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INVESTIGATION OF MOTOR INHIBITION INFLUENCE ON WORKING MEMORY
REPRESENTATIONS

by

Alec Mather

A thesis submitted in partial fulfillment of the requirements
for graduation with Honors in the Psychology

Jan Wessel
Thesis Mentor

Spring 2019

All requirements for graduation with Honors in the
Psychology have been completed.

Debra Johnson
Psychology Honors Advisor

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Abstract

Motor inhibition is a cognitive control ability that allows humans to rapidly stop an action even after initiation. Previous research has demonstrated that motor inhibition influence can extend beyond the action one is trying to suppress (Wessel & Aron, 2017). For example, stopping an action initiated in the right hand will also decrease muscle excitability in task-unrelated leg muscles. This discovery led to a global theory of inhibition, which tries to explain this non-selective nature of the inhibition process. Researchers began studying this inhibitory process with Electroencephalography (EEG) and found that the psychological motor inhibition process was reflected in a neural signature, known as the fronto-central P3 event-related potential, that indexes successful response inhibition (Wessel & Aron, 2015). With a way to index response inhibition, scientists were able to demonstrate P3 activation and decreased muscle excitability in task-unrelated muscles when following surprising events (Dutra et al., 2018), which lead to a reappraisal of the breadth of psychologically relevant events that induce inhibitory effects. Scientists then began to wonder if this global theory of inhibition extended even beyond the motor system itself. Indeed, working memory was also inhibited when the P3 component was activated via surprise (Wessel et al., 2016). In our study, we designed a new way of quantifying this disruption of working memory by inhibitory control systems during a hybrid working memory/go-nogo task. Specifically, we tested working memory precision when participants received a go-trial (no inhibition) or a nogo-trial (inhibition).

Introduction

Motor inhibition is a cognitive control ability that allows humans to stop an action before, during or after initiation, but before its final execution. It is important to study motor inhibition because it is a flexible, adaptive system that is applied in our everyday lives. For example, imagine that you are driving a car and need to make a right turn. Upon starting to turn your steering wheel you see a child running from around the corner directly into your path. Motor inhibition is our ability to cancel our ongoing actions, accelerating into the turn, so that we can reevaluate our environment. Motor inhibition is most commonly studied using the Stop-Signal (Logan et al., 1983) or Go-Nogo (Wessel, 2018) tasks. The Stop-Signal task more so resembles our “car driving” example because all trials begin by looking for a response, action initiation, but a subset of trials have a delayed signal that will tell the participant that they should *not* respond. This means that participants will have to stop themselves from responding *after* having initiated a response, similar to stopping a car to avoid the child running into the street. The Go-Nogo paradigm differs slightly in that only a subset of trials (~70%) ask for a response, go-trials, while the rest of the trials tell the participant not to respond (~30%), nogo-trials. It is important in both of these paradigms that participants make errors by responding on trials when they shouldn't. This is important because participants with a 100% success rate are probably deciding how to respond rather than canceling actions. However, if the participant is making mistakes we know that they are probably initiating responses and sometimes able to inhibit them, and other times not able to inhibit them. It is the process of canceling an action that has already been initiated that we are interested in studying. On a neural level, stop trials on both of these paradigms have been shown to recruit a fronto-basal ganglia network in order to successfully inhibit ongoing motor potentials (Aron et al., 2014; Jahanshahi et al., 2015; Wessel & Aron, 2017; Ridderinkhof et al., 2011). This fronto-basal ganglia network begins at the presupplementary motor area (preSMA) and right inferior frontal cortex (IFC) - prefrontal nodes trigger the stopping process. These nodes in the right IFC and preSMA project hyperdirect pathways to the subthalamic nucleus (STN). The STN then excites the Globus Pallidus Pars Interna (GPi), which then suppresses the thalamocortical drive. The thalamocortical drive projects to the primary motor and premotor cortex, which, when suppressed, reduces the likelihood that the action will be executed. However, motor potentials may not be the only system that gets suppressed when this fronto-basal ganglia system is engaged. Recent work has

demonstrated that surprising events can engage the same independent component signature that represents fronto-basal ganglia recruitment (Dutra et al., 2018). Using surprising events to engage this network, a study published in the Nature Communications journal demonstrated that engaging the fronto-basal ganglia network disrupted ongoing working memory (WM) (Wessel et al., 2016). Therefore, it is fair to ask whether the inhibitory influence of this system, engaged via motor inhibition, can suppress more than just motor potentials, could it also disrupt ongoing cognitive representations? This would make the prediction that if an action had to be stopped, the fronto-basal ganglia system would be recruited and cognitive representations would be inhibited. In this study, we tested whether stopping an action disrupts the visual working memory system during a hybrid working memory/go-nogo task.

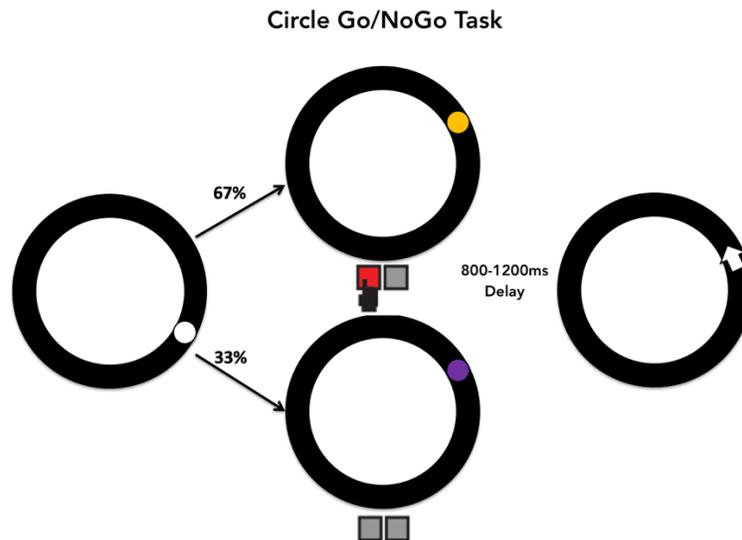
Circle Go-NoGo (CGNG) Task Description

The task we designed is a hybrid visual working memory/go-nogo task. The idea was to create a task in which participants had to maintain a visual representation in their mind and also respond or cancel their response. The working memory element of this task is the participant's ability to hold a visual representation of the location of the memory stimulus' color change. It is assumed that a strong working memory representation would yield responses that are closer to the true location, and that disrupted working memory representations would yield responses at greater variance (i.e. less accurate responses). In this paradigm we created 3 trial types: go-trials, nogo-trials and baseline trials. Go-trials tell the participant to make a response, nogo-trials tell the participant to cancel their response and baseline trials were performed in separate blocks and do not require a go/nogo response; they only require a memory response. We predict that nogo-trials will recruit the fronto-basal ganglia network and disrupt visual working memory thereby leading to less accurate memory responses compared to trials that do not have a stop-signal. We also predict that baseline trials will yield the most accurate responses, as those trials demand less cognitive resources, allowing for a stronger visual working memory. In short, accuracy should go from best to worst in the following order: baseline trials, go trials, stop trials.

Experiment 1 Materials and Methods

This study included 25 young, healthy, University of Iowa undergraduate students. 6 Students were removed from the data set due for various reasons leaving 19 available for

analysis. Of those 19 participants we gathered the following demographic information: 7 male, 12 female; 17 right handed, 2 left handed; 4 Asian, 15 white; mean age = 18.32, STD = 0.58. Participants were compensated with class completion credit via the University of Iowa's online SONA recruitment website.



All stimuli were presented using Psychtoolbox under MATLAB 2015b. All stimuli were presented against a black background. The ring, upon which the memory dot stimulus traveled, had a radius of 225 pixels and a thickness of 60 pixels. The go/stop-signal colors were counterbalanced between purple (RGB: [255 165 0]) and yellow (RGB: [225 0 255]). Baseline trials color change was always presented with a light coral color (RGB: [255 128 128]). The memory stimulus had a radius of 30 pixels so as to fit inside the width of the ring. Participants used foot pedals to make a response with their right (clockwise) or left (counter clockwise) foot.

The task began with the ring and fixation cross at the center of the screen. After 500ms a black dot, the memory stimulus, would appear somewhere on the ring and start traveling either clockwise (50%) or counterclockwise (50%). The dot will travel approximately 60-70% of the ring's circumference. While traveling the dot would change colors at a randomly selected point along the middle one third of its full path length. The color to which the dot changes (we used purple and yellow) will tell the participant whether or not they should respond. One color would signify the go-signal (2/3 of trials), and the other the nogo-signal (1/3 of trials). For example, if a participant is assigned purple as his/her go signal, then receiving a purple color change would signify a go-trial, and yellow would signify a nogo-trial. Color patterns were counterbalanced among participants. The responses were made with foot pedals and mapped onto the direction

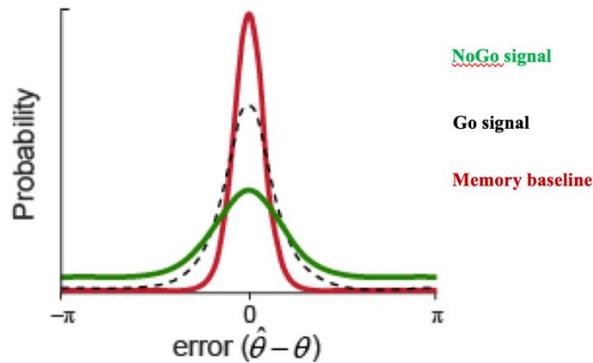
that the dot was traveling around the ring. For example, if the dot was traveling clockwise and changed into the go-signal, then the participant would respond on the right foot pedal using their right foot, and vice-versa. If the dot changed colors into the stop-signal, the participants were instructed to stop themselves from responding. After the dot finished traveling its full path length everything on the screen would vanish for a delay period of 1000ms. Then, the ring would appear again with the question printed on the screen, “Where on the ring did the dot change colors?” This aspect of the experiment evaluated the accuracy of working memory retrieval. The participant would then use the mouse, set to the center of the screen at the end of each trial, to click the spot on the ring where they think the dot changed colors; this was probed regardless of signal type. The location of their click would generate a black dot on the ring, signifying their attempt, along with that trial’s go/stop-signal to indicate the true location of the color change. The response and true locations would last for 1000ms. Finally, everything would vanish off the screen and wait for a variable inter-trial-interval of 800-1200ms. These trials were repeated for 8 blocks of 54 trials per block. There was also a separate, baseline portion of this task with the following amendments: no responses were made with foot pedals, dot color changes were always a light coral color (a mathematical blend between the go/stop-signal colors), participants only goal was to remember where the dot changed colors. Baseline trials were repeated for 3 blocks of 48 trials per block. The order of completing the 3 blocks of baseline trials and 8 blocks of the full experiment were counterbalanced among participants.

Experiment 1 Results

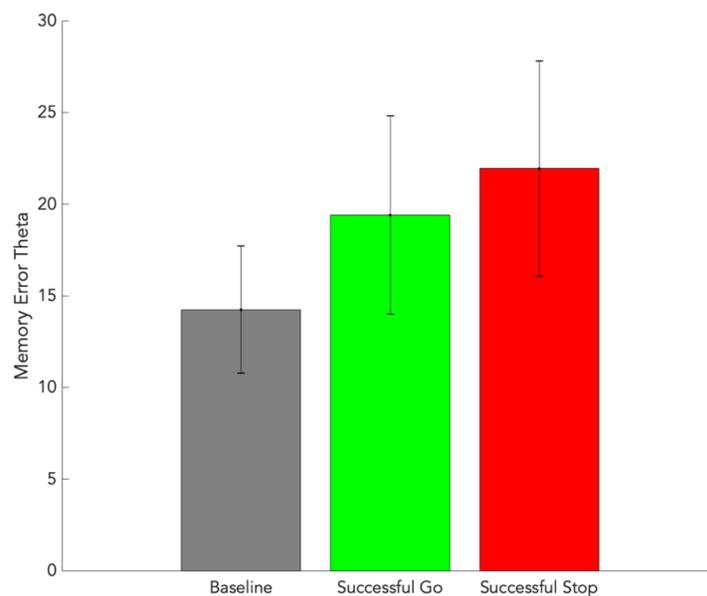
Analysis was done using MATLAB 2015b.

We collected 4 dependent variables based on the trial type and outcome of the trial which were RT (reaction time), Error Rates (pressing the wrong foot pedal on a go-trial), Successful Stops percentage (not making a response on a nogo-trial), Failed Stops percentage (making a response on a nogo-trial), and Miss Rates (not making a response on a go-trial).

Memory accuracy was measured in degrees and scaled between -180° and 180° . An accuracy of 0° would indicate a response location directly on the true value of the color change. Accuracies in the negatives would indicate a response value counter clockwise in relation to the true location, and positive numbers clockwise to the true location. A normal predicted distribution of the “forgetting curve” can be demonstrated by the figure below.



Overall memory thetas (distance between true value and response in degrees) were calculated for baseline trials ($\theta = 14.24$, $STD = 3.46$), successful go-trials ($\theta = 19.4$, $STD = 5.41$) and successful stop trials ($\theta = 21.94$, $STD = 5.87$). Thetas were calculated by taking the standard deviation in order to increasingly punish responses further from the target. A one-way ANOVA revealed a significant difference between baseline, successful go and successful nogo group means ($F(3/54) = 14.96$, $p < 0.001$). T-tests were then used to compare all individual participant successful go-trials and successful stop trials to reveal a significant effect ($p = 0.038$, $t = 2.02$) of response inhibition on memory. This means that successfully stopping one's self on a nogo trial was causing a significant disruption of the visual working memory. We also performed t-tests between successful go-trials and baseline trials to reveal a significant difference ($p = 0.001$), and also between successful nogo-trials and baseline trials to reveal a significant effect ($p < 0.001$).



Experiment 2 Intro

As discussed earlier, surprising events can also recruit the fronto-basal ganglia structure (Dutra et al., 2018). This introduces the problem of what exactly could be disrupting visual working memory in this paradigm. Is it, as we predicted, motor inhibition on nogo-trials, or is the fact that nogo stimuli are significantly less frequent, and therefore possibly surprising? In order to address this problem, our second experiment reverses the go to nogo trial ratio (1/3 go, 2/3 nogo). If our theory is correct, that visual working memory is disrupted by action inhibition, then response thetas should remain consistent with experiment 1. However, if visual working memory is being disrupted by the infrequent stimuli, then we should see the reverse effect between go and nogo trials and the new order of most accurate to least accurate trials should be the following: baseline, nogo, go.

Experiment 2 Materials and Methods

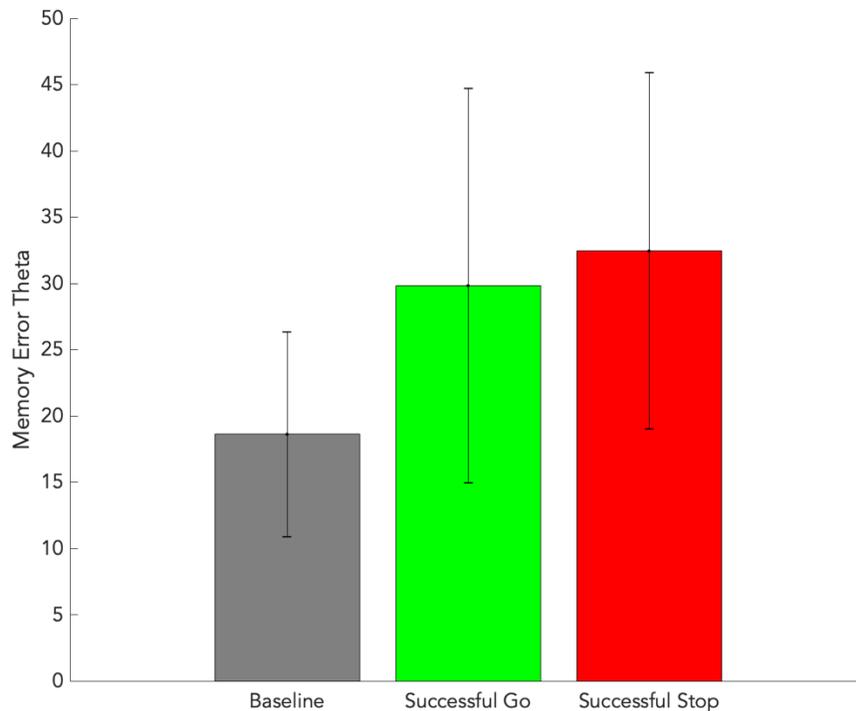
This study included 19 young, healthy, University of Iowa undergraduate students. One participant was excluded from the data set because they had a 100% successful stop rate indicating a very high likelihood of proactive inhibition. Of the remaining 18 participants we gathered the following demographic information: 2 male, 17 female; 17 right handed, 2 left handed; 2 Asian, 1 Black, 1 half White half Black, 15 white; mean age = 19.11, STD = 1.7. Participants were compensated with class completion credit via the University of Iowa's online SONA recruitment website.

Experimental design was exactly the same as experiment 1, except the go to nogo-trial ratio was flipped so that we had 1/3rd go-trials and 2/3rd nogo-trials.

Experiment 2 Results

Overall memory thetas were calculated for baseline trials ($\theta = 18.62$, STD = 7.92), successful go-trials ($\theta = 29.56$, STD = 15.26) and successful stop trials ($\theta = 32.59$, STD = 13.8). A one-way ANOVA revealed a significant difference between baseline, successful go and successful nogo group means ($F(3/51) = 8.6388$, $p < 0.001$). T-tests were used to compare successful go vs. baseline ($p < 0.001$, $t = 1.74$) and successful nogo vs. baseline ($p < 0.001$). T-

tests were then used to compare all individual participant successful go-trials and successful stop trials to reveal no significant effect ($p = 0.11$).



Discussion

In line with our hypothesis, we found that stopping an action lead to a less accurate recollection of a visual stimulus that required a stop compared to trials that required a response. There are still a few explanations that could be causing this effect. Our theory is that motor inhibition recruits the fronto-basal ganglia structure which causes a global inhibition process (Wessel & Aron, 2017) that extends to cognitive processes, disrupting visual working memory representations. Another explanation is that working memory and motor inhibition draw upon a shared resource causing a processing bottleneck (Chiu & Egner, 2017). In order to see if this is the case, we would need to modify our experiment such that the go/stop signal is presented separately from the stimulus to-be-remembered. In a follow up experiment, we could separate the two by first changing the target stimulus into a constant, memory response color, and then changing it into the go/stop signal. By delaying the timings between visual memory encoding and the response signal, we would be able to see if working memory and motor inhibition are

drawing upon the same resources. If this is the case, then we should see no effect of memory accuracy between successful go trials and successful stop trials. However, if these two processes are not drawing upon a shared resource, then we should still see the same effect.

We also found that compared to trials where participants received a go/nogo stimulus, baseline trials were significantly more accurate. Our theory for this difference is that there is a processing bottleneck for resources when participants need to react to a stimulus with an action. This means that on baseline trials, trials where participants don't need to react to a go/nogo stimulus, there are more cognitive resources available for working memory maintenance.

It is important for us to discuss the results of experiment 2 because although nogo-trials were still the least accurate, our effect between successful go trials and successful stop trials was no longer significant. This means that working memory disruption was being caused, at least partially, by infrequent stimuli. That being said, there is a concern with experiment 2 that because making a response is so rare, participants might be “autopiloting” their responses. This “autopilot mode” is known as proactive inhibition, which means that people proactively plan *not* to respond *before* the stimulus comes. Evidence of proactive inhibition in our task can be interpreted by successful go reaction times. Experiment 1 successful go-trial reaction times (RT = 494.31, STD = 7.25) were faster than experiment 2 successful go-trial reaction times (RT = 515.74, STD = 11.84); however, the difference was not significant ($p = 0.079$). In order to detect proactive inhibition, we should run a follow up EEG study to see if nogo-trials are still recruiting the fronto-basal ganglia. We know that stopping recruits this system via a P3 component signature. If we do not see evidence of said component on nogo-trials, then we know that the participant is mostly likely proactively inhibiting their responses, which would open the door again to the possibility that our original hypothesis is true; fronto-basal ganglionic recruitment via motor inhibition extends beyond the motor system and into cognitive representations. However, if we do still see a P3 signature on nogo-trials, signifying no proactive inhibition, then we know that we are testing our hypothesis correctly. If we do not see a P3 signature on nogo-trials then participants are likely relying on proactive inhibition.

Because our results from experiment 2 were almost significant ($p = 0.11$), it is also necessary to check to make sure that our experiment has enough power to detect the effect we're looking for. Effect size was calculated from experiment 1 ($d = 0.4496$, $M_1 = 19.4$, $M_2 = 21.94$, $SD_{\text{pooled}} = 5.6436$). We then measured, post-hoc, the achieved power from experiment 2 ($n = 18$,

$d = 0.4496$, $\alpha = 0.05$, $\beta = 0.574$) which revealed that we had a 43% chance of making a type II error. In order to achieve sufficient power ($\beta = 0.8$), we performed an a priori calculation to find the necessary sample size to detect the effect we're looking for. We calculated that 41 participants will give us sufficient power to either reject or accept the null hypothesis. Therefore, it is most likely necessary to repeat this study with a higher sample size to achieve sufficient statistical power.

Summary

In summary, the circle task has provided us with valuable insight on the influential nature that motor inhibition may have on cognitive processes; however, there are many things to explore before we can confirm our hypothesis. Regardless, we walk away from these first two experiments with a few key takeaways. We know that motor inhibition is, at least partially, disrupting visual working memory because of the significant effect between successful go/nogo trials in experiment 1. We say “partially” because reversing the go/nogo ratio in experiment 2, in order to determine if stimulus frequency played a factor in disruption, seemed to void the significant difference between successful go/nogo trials. Future experiments should investigate known neural signatures, such as P3 component onset, to confirm motor inhibition correlations between successful go/nogo trials and response thetas.

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