

Withholding a Reward-driven Action: Studies of the Rise and Fall of Motor Activation and the Effect of Cognitive Depletion

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Abstract

■ Controlling an inappropriate response tendency in the face of a reward-predicting stimulus likely depends on the strength of the reward-driven activation, the strength of a putative top-down control process, and their relative timing. We developed a rewarded go/no-go paradigm to investigate such dynamics. Participants made rapid responses (on go trials) to high versus low reward-predicting stimuli and sometimes had to withhold responding (on no-go trials) in the face of the same stimuli. Behaviorally, for high versus low reward stimuli, responses were faster on go trials, and there were more errors of commission on no-go trials. We used single-pulse TMS to map out the corticospinal excitability dynamics, especially on no-go trials where control is needed. For successful no-go trials, there was an early rise in motor activation that was then sharply reduced beneath

baseline. This activation–reduction pattern was more pronounced for high- versus low-reward trials and in individuals with greater motivational drive for reward. A follow-on experiment showed that, when participants were fatigued by an effortful task, they made more errors on no-go trials for high versus low reward stimuli. Together, these studies show that, when a response is inappropriate, reward-predicting stimuli induce early motor activation, followed by a top–down effortful control process (which we interpret as response suppression) that depends on the strength of the preceding activation. Our findings provide novel information about the activation–suppression dynamics during control over reward-driven actions, and they illustrate how fatigue or depletion leads to control failures in the face of reward. ■

INTRODUCTION

One way that inappropriate action tendencies are controlled is via response suppression. In the laboratory, action tendencies are typically induced by creating or capitalizing on a strong relationship between a stimulus and a particular response (e.g., an arrow pointing right signals a right-hand response in the stop-signal task; Logan, 1994). Although these tasks have yielded insights into how inappropriate action tendencies are controlled, including neural circuits, motor dynamics, and factors that influence control, their relevance to daily life is limited (for reviews on response suppression, see Bari & Robbins, 2013; Ridderinkhof, Forstmann, Wylie, Burle, & van den Wildenberg, 2011; Stinear, Coxon, & Byblow, 2009; Aron, 2007). This is because, unlike the action provocations in these “cold” cognitive psychology tasks, real-world provocations are often driven by the reward-predicting properties of a stimulus (e.g., a tasty food).

In an effort to extend response suppression research to more real-world situations, we recently developed a behavioral paradigm that requires control in the face of motivationally driven provocations (Freeman, Alvernaz, Tonnesen, Linderman, & Aron, 2015; Freeman, Razhas, & Aron, 2014). In this paradigm, participants were either

permitted to respond for a small juice reward (go trials) or not permitted to respond (no-go trials), both in the face of a task-irrelevant stimulus that was earlier associated with juice via Pavlovian conditioning. This led participants, on go trials, to respond more quickly and, on no-go trials, to make more errors of inappropriate responding. For the same task, we used single-pulse TMS (spTMS) over primary motor cortex to measure motor system activity. We showed that, on go trials, the stimulus associated with juice (relative to a stimulus that was not associated with juice) increased motor excitability at 250 msec, whereas on successful no-go trials, there was a beneath-baseline reduction at the same time point. We interpreted this reduction as evidence for a response suppression process that helped mitigate the motivationally triggered activation, yielding an activation–suppression dynamic. However, because motor excitability was only measured at a single time point, those studies did not reveal the finer-grained dynamics of the predicted motor activation and motor reduction processes—how fast the activation appears, how high it reaches, when the control kicks in, and how long it lasts. Moreover, without a picture of the dynamics, those studies could not firmly show that the motor reduction was because of a control process that relates to the strength of the preceding activation. In those studies, TMS was limited to a single time point because of the waning influence of the Pavlovian stimulus

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from satiation over time (thus limiting trial numbers). Here, we sought to capture the putative activation–suppression dynamics using a new paradigm.

Now, rather than using Pavlovian-conditioned stimuli associated with juice, we used stimuli that predicted potential monetary rewards on a given trial. On go trials, participants made instrumental responses to obtain a reward in points (later converted to money). The number of potential points on a given trial was indicated by a colored rectangle (one color: high reward, the other: low reward) that was placed behind the go/no-go cue. On no-go trials, participants were required to withhold their pressing, despite the potential provocation induced by the reward stimulus. Because the reward stimulus was now task relevant and because it entailed monetary reward, there were no restrictions on trial numbers. Accordingly, TMS pulses were delivered at 100, 150, 200, and 250 msec after stimulus onset on different trials. We tested the hypothesis that the reward-predicting stimuli would evoke an early rise in motor excitability on both go and no-go trials and that this reward-driven activation would be immediately followed by a sharp reduction in motor excitability on no-go trials. We also hypothesized that, on no-go trials, the high reward stimulus would show a steeper early activation, and we were interested to examine if this sharper rise would be accompanied by a steeper subsequent reduction—which could reflect a more effortful control process that helps mitigate the increased activation. An alternative possibility is that a similar amount of control would be exerted for high- and low-reward trials, which predicts parallel reduction slopes after a greater initial activation for high-reward trials. In a second study, we tested the idea that the reduction phase reflects top–down control over the activation. We did this by first engaging the participants in an effortful task, which should “deplete” top–down resources; we then examined their ability to withhold responding on high- versus low-reward no-go trials.

EXPERIMENT 1

Methods

Participants

There were 30 participants (16 women; mean age = 20.73 years, $SD = 2.7$ years; all right-handed). Two were excluded for having oversaturated motor evoked potentials (MEPs; i.e., $MEPs > 2$ mV), and two were excluded because of technical malfunctions with the TMS equipment. Thus, all analyses were run on 26 participants, who provided informed consent and passed TMS safety screening.

Task and Procedure

Each participant sat in front of an iMac (Apple, Inc., Cupertino, CA) with a 20-in. monitor (60-Hz refresh rate). On each trial, participants saw either a black triangle or

a black square in the center of the screen for 1.75 sec (Figure 1A). Participants were instructed to respond to one of the shapes (go cue) and to withhold responding to the other shape (no-go cue). Go and no-go cues were equiprobable (i.e., 50/50), and the shapes were counterbalanced across participants.

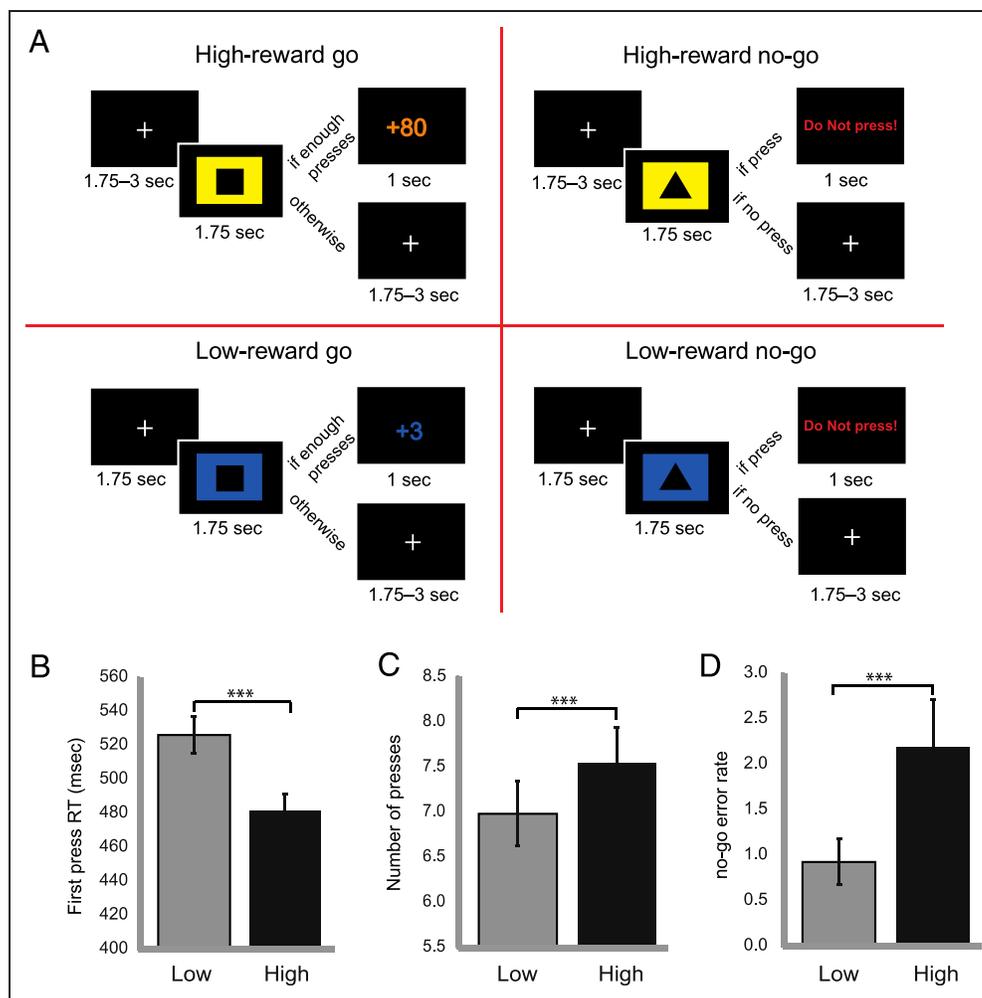
Upon presentation of the go cue, participants could continuously press a button with their right index finger to obtain points, which they were told would translate into money at the end of the experiment. Presses were to be made only during the 1.75-sec duration of the go trial, and participants were instructed to stop pressing once the go cue disappeared. Points were delivered on a variable ratio reward schedule. For the first block (designated as the “learning block”), the required number of presses ranged from four to nine presses based on a uniform distribution. After the learning block, the range was adjusted based on the participant’s mean number of presses (rounded to the nearest integer) during the learning block. To maximize motivational drive for reward, the range was adjusted such that the participant could obtain a reward on most (i.e., $>50\%$) but not all go trials. Thus, a mean press rate of 5 yielded a range of 2–7, a mean press rate of 6 yielded a range of 3–8, and so on. The average proportion of rewarded go trials was 0.64 ($SD = 0.13$) across participants. Information regarding the number of presses required for reward was not disclosed to the participants, although they were informed that the required number of presses would vary across trials. If the button was pressed enough times on a given trial, the amount of points earned was displayed at the center of the screen (e.g., “+50”; Figure 1A). If the button was not pressed enough times, a fixation cross appeared, and the intertrial interval (ITI) period began.

The number of possible points to be earned on a given trial was indicated by a large, colored (blue or yellow) rectangle that surrounded the go cue and was presented simultaneously. If enough presses were made on a high-reward go trial, participants received between 50 and 100 points (in increments of 10, chosen randomly). For low-reward go trials, participants received between 1 and 5 points (in increments of 1, chosen randomly). Participants were informed before the experiment that approximately 1000 points yielded \$1. High- and low-reward colors were counterbalanced across participants.

Upon presentation of the no-go cue, participants were required to withhold responding. If a press was mistakenly made on a no-go trial, a red error message reading, “Do Not Press the Button!”, was flashed for 1 sec. The no-go cue was also surrounded by a blue or yellow rectangle (which signals reward on go trials), thereby manipulating participants’ motivational drive even while they were required to withhold a response (Figure 1A).

All trials were separated by a fixation cross for a variable ITI of 1.75–3 sec (in increments of 0.25 sec, chosen randomly). Go and no-go cues were presented pseudo-randomly such that no more than four go or no-go cues

Figure 1. Rewarded go/no-go task and behavior. On go trials (left), continuous presses were made in effort to receive points (later converted to money). If enough presses were made on a given trial, the earned points were displayed to the participant; otherwise, no points were displayed, and the next trial began. The amount of potential points to be earned on a given trial was indicated by the background color. One color (shown as yellow here) was associated with substantially higher point rewards than the other color (shown as blue here). On no-go trials (right), responding was to be withheld; else, a red error message appeared. Go and no-go trials were equiprobable. (B) On go trials, the first press RT was significantly faster for high- compared with low-reward trials. (C) On go trials, more presses were made for high- compared with low-reward trials. (D) On no-go trials, the error rate was significantly greater for high- compared with low-reward trials. This indicates that the high-reward background stimulus provoked responding on both go and no-go trials. Error bars represent *SEM* across participants. *** $p < .001$.



could occur in succession. There were 14 total blocks with 52 trials in each block, yielding 728 total trials. At the end of each block, the number of cumulative points the participant had earned appeared at the top of the screen. At the end of the experiment, the total number of points earned was divided by 1000 and then converted to a rounded dollar amount. The mode for the total money earned across participants was \$9. After the experiment concluded, all participants completed the Barratt Impulsivity Scale (BIS-11) questionnaire (Patton, 1995).

TMS

TMS was delivered using a MagStim 200-2 system (MagStim, Whitland, UK) and a 70-mm figure-of-eight coil. Surface EMG was recorded from the first dorsal interosseous muscle of the right hand (corresponding to the task-relevant index finger) via 10-mm-diameter Ag–AgCl hydrogel electrodes (Medical Supplies, Inc., Newbury Park, CA).

The coil was placed 5 cm lateral and 2 cm anterior to the vertex and repositioned while delivering a TMS

stimulus to locate the position where the largest MEPs were observed consistently. The angle of the coil was approximately 45° from the central sulcus. We measured resting motor threshold, defined as the minimum stimulation intensity required to induce 0.1-mV peak-to-peak amplitude MEP in 5 of 10 consecutive stimulations (Rossini et al., 1994). Next, starting at resting motor threshold, the maximum MEP size was determined by increasing stimulus intensity in 3%–4% increments until the MEP amplitude no longer increased. Finally, the TMS stimulus intensity was adjusted to produce a MEP that was approximately half of the maximum MEP amplitude while the participant was performing the task in a practice session. This ensured that the test stimulus intensity was on the ascending limb of the individual's stimulus–response curve, so that both increases and decreases in corticospinal excitability could be detected (Devanne, Lavoie, & Capaday, 1997). This was the intensity used during the experiment proper (mean intensity across participants was 44.7% stimulator output, $SD = 8.16\%$). To measure the dynamics of corticospinal excitability across time, there were four pulse times after stimulus onset (100, 150, 200, and 250 msec),

yielding 42 trials per condition at each time point. There was also one pulse time 500 msec before stimulus onset to provide a baseline measure (56 trials). To optimize EMG over the first dorsal interosseous muscle, the right index finger moved inward to press a vertical key.

Behavioral Analysis

We compared high- and low-reward trials using three dependent measures: (1) the median RT to the first press on go trials (henceforth called first press RT), (2) the mean number of presses during the 1.75-sec response interval, and (3) the percentage of commission errors on no-go trials. As the first block was considered a “learning block” (where participants learned the color–reward associations), these trials were excluded from all analyses. Trials were also excluded if first press RT was less than 100 msec or if a response was not made on a go trial. Differences between high- and low-reward conditions were evaluated using two-tailed, paired *t* tests.

EMG Analysis

Preprocessing and normalization. An EMG sweep started 200 msec before stimulation. MEPs were identified from the EMG using in-house software developed in MATLAB (The MathWorks, Natick, MA). Trials were excluded if the root mean square EMG in the 100 msec before the TMS pulse was greater than 0.01 mV or if the MEP was less than 0.05 mV. We also excluded trials if the amplitude maxed out at +1 or –1 mV, because we used a CED MICRO 1401 system that has a cutoff at 2 mV (range of +1 to –1 mV). Thus, we could not be sure of the true MEP amplitude when it exceeded 2 mV (e.g., 2.1 and 4 mV). For this reason, we elected to exclude such MEPs that “maxed out,” as we feel that this provides the most accurate version of the MEP data set. Median peak-to-peak amplitudes of MEPs were calculated for all conditions at each time point. Then, the median MEP for each condition was divided (i.e., normalized) by the median MEP of the baseline trials (i.e., the time point at 500 msec before stimulus onset). An examination of the normalized root mean square values for the 100-msec time window before the TMS pulse showed no significant main effects or interactions (all *ps* > .05), demonstrating that the MEP patterns described below were not contaminated by differences in the pre-TMS period.

Go and no-go dynamics. To provide a detailed picture of the dynamics, we conducted several analyses. First, we separately evaluated go and no-go trials using ANOVAs with Reward (high, low) and Pulse time (100, 150, 200, 250 msec) as factors. For all analyses, we excluded no-go trials where a press was made (commission error). All go trials were analyzed, regardless of whether enough presses were made to earn points on the trial. Planned comparisons for high versus low reward were made at each

of the four time points using paired *t* tests with an alpha value set at .05. Because of the strong prediction of larger MEPs for high versus low reward on go trials, one-tailed *t* tests were used for this analysis, whereas two-tailed *t* tests were used for the no-go analysis (because the timing could not be predicted a priori). Unless otherwise specified, all reported *p* values were corrected for four comparisons using the Holm–Bonferroni procedure.

Percent change across time points on no-go trials. To better capture the change across time for no-go trials, we calculated the percent change of the “activation phase” (i.e., where MEPs were predicted to increase across time, reflecting response prepotency) and the “reduction phase” (i.e., where MEPs were predicted to decrease across time, likely reflecting response suppression). We entered the percent change values into a repeated-measures ANOVA with Reward (high, low) and Phase (activation, reduction) as factors. We then used two-tailed, one-sample *t* tests to examine differences between each condition and a value of zero (representing no change). Pairwise comparisons across conditions were then made using two-tailed, paired *t* tests. Unless otherwise specified, all reported *p* values in this analysis were corrected for eight comparisons using the Holm–Bonferroni procedure.

Relationship between reward-based activation and reward-based reduction on no-go trials. We were interested in examining the relationship between motor excitability during the (predicted) activation and reduction phases, particularly as a function of the reward value. Thus, in each participant, we calculated “reward-based” (high minus low reward) difference scores for each phase. Specifically, we subtracted the percent change for high reward from the percent change for low reward in the activation and reduction phases. A Pearson’s correlation was then used to test the relationship between participants’ reward-based activation and their reward-based reduction.

Relationship between no-go dynamics and error rates. We postulated that the motor dynamics on no-go trials would relate to participants’ self-control failures. We therefore examined how the activation, the reduction, and the activation–reduction processes together related to participants’ overall error rates on no-go trials (including high- and low-reward trials). To quantify participants’ activation and reduction levels, we computed an average score of percent change for the activation and reduction phases separately and correlated these measures with participants’ overall no-go error rates using Pearson’s correlations. We also correlated their no-go error rates with a composite measure of motor activity in both the activation and reduction phases—henceforth called the “activation–reduction index.” To calculate the activation–reduction index, we first summed the activation and reduction phases for high- and low-reward trials

separately. We then took the average of these two scores to generate an index that reflects both phases and reward values. In essence, this measure provides an index of the strength of the reduction process when taking into account the preceding activation. A Pearson's correlation was then used to test the relationship between participants' overall error rates and the activation–reduction index.

Go and no-go dynamics for fast and slow RT groups.

In addition to characterizing the overall dynamics, we reasoned that the motor dynamics in a reward task might depend on participants' basic motivational drive for reward. To examine this, we conducted a median split on the 26 participants based on their RTs, which we used as a behavioral index of motivational drive for reward (faster RT corresponds to higher motivation for reward; Avila & Lin, 2014; Clithero, Reeck, Carter, Smith, & Huettel, 2011). Specifically, we computed the average of the median high-reward RT and the median low-reward RT and took this average as the behavioral index of motivational drive for reward. We then conducted the same dynamics analyses as above for both fast and slow RT groups. Unless otherwise specified, all reported p values were corrected using the Holm–Bonferroni procedure.

Relationship between trait impulsivity and reward-based MEP differences. We acquired answers to a single questionnaire—the BIS-11—to explore a possible relationship between trait impulsivity and sensitivity to reward. We correlated participants' overall BIS-11 scores with their reward-based (high-minus-low difference score) RT, no-go errors, no-go activation phase (percent change from 100 to 150 msec), and the peak activation point on no-go trials (at 150 msec).

Results

Behavior

On go trials, first press RTs were significantly faster for high reward ($M = 480.8$ msec, $SD = 51.4$ msec) versus low reward ($M = 525.5$ msec, $SD = 55.4$ msec), $t(25) = 7.3$, $p < .001$ (Figure 1B), showing that the action was invigorated. Participants also made more presses on high-reward ($M = 7.55$, $SD = 2$) versus low-reward ($M = 6.99$, $SD = 1.8$) trials during the 1.75-sec interval, $t(25) = 6.2$, $p < .001$ (Figure 1C). On no-go trials, there was a higher percentage of commission errors for high reward ($M = 2.2\%$, $SD = 2.7\%$) versus low reward ($M = 0.93\%$, $SD = 1.3\%$), $t(25) = 3.6$, $p = .001$ (Figure 1D), suggesting that the action was also invigorated on no-go trials, which might make it more difficult to withhold. It is worth noting that, although there was a differential increase in no-go error rates for high- versus low-reward trials, no-go error rates for both trial types were low. Thus, our MEP analysis focused solely on successful no-go trials, as there were

insufficient trial numbers to analyze unsuccessful no-go trials.

MEPs

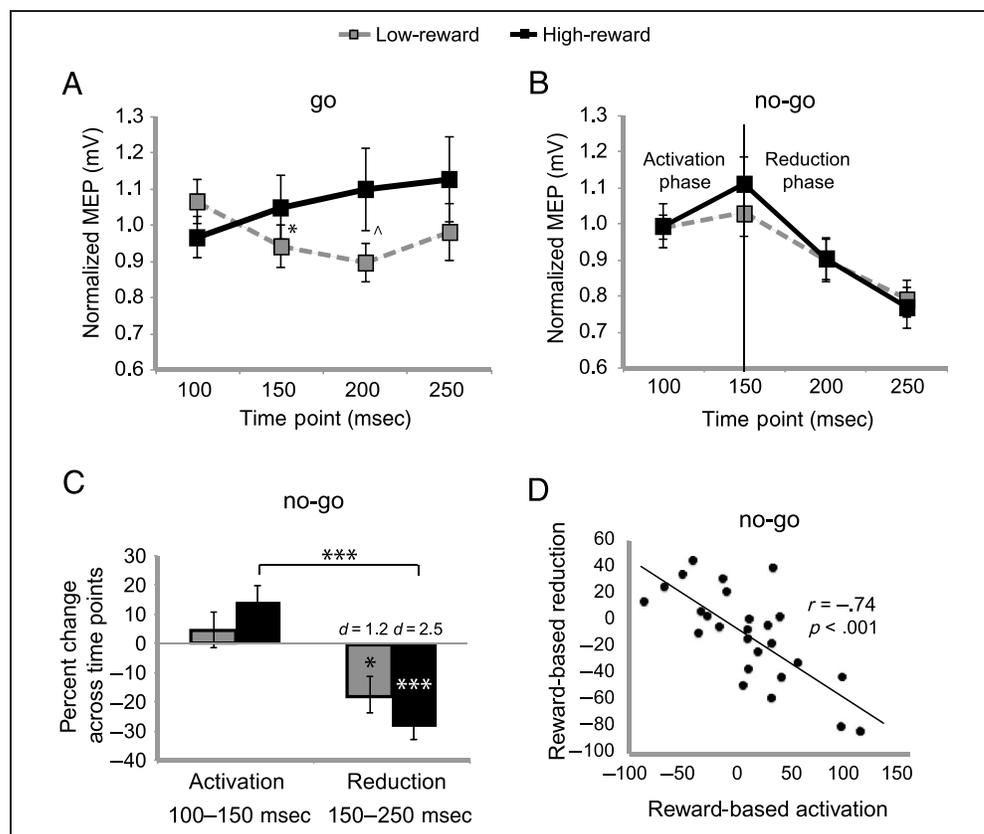
This study aimed to examine the dynamics at 100, 150, 200, and 250 msec after high and low reward stimuli on go and no-go trials separately. We were particularly interested in examining the putative activation–reduction dynamics on no-go trials and how this was different for high versus low reward stimuli.

Go and no-go dynamics. For go trials, there was a significant main effect of Reward ($F(1, 25) = 5.87$, $p = .023$) with MEPs for high reward greater than low reward. There was also a significant Reward \times Pulse time interaction ($F(3, 75) = 2.97$, $p = .037$). For high-reward go trials, there was a significant linear increase in motor excitability across the overall mean values in the four time points ($r_3 = 0.95$, $p = .026$); whereas, for low-reward go trials, motor excitability decreased from 100–200 msec, followed by an increase from 200–250 msec (Figure 2A). Follow-up t tests showed significantly elevated MEPs for the high reward stimulus at 150 msec ($t(25) = 2.41$, $p = .047$) and marginally elevated MEPs at 200 msec ($t(25) = 2.16$, $p = .06$; Figure 2A). This shows that very early motor activity is influenced by the value of a reward-predicting stimulus, which is consistent with several previous studies (Mooshagian, Keisler, Zimmermann, Schweickert, & Wassermann, 2015; Suzuki et al., 2014; Klein, Olivier, & Duque, 2012; Klein-Flügge & Bestmann, 2012).¹

For no-go trials, there was a main effect of Pulse time ($F(3, 75) = 16.09$, $p < .001$), where an initial increase in MEPs was followed by a sharp decrease (Figure 2B). The high-reward trials evidenced a greater early elevation in MEPs (at the 150-msec time point) compared with low-reward trials (1.11 vs. 1.03 mV), although the difference was not significant ($t(25) = 1.1$, ns ; Figure 2B). After the initial activation, there was a steep, beneath-baseline reduction in motor excitability for both high- and low-reward trials (250-msec time point vs. baseline: $p < .001$ for high and low reward). Thus, as predicted, no-go trials exhibited a pattern where an initial activation was followed by a steep reduction in motor excitability. We now explore the activation and reduction dynamics in more detail.

Percent change across time points on no-go trials. The activation phase on no-go trials evidently occurred from 100 to 150 msec after stimulus onset, whereas the reduction phase occurred from 150 to 250 msec (Figure 2B). To quantify the MEP change across time, we calculated the percent change from 100 to 150 msec (constituting the activation phase) as well as the percent change from 150 to 250 msec (constituting the reduction phase) for the high- and low-reward stimuli. In the activation phase, there was some evidence for an early increase in motor excitability for the high ($t(25) = 2.3$; $p = .03$, uncorrected;

Figure 2. TMS dynamics in Experiment 1. (A) High-reward go trials showed a linear increase in motor excitability across the four time points. Low-reward go trials showed an initial decrease in motor excitability (from 100 to 200 msec), followed by an increase from 200 to 250 msec. (B) On no-go trials, high and low reward showed an initial increase in motor excitability from 100 to 150 msec (activation phase), followed by a decrease from 150 to 250 msec (reduction phase). (C) On no-go trials, the percent change for the reduction phase was twice as strong (measured via effect size) for high- versus low-reward trials, and only high-reward trials showed a significant difference between percent change in the activation and reduction phases. (D) On no-go trials, greater reward-based (i.e., high minus low reward) activation was related to a greater reward-based reduction. Error bars represent *SEM* across participants. $\wedge p < .06$, $*p < .05$, $***p < .001$. All *p* values are adjusted according to Holm–Bonferroni correction.



$d = 0.45$) but not the low ($t(25) < 1$; *ns*, uncorrected; $d = 0.15$) reward stimulus. In the reduction phase, both the high and low reward stimuli showed significant decreases in motor excitability ($t(25) = 6.31$, $p < .001$ and $t(25) = 3.18$, $p = .03$, respectively); however, the effect size was more than twice as large for the high reward stimulus (high reward: $d = 2.5$, low reward: $d = 1.2$; Figure 2C). Moreover, only the high reward stimulus showed a difference in the percent change values between the activation and reduction phases (high reward: $t(25) = 4.96$, $p < .001$; low reward: $t(25) = 2.18$, *ns*; Figure 2C). Taken together, these results support the hypothesized activation–reduction dynamics and also suggest that a larger initial increase in motor excitability (induced by the high reward stimulus) influences the dynamics of the reduction phase. This is in contrast to the possibility that the activation and reduction processes are independent of one another, which would result in similar reduction slopes regardless of differences in initial activation.

Relationship between reward-based activation and reward-based reduction on no-go trials. We next asked if, across participants, the reward-based activation

(percent change in MEPs for high minus low reward from 100 to 150 msec) correlated with the reward-based reduction (percent change in MEPs for high minus low reward from 150 to 250 msec). There was a strong negative correlation ($r_{25} = -.74$, $p < .001$), such that participants who showed stronger reward-based activation also showed a stronger reward-based reduction (Figure 2D). For exploratory purposes, we also tested the relationship between the activation and reduction phases for the low- and high-reward no-go trials separately. For low-reward trials, there was a significant correlation between the activation and reduction processes ($r_{25} = -.52$, $p = .006$). For high-reward trials, the relationship between the activation and reduction processes did not reach significance ($r_{25} = -.32$, $p = .11$). However, as the results were strongly influenced by one significant outlier (Mahalanobis distance > 3), the relationship was significant with a non-parametric Spearman's test ($\rho = -.49$, $p = .01$) and when the outlier was removed from the analysis ($r_{24} = -.43$, $p = .03$). Together, these results show that the degree of reduction on no-go trials is influenced by the strength of the preceding activation. This could be explained by mere passive decay (what rises higher has further to fall) or by

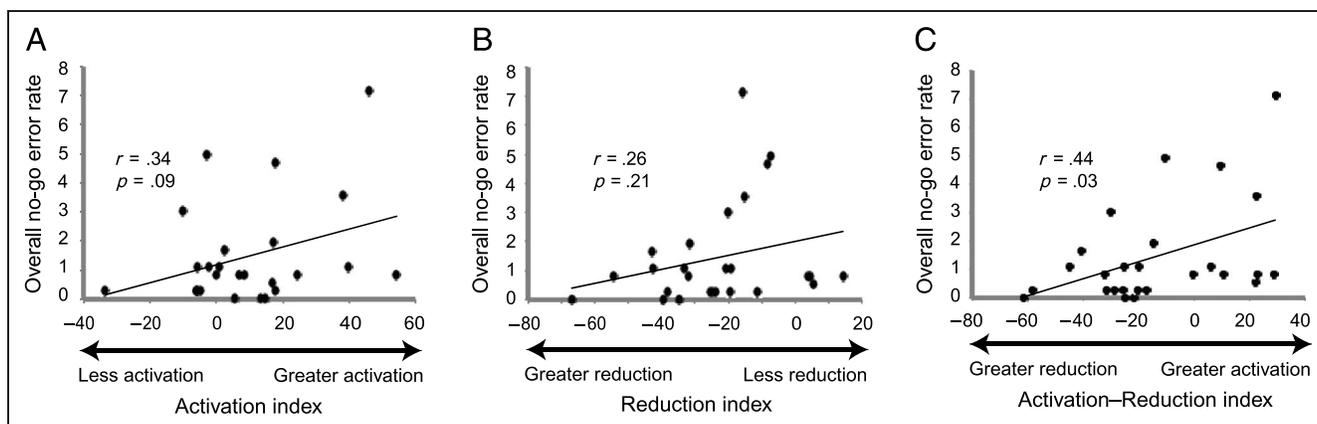


Figure 3. Relationship between the no-go error rate and the different phases of no-go trials. (A) Greater activation from 100 to 150 msec was marginally positively correlated with the no-go error rate. (B) Greater reduction from 150 to 250 msec was positively (but nonsignificantly) correlated with the no-go error rate. (C) The activation–reduction index on no-go trials (a composite measure of the activation and reduction phases) showed a significant positive correlation with the no-go error rate.

a top–down control process. This distinction is tested in Experiment 2.

Relationship between no-go dynamics and error rates.

We next examined how the activation, the reduction, and the activation–reduction processes together (reflected in the activation–reduction index) related to participants' self-control failures. Results showed that neither the activation nor reduction processes significantly correlated with participants' overall no-go error rates (activation phase: $r_{25} = .34$, $p = .09$; reduction phase: $r_{25} = .26$, $p = .21$).

However, there was a significant correlation between the activation–reduction index and no-go error rates ($r_{25} = .44$, $p = .02$; Figure 3A–C). Specifically, those people who showed a relatively larger increase in the activation phase compared with the decrease in the reduction phase made more errors on no-go trials.

Go and no-go dynamics for fast and slow RT groups.

The strength of response activation in a reward task such as this likely depends on the participant's basic level of motivational drive for reward. We therefore split participants

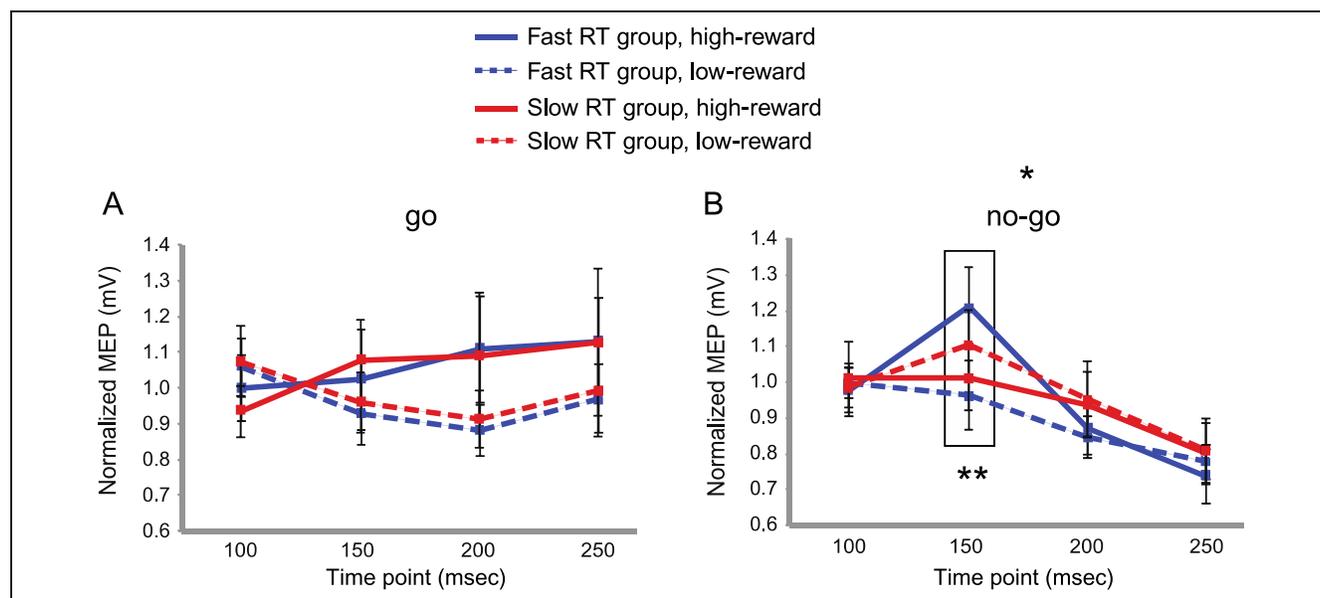


Figure 4. TMS dynamics based on fast and slow RT groups in Experiment 1. (A) Go trial dynamics. High-reward go trials showed an increase in motor excitability across all four time points, whereas low-reward go trials showed a decrease from 100 to 200 msec, followed by an increase from 200 to 250 msec. High- and low-reward go trials in the slow RT group largely resembled that of the fast RT group. (B) High-reward no-go trials in the fast RT group showed an initial steep increase (from 100 to 150 msec), followed by a sharp decrease (from 150 to 250 msec). This pattern was markedly different than low-reward no-go trials, which did not show the initial increase and also a less steep decrease. In contrast to the fast RT group, the slow RT group showed no differences in motor excitability between high- and low-reward no-go trials during the activation and reduction phases. Follow-up analyses showed that group differences in the reward motor dynamics were only in the 150-msec time point. Error bars represent SEM across participants. * $p < .05$ for Reward \times Pulse time \times Group interaction, ** $p < .05$ for Reward \times Group interaction.

into two groups based on RT on go trials (fast RT vs. slow RT, ostensibly reflecting high and low motivation, respectively). For go trials, a mixed ANOVA with the Reward (high, low) and Pulse time (100, 150, 200, 250 msec) as within-participant factors and Group as a between-participant factor (fast RT, slow RT) revealed a significant main effect of Reward ($F(1, 24) = 5.68, p = .026$), with greater MEPs for high- versus low-reward trials. There was also a significant Reward \times Pulse time interaction ($F(3,72) = 2.86, p = .043$; Figure 4A), as was the case in the main analysis with all participants. There was no main effect of or interactions with Group.

For no-go trials, there was a significant main effect of Pulse time ($F(3, 72) = 15.83, p < .001$), in which MEPs began to decrease at 200 msec after stimulus onset. The Reward \times Pulse time \times Group interaction was also significant ($F(3, 72) = 2.75, p = .049$; Figure 4B). We investigated the triple interaction with separate Reward \times Group ANOVAs for each of the four time points, as this would help reveal the specific time points that showed group differences in the reward motor dynamics. We

found a significant Reward \times Group interaction at only the 150-msec time point ($F(1, 24) = 7.46, p = .01$), in which the fast RT group showed a larger MEP difference between high- and low-reward trials than the slow RT group. This impression was confirmed with t tests that showed a significant high versus low reward difference in the fast RT group ($t(26) = 2.61, p = .02$) but no difference in the slow RT group ($t < 1, ns$). It should also be noted that the difference in overall no-go dynamics between the two groups cannot be readily explained by the group difference in overall RT, as this would predict similar patterns of activity, but at different latencies (which was not seen here). Thus, the group that responded more quickly overall on go trials (more putative motivational drive) showed greater sensitivity to the high reward stimulus on no-go trials, particularly at the 150-msec time point.

Percent change across time points on no-go trials for fast and slow RT groups. We now examined the percent change from 100 to 150 and 150 to 250 msec for the

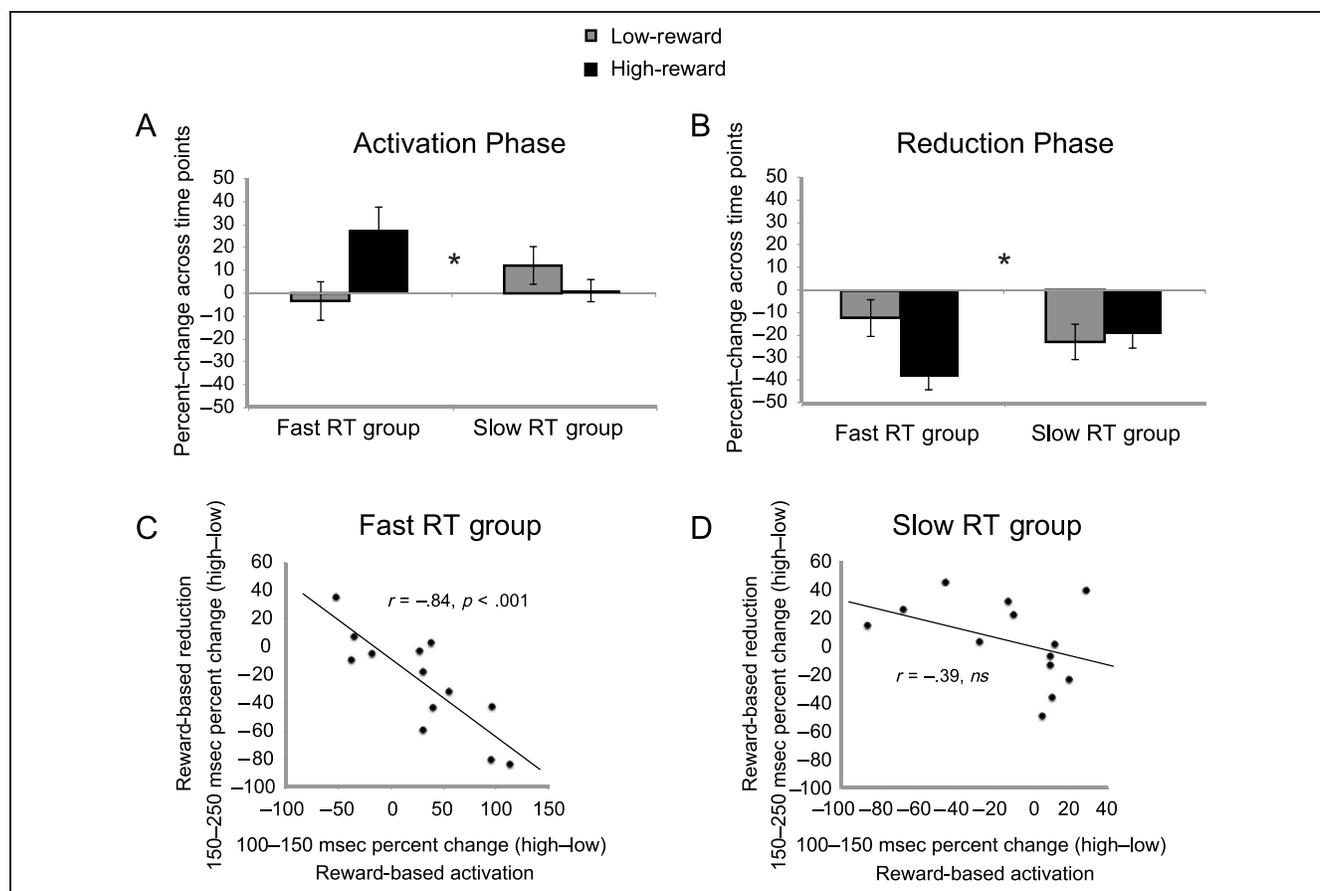


Figure 5. Percent change across time points for fast and slow RT groups on no-go trials, Experiment 1. (A) During the activation phase, the fast and slow RT groups showed different reward motor dynamics, resulting in a significant Group \times Reward interaction. Specifically, the fast RT group showed greater sensitivity to the high versus low reward stimulus. (B) During the reduction phase, there was also a significant Group \times Reward interaction, where the fast RT group again showed greater sensitivity to the high versus low reward stimulus. (C) The fast RT group showed a significant correlation between the degree of reward-based (i.e., high minus low reward) activation and reward-based reduction. (D) The correlation for the slow RT group did not reach significance; furthermore, a Fisher's r -to- z transformation test revealed that the correlation for the fast RT group was significantly greater than that for the slow RT group. Error bars represent *SEM* across participants. * $p < .05$ for Reward \times Group interaction.

activation and reduction phases on no-go trials, for the two groups. An ANOVA with Reward (high, low), Phase (activation, reduction), and Group (fast RT, slow RT) revealed a significant main effect of Phase ($F(1, 24) = 34.01, p < .001$) as well as a Reward \times Phase \times Group interaction ($F(1, 24) = 6.57, p = .017$). Follow-up ANOVAs for the activation and reduction phases separately showed significant Reward \times Group interactions for both phases (activation: $F(1, 24) = 5.62, p = .026$; reduction: $F(1, 24) = 5.41, p = .029$; Figure 5A and B). This is in line with the result above that pinpointed the 150-msec time point as the locus for differential reward motor dynamics across the groups, as it is the only time point that contributes to the percent change in both the activation and reduction phases.

Relationship between reward-based activation and reward-based reduction on no-go trials for fast and slow RT groups. A Pearson's correlation for the fast RT group showed a strong negative correlation between the reward-based activation and reward-based reduction across individuals ($r_{12} = -.84, p < .001$; Figure 5C). This correlation was not present for the slow RT group ($r_{12} = -.39, p = .18$; Figure 5D). A direct comparison of the two correlation coefficients using a Fisher r -to- z transformation showed that the correlation for the fast RT group was significantly stronger than that of the slow RT group ($Z = 1.82, p = .03$, one tailed). This again indicates that the reduction phase depends on the strength of preceding reward-based activation.

Relationship between trait impulsivity and reward-based MEP differences. Trait impulsivity was only significantly correlated with the peak activation point on no-go trials ($r_{25} = .39, p = .047$, uncorrected for four comparisons), such that higher impulsivity was related to greater sensitivity to the high versus low reward stimulus at the peak point of activation. This suggests that trait impulsivity is related to the reward value in the activation process.

Discussion

TMS was delivered at 100, 150, 200, or 250 msec after a high or low reward stimulus on go and no-go trials to map the dynamics of putative response activation and control. On go trials, the high reward stimulus produced an early motor activation (within 150 msec) that preceded the average RT by almost 350 msec. In contrast, the low reward stimulus showed an initial decrease in motor activation (from 100 to 200 msec), followed by an increase (from 200 to 250 msec),² resulting in a significant high versus low reward difference at 150 msec.

On no-go trials, the reward stimuli (especially the high reward stimulus) induced a brief increase in motor excitability (from 100 to 150 msec), followed by a sharp reduction (from 150 to 250 msec) that reached levels far

beneath the prestimulus baseline. Notably, those participants with greater activation also showed greater reduction on no-go trials. This suggests that the dynamics of the reduction phase depend on the strength of early reward-driven activation and also suggests that both processes are important when evaluating one's ability to withhold a reward-driven action. In support of this, we found that only a measure that takes into account both the activation and reduction processes together was predictive of participants' overall errors rates on no-go trials. This indicates that higher levels of reward-driven activation are detrimental if a proportionately larger reduction process does not follow. Furthermore, this result highlights the importance of using self-control paradigms that can capture both the provocation and control processes with high temporal resolution.

We also found that individuals with higher motivational drive showed greater sensitivity (i.e., stronger activation and reduction processes) to the high versus low reward stimulus on no-go trials. This indicates that the influence of reward value on the activation–reduction dynamics was highly dependent on participants' motivational drive for reward and also provides further support for the close activation–reduction relationship. Finally, individual difference analyses across all participants revealed that, at the peak point of activation (150 msec), trait impulsivity was positively correlated with the degree of reward-based activation. This indicates that trait impulsivity is related to the reward value in the activation process and, in accordance with our other findings, suggests that greater recruitment of control mechanisms may be required when impulsive individuals view a high reward stimulus. However, this results should be interpreted with caution, as the analysis was not corrected for multiple comparisons.

We interpret the sharp, beneath-baseline reduction in motor excitability on no-go trials as a top–down suppression process that depends on the strength of preceding activation. However, other accounts exist. For example, it is possible that a similar degree of control is instantiated on high- and low-reward no-go trials and that the steeper reduction on high-reward trials is simply a side effect of there being higher initial activation (i.e., further to “fall”). In this case, the reduction phase would reflect a control process that does not necessarily depend on the strength of preceding activation. It is also possible that the reduction phase merely reflects a passive withdrawal of voluntary drive, which could also manifest in reduced motor excitability. In the next experiment, we test these competing accounts using a well-established finding that failures in self-control tend to increase when immediately following a very demanding task (Heatherton & Wagner, 2012; Baumeister & Heatherton, 1996). We reasoned that, if there is top–down response suppression and its strength depends on the preceding activation, then there should be more effortful control recruited on high- compared with low-reward no-go trials. This then predicts that

depleting top-down resources with a demanding earlier task will increase the no-go error rate more for high- versus low-reward trials in our rewarded go/no-go paradigm.

EXPERIMENT 2

Two groups of participants performed the rewarded go/no-go paradigm before and after an extended working memory (WM) task (one group, easy; one group, difficult). We chose a WM manipulation because it allowed us to tax top-down control brain regions, including lateral pFC and parietal cortex (Zanto, Rubens, Thangavel, & Gazzaley, 2011; Owen, McMillan, Laird, & Bullmore, 2005; Braver et al., 2001) that would ostensibly be important for controlling the rapid activation on high-reward no-go trials that we observed in Experiment 1.

On the basis of our hypothesis that top-down response suppression was engaged more on high- versus low-reward no-go trials, we made two specific predictions. First, we predicted that, when top-down resources are depleted (i.e., the high-load group), the change in error rate for high-reward no-go trials would be greater than that for low-reward no-go trials. Second, we predicted that, when top-down resources are not depleted (i.e., the low-load group), there would be no difference between high- and low-reward trials.

Methods

Participants

Forty-two (10 male) participants were tested (mean age = 21.36 years, $SD = 5.5$ years; all right handed). Two participants were excluded because of technical malfunctions. One participant in the low-load group was excluded for having a high-reward error rate of 50%, which was more than 5 SDs from the group mean. Thus, all analyses were run on 39 participants, with 19 participants in the low-load group (mean age = 21.5 years, $SD = 7.7$ years) and 20 participants in the high-load group (mean age = 21.15 years, $SD = 1.9$ years). All participants provided institutional review board consent.

Task and Procedure

There were three parts to the procedure (see Figure 6A). In Part 1, all participants completed a task identical in design to the rewarded go/no-go task in Experiment 1 (Figure 1), with the only difference being that there were now four blocks of 48 trials, yielding 208 total trials. As in Experiment 1, the first block was considered a “learning block” and was not included in the analysis. The data from this rewarded go/no-go task served as a baseline measure for each participant to determine the change in the no-go error rate after the WM manipulation.

In Part 2, participants were assigned to either the low- or high-load WM group. For both groups, consonant letters appeared one at a time on the screen (letter duration = 0.75 sec, ITI duration = 1.75 sec). Participants in the low group were instructed to make a response (using their left index finger to make the tasks as orthogonal as possible) as quickly as possible every time the letter “P” appeared on the screen (on trial n). For all other letters, no response was to be made. Participants in the high group were instructed to make a response (using their left index finger) as quickly as possible every time they saw the same letter as presented three letters before (on trial $n-3$). For all other letters, no response was to be made. Participants in this group were told that, to complete the 3-back task, they had to hold three letters at a time in WM and continuously update the three letters with every new letter presentation. For both groups, there were six blocks of 100 analyzable trials (for the 3-back task, the first three trials of each block were excluded), yielding 600 analyzable trials. Participants were given a 20-sec break between each of the six blocks. All participants completed a practice session of 30 trials. In total, Part 2 took approximately 30 minutes for both groups.

In Part 3, participants again completed the rewarded go/no-go task. However, there were now three blocks of 48 trials (yielding 144 total trials), but with no learning block (because the color-reward relationships had already been learned in Part 1). Thus, the number of analyzable trials was identical for Parts 1 and 3.

Data Analysis

The main dependent measure was the change in error rate on no-go trials from before the WM manipulation to after (pre to post). We therefore calculated post-minus-pre difference scores in the error rate for high and low reward in both load groups. To test for pre-to-post changes in error rate, we used one-sample t tests to compare the conditions against a value of zero (representing no pre-to-post change). We also directly compared the pre-to-post change for high versus low reward with paired t tests. On the basis of the strong directional predictions, one-tailed tests were used.

Results

We first verified that the WM manipulation was successful. A two-sample t test showed that performance in the low-load condition was significantly better than in the high-load condition ($t(38) = 8.58, p < .001$; Figure 6B). We then verified that, before the WM manipulation, there were no group differences in high- minus low-reward RT or total errors (all $ps > 0.2$; see Table 1 for behavioral measures). Finally, we verified that there were no pre-to-post changes or group differences for overall RT or number of presses ($t < 1, ns$), suggesting that cue processing speed

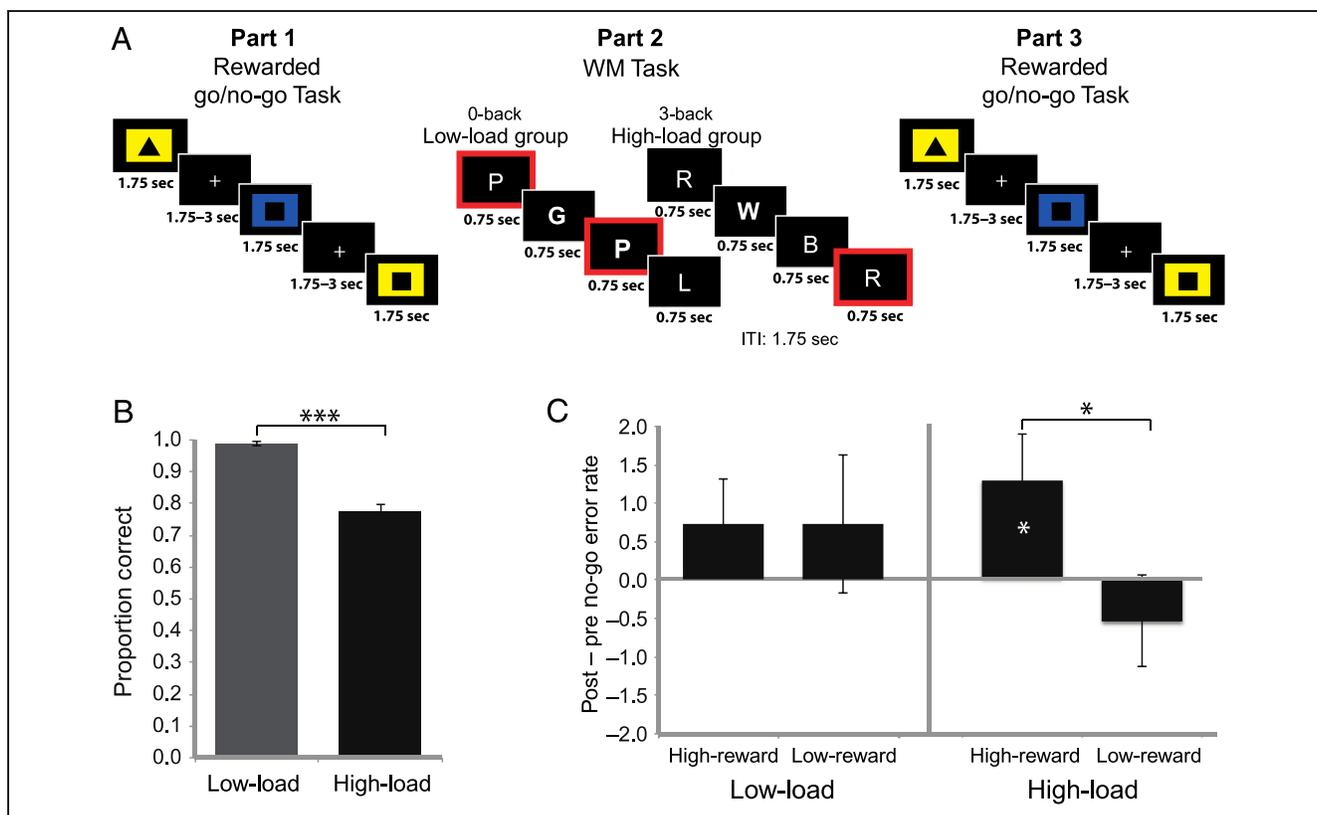


Figure 6. Experiment 2 task design and results. (A) There were three parts in the task. In Part 1, participants completed the rewarded go/no-go task (as in Experiment 1). This provided a baseline measurement for each participant's no-go error rate on high- and low-reward trials. In Part 2, participants either completed a cognitively demanding 3-back WM task (high load) or a less demanding 0-back task (low load). For the 0-back task, they were required to press a button (indicated by a red outline) whenever they saw the letter "P." For the 3-back task, they were required to press a button whenever the current letter matched the letter presented three letters before. In Part 3, participants completed another rewarded go/no-go task to examine the change in error rates on high- and low-reward no-go trials after the WM manipulation. (B) A proportion correct measurement showed that the high-load (3-back) task was significantly more difficult than the low-load (0-back) task. (C) Only high-reward trials in the high-load group showed a significant increase in the error rate after the WM manipulation. This increase was significantly greater than the low-reward trials in the high-load group. The low-load group showed no difference between high- and low-reward no-go trials and no pre-post changes in error rates. Error bars represent *SEM* across participants. * $p < .05$, *** $p < .001$.

and motivational drive were not affected by the WM manipulation.

Our main analysis showed that, in the high-load group, there was a significant pre-to-post increase in error rate for the high ($t(19) = 2.13, p = .02$) but not the low ($t(19) < 1$) reward stimulus and a significant difference between the high and low reward stimuli ($t(18) = 2.29, p = .02$; Figure 6C). For the low-load group, there were no significant pre-to-post changes for either the high or low reward stimulus, nor was there a difference between the two con-

ditions (all $ps > .2$; Figure 6C). A direct comparison between the groups using a mixed ANOVA with load (high, low) as a between-participant factor and reward (high, low) as a within-participant factor showed a trending interaction ($F(1, 37) = 2.1, p = .08$).

Discussion

One group of participants underwent the rewarded go/no-go task, then an easy (low-load) WM task, and then

Table 1. Behavioral Measures for Experiment 2

	Load	Overall RT (msec)	High RT (msec)	Low RT (msec)	Overall Error Rate	High Error Rate	Low Error Rate
Pre	Low	419.3 (62)	406.1 (64)	434.7 (67)	2.56 (2.33)	3.65 (3.47)	1.46 (2.68)
	High	418.4 (40)	408.2 (48)	431.1 (43)	1.60 (2.22)	1.81 (2.89)	1.39 (2.63)
Post	Low	410.6 (63)	393.4 (60)	425.7 (64)	4.93 (3.94)	4.39 (3.38)	2.19 (3.28)
	High	412.2 (43)	398.3 (43)	430.9 (48)	2.92 (4.80)	3.06 (3.92)	0.83 (3.13)

the rewarded go/no-go task again, whereas another group of participants did the same sequence but with a difficult (high-load) WM task. The high-load version was an effortful 3-back WM paradigm that putatively “depletes” cognitive resources commonly involved in top-down control (Chmielewski, Mückschel, Stock, & Beste, 2015; Mitchell, Macrae, & Gilchrist, 2002). In the high-load group, we observed a significant pre-to-post increase in the no-go error rate only for high-reward trials, which was significantly greater than the pre-to-post change for low-reward trials. This was not the case for the low-load group, most likely because top-down resources were not depleted. Ideally, there would also be a significant difference between groups, which was only present here at a trend level. However, the comparison was not between high WM and no intervening task but between high- and low-load WM (which would also be depleting to some extent). Notwithstanding, the increased error rate for high- versus low-reward no-go trials in the high-WM group suggests that greater top-down control is needed on high-reward no-go trials. This, along with the evidence for stable levels of motivation, argues against the possibility that the reduction phase reflects a withdrawal of voluntary drive and is in line with the hypothesis that it reflects a top-down suppression process that is related to the strength of preceding activation.

GENERAL DISCUSSION

Recent studies suggested that motivationally driven action tendencies can be countered by a response suppression mechanism (Freeman et al., 2014, 2015), but they did not reveal the putative activation-suppression dynamics. It was therefore unclear if the motor reduction previously observed on no-go trials was preceded by an early rise in reward-driven motor activation and whether the reduction was directly related to the putative early activation. Here, we employed a rewarded go/no-go paradigm with better characteristics for mapping out the corticospinal dynamics. In Experiment 1, we found that no-go trials showed an initial activation phase (within 150 msec), followed by a sharp reduction phase (within 200 msec) that fell beneath prestimulus baseline levels by 250-msec poststimulus onset. The activation-reduction pattern was more pronounced (i.e., showed a greater magnitude in the slope change) for high- versus low-reward no-go trials, indicating that the reduction phase was related to the degree of preceding activation. In support of the activation-reduction link, there was a strong correlation between the amount of activation and reduction across individuals, suggesting that both processes are important when evaluating one's ability to withhold a reward-driven action. In line with this, we found that a measure that takes into account both the activation and reduction processes together was predictive of participants' overall errors rates on no-go trials. Moreover, subgroup analyses revealed the importance of taking into account in-

dividuals' basic motivational drive for reward, as individuals with higher drive showed greater sensitivity (i.e., response activation) toward a high reward stimulus, followed by a steeper reduction slope. We hypothesized that the steeper reduction slope reflects greater top-down suppression. If so, then depleted cognitive resources should increase the no-go error rate more for high- versus low-reward trials, which is what we found in Experiment 2. Thus, our results show that, when it is inappropriate to respond, reward-predicting stimuli still induce an early rise in motor activation that is subsequently controlled. This leads to a reduction of motor excitability well beneath baseline, and the strength of this reduction appears to depend on the strength of the preceding activation. Together, these results suggest that controlling reward-driven responses may critically depend on the tight relationship between the activation and reduction phases and that a weakened reduction process can lead to failures in self-control.

What is the top-down control process that apparently “kicks in” on high-reward no-go trials? One possibility is that it is response suppression, as we have previously postulated. This is consistent with many response control studies that have demonstrated the recruitment of an active suppression mechanism that countermands an action tendency from a prepotent or an already-initiated response, for example, in the stop signal paradigm (Schmidt, Leventhal, Mallet, Chen, & Berke, 2013; Aron, 2007; Aron & Poldrack, 2006). It is also consistent with several stop signal and go/no-go studies that have used spTMS to characterize the timing of the putative response suppression process on stop or no-go trials. In particular, those studies have typically observed response suppression at 140–200 msec after stimulus onset (van den Wildenberg et al., 2010; Stinear et al., 2009; Coxon, Stinear, & Byblow, 2006; Yamanaka et al., 2002; Hoshiyama et al., 1997), which closely mirrors the timing in the current study.

An alternative top-down control process could be attentional modulation. This could direct resources away from the reward stimulus and/or toward the no-go cue (Hickey & Peelen, 2015; Harris, Hare, & Rangel, 2013; Giesbrecht, Woldorff, Song, & Mangun, 2003; Hopfinger, Buonocore, & Mangun, 2000). For example, the study by Harris et al. (2013) found evidence for an early attentional filtering mechanism during the exercise of self-control in the face of appetitive food items. Notably, the attentional filtering mechanism was instantiated 150–200 msec after stimulus onset, which also closely resembles the timing of the reduction phase in the current study. It is therefore possible that, here, the reduction phase actually reflects a reduction in motivational drive after reduced processing (via attentional control) of the reward stimulus. Notwithstanding this possibility, this attention explanation has difficulty accounting for the beneath-baseline reduction we observed. Instead, an attentional filtering of the reward stimulus would more likely cause a reduction in motor excitability to prestimulus onset levels where no stimulus is displayed.

Future studies could more definitively disentangle such mechanisms with functional neuroimaging. For example, a response suppression account predicts the involvement of regions implicated in stopping action, such as the right inferior frontal gyrus, pre-supplementary motor area, and subthalamic nucleus (Chambers, Garavan, & Bellgrove, 2009; Aron, 2007; Aron & Poldrack, 2006; Garavan, Hester, Murphy, Fassbender, & Kelly, 2006). Alternatively, the attentional control account predicts the involvement of regions implicated in downmodulating task-irrelevant distractors, such as the superior frontal cortex, inferior frontal junction, and parietal cortex (Zanto et al., 2011; Giesbrecht et al., 2003; Hopfinger et al., 2000). Ultimately, clarifying the underlying mechanism could help inform when and how control is implemented over reward-driven provocations. In turn, this information could be useful in determining optimal tasks that may be used to train individuals over extended periods in an effort to reduce failures in self-control.

Here, we show that one condition that leads to increased failures in self-control is when a strong activation must be withheld (i.e., on high-reward no-go trials) after top-down resources have been heavily taxed. This result fits with a large literature that finds increased failures in self-control immediately after cognitive resources have been “depleted” in a separate effortful task (often referred to as “ego depletion”; Hagger, Wood, & Stiff, 2010; Baumeister & Heatherton, 1996). To account for these findings, it is thought that self-control draws from a somewhat global, limited resource and that exhausting it reduces the amount (or allocation) of available self-control resources to be deployed in the near future (Baumeister, 2014; Gailliot et al., 2007; Baumeister & Heatherton, 1996). An alternative theory explains the decrement in self-control as a decrease in participants’ motivational state during the second task (Inzlicht, Schmeichel, & Macrae, 2014). Our results argue against the motivational account, as we found no pre-to-post changes in participants’ motivational drive for reward (measured via RT and number of presses). Moreover, it is unclear why the motivational change would only occur for high-reward no-go trials, as the low-reward no-go trials showed no pre-to-post change. Instead, our results suggest that the reduction phase reflects a top-down control process and that the implementation of top-down control is affected by a demanding WM task.

The current approach has greater ecological validity than typical studies of response control, as we have studied the control over a reward-driven response tendency rather than merely a response tendency that is preestablished or automatic (as in the Simon or Flanker tasks). Yet, our approach is still limited by the fact that the no-go cue is an external signal.³ In many real-world situations of self-control, there is no cue or signal instructing individuals to withhold an action. Instead, people must often generate the control process in an endogenous manner (there are, however, some real-world situations

that are analogous to the current case; for example, the no-go trials in the current study are perhaps analogous to the scenario in which a smoker views a pack of cigarettes that has a large warning message on the front). Although paradigms have been designed to investigate endogenous control (for a review, see Ridderinkhof, van den Wildenberg, & Brass, 2014; Filevich, Kühn, & Haggard, 2012), studying endogenous control poses several challenges. For one thing, withholding a response endogenously is a subjective, decision-based process, which makes it difficult to measure a response inhibition failure. For another, the timing of the activation and control processes is more variable, which could limit the use of techniques such as spTMS to map the dynamics. Future studies will therefore benefit from discovering neural markers that signify both the activation and control processes within a single task, as this will allow a “readout” of their timing and relative strength during endogenous recruitment. A second limitation of the current study is that, from Experiment 2, we could only infer that the increase in errors was because of a change in the reduction phase dynamics. A future study could more definitively establish that this is the case using spTMS with the rewarded go/no-go task after a depletion manipulation. We predict that, whereas the activation phase would show a similar pattern as we observed here, the reduction phase on high-reward trials would show a less steep decrease in motor activity, thereby eroding its relationship with the preceding activation.

In conclusion, we show that, when a reward-driven action was withheld, there was an initial rise in motor activation that was modulated by the value of the reward-predicting stimulus and the individual’s motivational drive for reward. Furthermore, the initial activation phase was followed by a steep reduction in motor excitability, with the degree of the reduction corresponding to the strength of preceding activation. This pattern of dynamics, along with the observation that an effortful task apparently depletes the ability to withhold a response in the face of the high reward stimulus, suggests that the control process involved top-down response suppression. Future studies could validate this, which would highlight the importance of using response suppression to control provocations driven by the motivational content of a stimulus. More generally, these dynamics suggest that failures in controlling reward-driven actions may be due to insufficient or depleted response suppression mechanisms that follow a quick rise of reward-driven activation. This may explain why self-control is more difficult and fails more often when following demanding tasks (van der Linden, Frese, & Meijman, 2003; Baumeister, Bratslavsky, Muraven, & Tice, 1998) or consumption of substances (e.g., alcohol; Kähkönen, Wilenius, Nikulin, Ollikainen, & Ilmoniemi, 2003; Moselhy, Georgiou, & Kahn, 2001) that reduce functioning in brain regions involved in top-down control. Specifically, our findings suggest that reduced functioning in top-down control

may lead to a weakened suppression process, contributing to failures in self-control.

Acknowledgments

This work was supported by U.S. National Institutes of Health grant DA026452 and James S. McDonnell Scholar Award 220020375 to A. R. A. and by a National Science Foundation Graduate Research Fellowship to S. M. F. We would also like to thank Dianze Lu for assistance with data collection.

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Notes

1. These early MEP differences between high- and low-reward trials are probably not merely because of faster RTs on high-reward trials for several reasons. First, the MEP differences were, on average, more than 280 msec before the mean high-reward RT. As previous studies have shown that increases in MEP amplitude resulting from voluntary movement initiation generally occur only about 100 msec before the RT (Stinear et al., 2009), this result is likely independent of RT differences. Second, we computed RT difference scores (high minus low reward) for each individual and correlated these with their MEP difference scores (high minus low reward) at the 150- and 200-msec time points. If the high versus low MEP results were merely because of differences in RT, we would expect that participants with a larger behavioral effect (high vs. low) would also show a larger MEP effect (high vs. low). There was no evidence for a significant interparticipant correlation across these difference scores at the 150- or 200-msec time point ($p > .4$), indicating that the MEP differences were likely not influenced by differences in RT. Finally, the finding of differential MEP activity as a result of differential reward value has been found in several previous studies that have pulsed in a response-locked fashion (e.g., Klein-Flügge & Bestmann, 2012), suggesting that the differences observed here are not because of the stimulus-locked TMS pulses.

2. Whereas the increase in motor excitability for high-reward go trials was in line with our predictions, the initial decrease on low-reward go trials was surprising. One intriguing possibility for the decrease is that making effortful instrumental responses to a low reward stimulus may be somewhat aversive (Talmi, Dayan, Kiebel, Frith, & Dolan, 2009; Hare, O'Doherty, Camerer, Schultz, & Rangel, 2008), which could in turn trigger a quick inhibitory response over the motor system (Verbruggen, Best, Bowditch, Stevens, & McLaren, 2014). This possibility warrants further investigation, particularly in light of the translational implications of establishing a physiological link between stimulus aversion and its influence on triggering motor inhibition (Chiu, Cools, & Aron, 2014).

3. There are also some remaining questions pertaining to the results. For example, it is unclear if the dynamics observed in the current study would resemble the dynamics in the paradigm used in Freeman et al. (2014), where the background stimulus is a task-irrelevant Pavlovian cue that motivates instrumental responding. It is also unclear why the initial reward-based activation process on no-go trials (from 100 to 150 msec) did not more closely match the go trials during the same period. Finally, we are not certain why the fast RT group's greater sensitivity to the high reward stimulus was observed on no-go trials but was not observed on go trials.

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