrast, when stimuli provide no discriminative information (masked noise images), decisions were better predicted by internal prestimulus neuronal activity, that is, by spontaneous fluctuations.[4] Our results might be consistent with these studies because we showed stronger influence of internal fluctuations (i.e., RTs) on the CE when the external stimulation was not able to influence it (i.e., at intermediate mask-target SOAs). Fn4

The present study also demonstrates what was suspected in many previous studies: A null CE at the intermediate mask-target SOAs is a transition point that reflects a type of effect that is as meaningful as the ones observed at the other mask-target SOAs (e.g., Bowman, Schlaghecken, & Eimer, 2006; Eimer & Schlaghecken, 2003; Lingnau & Vorberg, 2005). Indeed, no effect at the intermediate SOAs does not mean that no processing happens at all. Instead, it means that some prime processing truly happens, but that the priming effect is null because it is averaged between fast and slow RTs. This can be observed not only in the context of the present stimuli and procedure, but also in various masked priming tasks in which the mask-target SOA was not appropriately settled to observe a sizable overall CE. Indeed, a transition between positive CE and null/negative CE across response times could be either because of some object-updating processes, automatic motor inhibition, or a decay of the mental representation elicited by the prime. This suggests that the RT distributional analysis should be used to examine any masked priming effect; otherwise, important information is missed.

[4] Noteworthy, even if RT fluctuations might be considered as a poststimulus measure, these fluctuations are internal because there are isolated from the influence of the stimulus.

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Furthermore, we showed that target sequence—a systematic source of RT fluctuations coming from the external environment—modulated unconscious motor inhibition. Following the studies of Gao et al. (2009) and Soetens, Boer, and Hueting (1985), we examined the impact of two types of target sequences (1) the influence of the immediately preceding target on the RT/CE of the current trial (*first-order target sequence*), and (2) the influence of a sequence of three targets on the RT/CE of the current trial (*higher-order target sequence*).

With regard to first-order target sequence, the presence of a complete repetition or complete alternation between the previous target and the current prime and target seems to influence the direction of the CE (PCE vs. NCE). It is plausible that responding to the previous target increases the level of baseline motor activation in favor of this particular response during the intertribal interval. Therefore, when both the current prime and target request this response again (i.e., the compatible/target repetition condition in Figure 6), (a) executing the same response once more might be faster (response repetition effect), and (b) inhibiting subliminally triggered activation of that response might be more difficult (response-to-prime repetition effect). In contrast, when both the current prime and target request the opposite response (i.e., the compatible/target alternation condition in Figure 6), (a) executing this opposite response might be slower (response alternation effect), and (b) inhibiting subliminally triggered activation of that opposite response might be easier (response-to-prime alternation effect).

With regard to higher-order target sequence, we replicated the results of Soetens, Boer, and Hueting (1985). RTs increased with the increase in higher-order alternations when the immediately previous target was a repetition of the current target, and the reverse pattern was observed when the immediately previous target was an alternation of the current target. Crucially, we found that the CE modulations followed RT modulations because of target sequences at intermediate SOAs. Indeed, both RTs and the NCE increased with higher-order alternations when first-order sequences were repetitions. Similarly, both RTs and the NCE decreased with higher-order alternations when first-order Sequences were alternations. A similar pattern of results was found at long SOAs, but the effects were weaker. Thus, RT modulations because of higher-order target sequence influence the CE in the same direction: Longer RTs because of target sequences are associated with more NCE, which reflects more motor inhibition. It is important that this higher-order target sequence variable is known to influence strategic expectancies (Gao et al., 2009) and has nothing do to do with automatic motor inhibition. However, this seemingly irrelevant variable influences automatic inhibition, simply because it modulates RTs. It is plausible that automatic motor inhibition is potentially modulated by any factor that modulates RTs, and thus by many factors that have previously been considered as irrelevant, such as posterror slowing, fatigue, and so forth.

Noteworthy, the magnitude of the maximal RT difference because of target sequence was about 35 ms, whereas the magnitude of the RT difference between quintile 1 (very fast trials) and quintile 5 (very slow trials) was about 150–175 ms. Therefore, even though systematic sources of RT fluctuations might account for some modulations of the CE, internal spontaneous fluctuations of RTs might also play an important role in CE modulations. What are the causes of these fluctuations? One plausible source of RT fluctuations is spontaneous adjustments in the speed–accuracy trade-off (SAT) from one trial to the other (Bogacz et al., 2010). Indeed, in conflict tasks, both accuracy and speed are equally required, but both requirements are impossible to achieve perfectly in every trial. In this context, participants spontaneously fluctuated from more impulsive and faster decisions to more cautious and accurate decisions to the target from one trial to the other. It was recently found that the SAT modulates the distance between baseline activity and response threshold in the pre-SMA decision-masking center (Bogacz, Wagenmakers, Forstmann, & Nieuwenhuis, 2010; Forstmann, Dutilh, et al., 2008). Pre-SMA region is also involved in deliberate target-response translation in conflict tasks (Ridderinkhof, Forstmann, Wylie, Burle, & van den Wildenberg, 2011). As suggested by the dual route model, action selection can be driven either by the automatic irrelevant response or by deliberate intention (deliberate target-response translation; Ridderinkhof et al., 2011). Effortful deliberate action selection is more or less effective depending on evidence that had accumulated over time on a given trial. For more cautious and longer responses, the distance between baseline activity and response threshold is larger and evidence accumulate over a more extended period of time before crossing the response threshold. Responses are more accurate because evidence from the target accrued sufficiently: At this stage of processing, action selection is intention-guided (i.e., voluntary decision is effective). For more impulsive and faster response, baseline-to-threshold distance is shorter and evidence accumulates only over a very short period of time before reaching the threshold of overt response. At this early stage of processing, action selection is not perfectly intention-guided (voluntary decision is not fully effective). Thus, spontaneous adjustments in SAT toward a more careful response mode might enable a better selection of the relevant response in face of conflict. This more careful mode of decision to the target might also provide more time for automatic motor inhibitory mechanisms to be effective.

Previously, Sumner et al. (2007) have suggested that automatic inhibitory mechanisms contribute to voluntary decisional control, because they rapidly suppress partial activation of strongly established stimulus–response associations so that the most strongly established actions are not inevitably executed. In light of the present study, we suggest that the relationship between voluntary decision processes and automatic inhibitory processes might be more complex. Indeed, one plausible source of spontaneous RT fluctuations in conflict tasks is variations of the voluntary decisional process (from more impulsive/fast decisions to more cautious/slow decisions), which might affect unconscious automatic inhibition of the prime-induced response. Thus, not only automatic inhibitory processes seem to contribute to voluntary control (as suggested by Sumner et al., 2007), but voluntary decisional control might also contribute to automatic inhibitory processes.

Conclusion

To conclude, the present study shows that RT fluctuations modulated automatic inhibition of irrelevant actions. Moreover, these internal fluctuations of time and external changes of time (i.e., the different mask-target SOAs) interacted during motor inhibition. When external time had a strong impact on motor facilitation or inhibition, the impact of internal fluctuations on motor facilitation or inhibition seems to be weaker. Moreover, a

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

systematic source of RT fluctuations—RT fluctuations because of target sequences—modulate automatic motor inhibition. However, the whole RT fluctuations were larger than the one because of target sequence, suggesting that internal spontaneous fluctuations of RT might also play an important role in CE modulations. In this task in which both accuracy and speed are equally required but both requirements are impossible to achieve in every trial, it is likely that spontaneous RT fluctuations are because of trial-to-trial changes from more cautious and accurate voluntary decisions to more impulsive and faster voluntary decisions. This suggests that fluctuations in the level of caution in voluntary decisions modulate motor inhibition of irrelevant actions, though the latter process is involuntary, automatic, and unconscious.

References

Aron, A. R., & Poldrack, R. A. (2006). Cortical and subcortical contributions to Stop signal response inhibition: Role of the subthalamic nucleus. *The Journal of Neuroscience, 26,* 2424–2433. http://dx.doi.org/10.1523/JNEUROSCI.4682-05.2006

Aron, A. R., Schlaghecken, F., Fletcher, P. C., Bullmore, E. T., Eimer, M., Barker, R., . . . Robbins, T. W. (2003). Inhibition of subliminally primed responses is mediated by the caudate and thalamus: Evidence from functional MRI and Huntington's disease. *Brain: A Journal of Neurology, 126,* 713–723. http://dx.doi.org/10.1093/brain/awg067

Atas, A., San Anton, E., & Cleeremans, A. (2014). The reversal of perceptual and motor compatibility effects differs qualitatively between metacontrast and random-line masks. *Psychological Research,* ●●●, 1–16. AQ: 6

Bode, S., Sewell, D. K., Lilburn, S., Forte, J. D., Smith, P. L., & Stahl, J. (2012). Predicting perceptual decision biases from early brain activity. *The Journal of Neuroscience, 32,* 12488–12498. http://dx.doi.org/10.1523/JNEUROSCI.1708-12.2012

Bogacz, R., Wagenmakers, E. J., Forstmann, B. U., & Nieuwenhuis, S. (2010). The neural basis of the speed-accuracy tradeoff. *Trends in Neurosciences, 33,* 10–16. http://dx.doi.org/10.1016/j.tins.2009.09.002

Bowman, H., Schlaghecken, F., & Eimer, M. (2006). A neural network model of inhibitory processes in subliminal priming. *Visual Cognition, 13,* 401–480. http://dx.doi.org/10.1080/13506280444000823

Boy, F., Evans, C. J., Edden, R. A., Singh, K. D., Husain, M., & Sumner, P. (2010). Individual differences in subconscious motor control predicted by GABA concentration in SMA. *Current Biology, 20,* 1779–1785. http://dx.doi.org/10.1016/j.cub.2010.09.003

Boy, F., Husain, M., & Sumner, P. (2010). Unconscious inhibition separates two forms of cognitive control. *Proceedings of the National Academy of Sciences of the United States of America, 107,* 11134–11139. http://dx.doi.org/10.1073/pnas.1001925107

Boy, F., & Sumner, P. (2010). Tight coupling between positive and reversed priming in the masked prime paradigm. *Journal of Experimental Psychology: Human Perception and Performance, 36,* 892–905. http://dx.doi.org/10.1037/a0017173

Chevrier, A. D., Noseworthy, M. D., & Schachar, R. (2007). Dissociation of response inhibition and performance monitoring in the stop signal task using event-related fMRI. *Human Brain Mapping, 28,* 1347–1358. http://dx.doi.org/10.1002/hbm.20355

Craft, J. L., & Simon, J. R. (1970). Processing symbolic information from a visual display: Interference from an irrelevant directional cue. *Journal of Experimental Psychology, 83,* 415–420. http://dx.doi.org/10.1037/h0028843

De Jong, R., Liang, C. C., & Lauber, E. (1994). Conditional and unconditional automaticity: A dual-process model of effects of spatial stimulus-response correspondence. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 731–750. http://dx.doi.org/10.1037/0096-1523.20.4.731

Eimer, M. (1999). Facilitatory and inhibitory effects of masked prime stimuli on motor activation and behavioural performance. *Acta Psychologica, 101,* 293–313. http://dx.doi.org/10.1016/S0001-6918(99)00009-8

Eimer, M., & Schlaghecken, F. (1998). Effects of masked stimuli on motor activation: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance, 24,* 1737–1747. http://dx.doi.org/10.1037/0096-1523.24.6.1737

Eimer, M., & Schlaghecken, F. (2002). Links between conscious awareness and response inhibition: Evidence from masked priming. *Psychonomic Bulletin & Review, 9,* 514–520. http://dx.doi.org/10.3758/BF03196307

Eimer, M., & Schlaghecken, F. (2003). Response facilitation and inhibition in subliminal priming. *Biological Psychology, 64,* 7–26. http://dx.doi.org/10.1016/S0301-0511(03)00100-5

Forstmann, B. U., Dutilh, G., Brown, S., Neumann, J., von Cramon, D. Y., Ridderinkhof, K. R., & Wagenmakers, E. J. (2008). Striatum and pre-SMA facilitate decision-making under time pressure. *Proceedings of the National Academy of Sciences of the United States of America, 105,* 17538–17542. http://dx.doi.org/10.1073/pnas.0805903105

Forstmann, B. U., van den Wildenberg, W. P., & Ridderinkhof, K. R. (2008). Neural mechanisms, temporal dynamics, and individual differences in interference control. *Journal of Cognitive Neuroscience, 20,* 1854–1865. http://dx.doi.org/10.1162/jocn.2008.20122

Gaillard, R., Dehaene, S., Adam, C., Clémenceau, S., Hasboun, D., Baulac, M., . . . Naccache, L. (2009). Converging intracranial markers of conscious access. *PLoS Biology, 7,* e61. http://dx.doi.org/10.1371/journal.pbio.1000061

Gao, J., Wong-Lin, K., Holmes, P., Simen, P., & Cohen, J. D. (2009). Sequential effects in two-choice reaction time tasks: Decomposition and synthesis of mechanisms. *Neural Computation, 21,* 2407–2436. http://dx.doi.org/10.1162/neco.2009.09-08-866

Gibson, J. J. (1979). *The ecological approach to visual perception.* ●●●. AQ: 7

Greenwald, A. G., Draine, S. C., & Abrams, R. L. (1996). Three cognitive markers of unconscious semantic activation. *Science, 273,* 1699–1702. http://dx.doi.org/10.1126/science.273.5282.1699

Grèze, J., & Decety, J. (2002). Does visual perception of object afford action? Evidence from a neuroimaging study. *Neuropsychologia, 40,* 212–222. http://dx.doi.org/10.1016/S0028-3932(01)00089-6

Hommel, B. (1997). Interactions between stimulus-stimulus congruence and stimulus-response compatibility. *Psychological Research, 59,* 248–260. http://dx.doi.org/10.1007/BF00439302

Jaśkowski, P. (2008). The negative compatibility effect with nonmasking flankers: A case for mask-triggered inhibition hypothesis. *Consciousness and Cognition, 17,* 765–777. http://dx.doi.org/10.1016/j.concog.2007.12.002

Jaśkowski, P., Białuńska, A., Tomanek, M., & Verleger, R. (2008). Mask- and distractor-triggered inhibitory processes in the priming of motor responses: An EEG study. *Psychophysiology, 45,* 70–85.

Jaśkowski, P., & Przekoracka-Krawczyk, A. (2005). On the role of mask structure in subliminal priming. *Acta Neurobiologiae Experimentalis, 65,* 409–417.

Jaśkowski, P., & Verleger, R. (2007). What determines the direction of subliminal priming. *Advances in Cognitive Psychology, 3,* 181–192. http://dx.doi.org/10.2478/v10053-008-0024-1

Klapp, S. T. (2005). Two versions of the negative compatibility effect: Comment on Lleras and Enns (2004). *Journal of Experimental Psychology: General, 134,* 431–435. http://dx.doi.org/10.1037/0096-3445.134.3.431

Konishi, S., Nakajima, K., Uchida, I., Kikyo, H., Kameyama, M., & Miyashita, Y. (1999). Common inhibitory mechanism in human inferior prefrontal cortex revealed by event-related functional MRI. *Brain: A*

tapraid5/zfn-xhp/zfn-xhp/zfn00115/zfn3185d15z | xppws | S=1 | 12/22/14 | 19:01 | Art: 2013-1484

Journal of Neurology, 122, 981–991. http://dx.doi.org/10.1093/brain/122.5.981

Leung, H. C., & Cai, W. (2007). Common and differential ventrolateral prefrontal activity during inhibition of hand and eye movements. *The Journal of Neuroscience, 27,* 9893–9900. http://dx.doi.org/10.1523/JNEUROSCI.2837-07.2007

Li, C. S., Huang, C., Constable, R. T., & Sinha, R. (2006). Imaging response inhibition in a stop-signal task: Neural correlates independent of signal monitoring and post-response processing. *The Journal of Neuroscience, 26,* 186–192. http://dx.doi.org/10.1523/JNEUROSCI.3741-05.2006

AQ: 8

Lingnau, A., & Vorberg, D. (2005). The time course of response inhibition in masked priming. *Perception & Psychophysics, 67,* 545–557. http://dx.doi.org/10.3758/BF03193330

Lleras, A., & Enns, J. T. (2004). Negative compatibility or object updating? A cautionary tale of mask-dependent priming. *Journal of Experimental Psychology: General, 133,* 475–493. http://dx.doi.org/10.1037/0096-3445.133.4.475

Lleras, A., & Enns, J. T. (2006). How much like a target can a mask be? Geometric, spatial, and temporal similarity in priming: A reply to Schlaghecken and Eimer (2006). *Journal of Experimental Psychology: General, 135,* 495–500. http://dx.doi.org/10.1037/0096-3445.135.3.495

Mattler, U. (2006). On the locus of priming and inverse priming effects. *Perception & Psychophysics, 68,* 975–991. http://dx.doi.org/10.3758/BF03193359

Maylor, E. A., Birak, K. S., & Schlaghecken, F. (2011). Inhibitory motor control in old age: Evidence for de-automatization? *Frontiers in Psychology, 2,* 132. http://dx.doi.org/10.3389/fpsyg.2011.00132

McBride, J., Boy, F., Husain, M., & Sumner, P. (2012). Automatic motor activation in the executive control of action. *Frontiers in Human Neuroscience, 6,* 82. http://dx.doi.org/10.3389/fnhum.2012.00082

Miller, E. K. (2000). The prefrontal cortex and cognitive control. *Nature Reviews Neuroscience, 1,* 59–65. http://dx.doi.org/10.1038/35036228

Praamstra, P., & Seiss, E. (2005). The neurophysiology of response competition: Motor cortex activation and inhibition following subliminal response priming. *Journal of Cognitive Neuroscience, 17,* 483–493. http://dx.doi.org/10.1162/0898929053279513

Ridderinkhof, K. R. (2002a). Activation and suppression in conflict tasks: Empirical clarification through distributional analyses. In W. Prinz & B. Hommel (Eds.), *Common mechanisms in perception and action: Attention & performance* (Vol. XIX, pp. 494–519). New York, NY: Oxford University Press.

Ridderinkhof, K. R. (2002b). Micro- and macro-adjustments of task set: Activation and suppression in conflict tasks. *Psychological Research, 66,* 312–323. http://dx.doi.org/10.1007/s00426-002-0104-7

Ridderinkhof, K. R., Forstmann, B. U., Wylie, S. A., Burle, B., & van den Wildenberg, W. P. M. (2011). Neurocognitive mechanisms of action control: Resisting the call of the sirens. *Wiley Interdisciplinary Reviews: Cognitive Science, 2,* 174–192. http://dx.doi.org/10.1002/wcs.99

Ridderinkhof, K. R., van den Wildenberg, W. P. M., Wijnen, J., Burle, B, & Posner, M. (Eds.). (2004). Response inhibition in conflict tasks is revealed in delta plots. Cognitive neuroscience of attention (pp. 369–377). New York, NY: Guilford Press.

AQ: 9

Schlaghecken, F., Bowman, H., & Eimer, M. (2006). Dissociating local and global levels of perceptuo-motor control in masked priming. *Journal of Experimental Psychology: Human Perception and Performance, 32,* 618–632. http://dx.doi.org/10.1037/0096-1523.32.3.618

Schlaghecken, F., & Eimer, M. (2000). A central-peripheral asymmetry in masked priming. *Perception & Psychophysics, 62,* 1367–1382. http://dx.doi.org/10.3758/BF03212139

Schlaghecken, F., & Eimer, M. (2002). Motor activation with and without inhibition: Evidence for a threshold mechanism in motor control. *Perception & Psychophysics, 64,* 148–162. http://dx.doi.org/10.3758/BF03194564

Schlaghecken, F., & Eimer, M. (2004). Masked prime stimuli can bias "free" choices between response alternatives. *Psychonomic Bulletin & Review, 11,* 463–468. http://dx.doi.org/10.3758/BF03196596

Schlaghecken, F., & Eimer, M. (2006). Active masks and active inhibition: A comment on Lleras and Enns (2004) and on Verleger, Jaskowski, Aydemir, van der Lubbe, and Groen (2004). *Journal of Experimental Psychology: General, 135,* 484–494. http://dx.doi.org/10.1037/0096-3445.135.3.484

Seiss, E., & Praamstra, P. (2004). The basal ganglia and inhibitory mechanisms in response selection: Evidence from subliminal priming of motor responses in Parkinson's disease. *Brain: A Journal of Neurology, 127,* 330–339. http://dx.doi.org/10.1093/brain/awh043

Seiss, E., & Praamstra, P. (2006). Time-course of masked response priming and inhibition in Parkinson's disease. *Neuropsychologia, 44,* 869–875. http://dx.doi.org/10.1016/j.neuropsychologia.2005.09.001

Shadlen, M. N., & Newsome, W. T. (2001). Neural basis of a perceptual decision in the parietal cortex (area LIP) of the rhesus monkey. *Journal of Neurophysiology, 86,* 1916–1936.

Soetens, E., Boer, L., & Hueting, J. (1985). Expectancy or automatic facilitation? Separating sequential effects in two-choice reaction time. *Journal of Experimental Psychology: Human Perception and Performance, 10,* 581–598. http://dx.doi.org/10.1037/0096-1523.10.4.581

Sumner, P. (2008). Mask-induced priming and the negative compatibility effect. *Experimental Psychology (formerly "Zeitschrift für Experimentelle Psychologie"), 55,* 133–141.

Sumner, P., & Brandwood, T. (2008). Oscillations in motor priming: Positive rebound follows the inhibitory phase in the masked prime paradigm. *Journal of Motor Behavior, 40,* 484–490. http://dx.doi.org/10.3200/JMBR.40.6.484-490

Sumner, P., Nachev, P., Morris, P., Peters, A. M., Jackson, S. R., Kennard, C., & Husain, M. (2007). Human medial frontal cortex mediates unconscious inhibition of voluntary action. *Neuron, 54,* 697–711. http://dx.doi.org/10.1016/j.neuron.2007.05.016

Tucker, M., & Ellis, R. (1998). On the relations between seen objects and components of potential actions. *Journal of Experimental Psychology: Human Perception and Performance, 24,* 830–846. http://dx.doi.org/10.1037/0096-1523.24.3.830

Valle-Inclán, F., & Redondo, M. (1998). On the automaticity of ipsilateral response activation in the Simon effect. *Psychophysiology, 35,* 366–371. http://dx.doi.org/10.1111/1469-8986.3540366

van Gaal, S., Ridderinkhof, K. R., Scholte, H. S., & Lamme, V. A. (2010). Unconscious activation of the prefrontal no-go network. *The Journal of Neuroscience, 30,* 4143–4150. http://dx.doi.org/10.1523/JNEUROSCI.2992-09.2010

Verleger, R., Jaśkowski, P., Aydemir, A., van der Lubbe, R. H. J., & Groen, M. (2004). Qualitative differences between conscious and nonconscious processing? On inverse priming induced by masked arrows. *Journal of Experimental Psychology: General, 133,* 494–515. http://dx.doi.org/10.1037/0096-3445.133.4.494

Wascher, E., Schatz, U., Kuder, T., & Verleger, R. (2001). Validity and boundary conditions of automatic response activation in the Simon task. *Journal of Experimental Psychology: Human Perception and Performance, 27,* 731–751. http://dx.doi.org/10.1037/0096-1523.27.3.731

Received October 16, 2013
Revision received October 15, 2014
Accepted November 6, 2014 ■