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Balancing Cognitive Demands: Control Adjustments in the Stop-Signal Paradigm

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Cognitive control enables flexible interaction with a dynamic environment. In 2 experiments, the authors investigated control adjustments in the stop-signal paradigm, a procedure that requires balancing speed (going) and caution (stopping) in a dual-task environment. Focusing on the slowing of go reaction times after stop signals, the authors tested 5 competing hypotheses for post-stop-signal adjustments: goal priority, error detection, conflict monitoring, surprise, and memory. Reaction times increased after both successful and failed inhibition, consistent with the goal priority hypothesis and inconsistent with the error detection and conflict hypotheses. Post-stop-signal slowing was greater if the go task stimulus repeated on consecutive trials, suggesting a contribution of memory. We also found evidence for slowing based on more than the immediately preceding stop signal. Post-stop-signal slowing was greater when stop signals occurred more frequently (Experiment 1), inconsistent with the surprise hypothesis, and when inhibition failed more frequently (Experiment 2). This suggests that more global manipulations encompassing many trials affect post-stop-signal adjustments.

Keywords: post-stop-signal slowing, proactive control, cognitive control, inhibition, stop-signal paradigm

Cognitive control, the ability to adapt mental processes to the demands of the task environment, is of central importance to goal-directed behavior (Logan, 1985; Miyake et al., 2000). Cognitive control often involves resolving competing demands. Many instances of control involve achieving a balance between going and stopping, speed and caution. For example, driving involves balancing when to accelerate and when to brake. In two experiments, we investigated the adjustments subjects made to balance speed and caution in the stop-signal paradigm (Logan & Cowan, 1984), which directly pits going against stopping. Previous research has shown that reaction time (RT) increases after both successful and failed inhibition (Rieger & Gauggel, 1999; Verbruggen, Logan, Liefooghe, & Vandierendonck, 2008). The first motivation for this research was to evaluate five competing hypotheses for these post-stop-signal adjustments: goal priority, error detection, response conflict, surprise, and memory. There is also evidence that subjects make adjustments proactively, increasing RT in anticipation of the need to stop (Logan, 1981; Logan & Burkell, 1986; Verbruggen & Logan, 2009b). The second motivation for this research was to investigate whether proactive increases in RT can be explained by the accumulation of post-stop-signal slowing.

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Stop-Signal Paradigm

The stop-signal paradigm is a common procedure for investigating response inhibition (Logan, 1994; Logan & Cowan, 1984; Verbruggen & Logan, 2008c). In the stop-signal paradigm, subjects typically perform a choice RT task (the "go" task), and they are asked to withhold their response when a stop signal occurs on a random subset of trials. The delay between the presentation of the go stimulus and the stop signal (stop-signal delay, or SSD) is varied to manipulate the probability of inhibition. When SSD is short, subjects usually inhibit their responses. As SSD increases, the probability of inhibition decreases. These findings have been explained with the "horse race" model (Logan & Cowan, 1984), which assumes that a go process, initiated by go stimulus onset, races against a stop process, initiated by stop-signal onset. If the go process finishes before the stop process, subjects fail to inhibit, producing a *signal-respond* trial. If the stop process finishes before the go process, subjects succeed at inhibiting, producing a *signal-inhibit* trial. Stop-signal delay biases the race between stop and go processes: Short SSDs bias the race in favor of stopping and thereby increase the probability of inhibiting; long SSDs bias the race in favor of going and thereby increase the probability of responding.

Post-Stop-Signal Slowing

Subjects can also bias the race between stopping and going by strategically speeding or slowing go RT. Speeding go RT decreases the probability of inhibition, whereas slowing go RT increases it. These adjustments can be triggered by local events in the experiment, such as the presentation of a stop signal or a cue indicating that stop signals are likely, or by global factors, such as the percentage of trials on which stop signals are presented. One category of adjustments in the stop-signal paradigm is post-stop-

signal slowing: RT often increases on trials following a stop signal (Rieger & Gauggel, 1999; Verbruggen et al., 2008). Some research has found that slowing occurs after both successful and failed inhibition (Rieger & Gauggel, 1999), some research has found greater slowing after successful inhibition (Emeric et al., 2007; Verbruggen et al., 2008), and some research has found greater slowing after failed inhibition (Schachar et al., 2004; Verbruggen et al., 2008). A major goal of the present research is to determine the conditions under which post-stop-signal slowing is triggered. Different hypotheses predict that different events will trigger post-stop-signal slowing, and our goal was to distinguish between the hypotheses.

Proactive Adjustment or Accumulation of Post-Stop-Signal Adjustments?

A second category of adjustments in the stop-signal paradigm is proactive slowing of go RT in anticipation of stop signals. Verbruggen and Logan (2009b) found strong evidence for proactive adjustment by presenting cues that indicated whether stop signals would be relevant for the next few trials. They found slowing on the first trial following the cue, before any stop signals were presented. Several investigators have found that go RTs are longer when stop signals occur more frequently (Emeric et al., 2007; Logan, 1981; Logan & Burkell, 1986), which they interpreted as proactive slowing in anticipation of stop signals. An alternative hypothesis is that the slowing results from accumulated post-stop-signal slowing. The more frequently stop signals occur, the more instances there are of post-stop-signal slowing, and these instances could aggregate to produce longer go RTs. A second goal of the present research was to distinguish between these hypotheses by evaluating how quickly go RT returns to baseline after a stop-signal trial.

Hypotheses for Post-Stop-Signal Adjustments

Our goal of determining the conditions under which post-stop-signal slowing is triggered is important because it allows us to distinguish between five hypotheses that have been proposed to account for post-stop-signal adjustments. Each hypothesis predicts that post-stop-signal slowing is triggered by a particular kind of event on a stop-signal trial: the mere occurrence of a stop signal, failed inhibition, successful inhibition, the occurrence of a rare event, or the occurrence of a stimulus associated with inhibition. The five hypotheses link these events and the adjustments in response to them to broader goals of the executive control system, such as balancing competing demands and adapting to dynamically changing environments.

The *goal priority hypothesis* (Leotti & Wager, 2010; Liddle et al., 2009) suggests that subjects shift their priority to the stop task after stop signals. Performance on the stop task requires caution, and performance on the go task requires speed. The occurrence of a stop signal indicates the need for caution, so subjects increase RT. The goal priority hypothesis predicts post-stop-signal slowing after all stop-signal trials (both signal-respond and signal-inhibit trials; see Figure 1a).

The *error detection hypothesis* (Laming, 1968; Rabbitt, 1966) suggests that subjects adjust their behavior in response to errors. When applied to the stop-signal paradigm (Schachar et al., 2004; Verbruggen et al., 2008), the error detection hypothesis claims that subjects slow go RT when they fail to inhibit, in order to reduce the probability of stopping errors in the future. The error is the event that signals adjustment, so the error detection hypothesis predicts slowing after failed inhibition (signal-respond trials) but not after successful inhibition (signal-inhibit trials; see Figure 1b).

The *response conflict hypothesis* (Botvinick, Braver, Barch, Carter, & Cohen, 2001) suggests that the recruitment of control processes occurs as a result of conflict, or coactivation of competing responses. This hypothesis has been applied to a variety of cognitive tasks (Botvinick et al., 2001). Schall and colleagues have applied it to the stop-signal paradigm (Ito, Stuphorn, Brown, & Schall 2003; Stuphorn & Schall, 2006; Stuphorn, Taylor, & Schall, 2000), arguing that the greatest coactivation of competing responses occurs on signal-inhibit trials, so slowing should be greater after signal-inhibit trials than after signal-respond trials (see Figure 1c). We evaluate this specific prediction and consider alternative interpretations of the conflict hypothesis in the General Discussion.

The *surprise hypothesis* predicts slowing after rare events, which elicit an orienting response that competes with normal processing (Notebaert et al., 2009; Núñez Castellar, Kühn, Fias, & Notebaert, 2010). When applied to the stop-signal paradigm, the surprise hypothesis predicts slowing after all stop signal trials (signal-respond and signal-inhibit) because stop signals occur on a minority of trials. In this respect, its predictions are similar to the goal priority hypothesis. However, this hypothesis also predicts greater slowing when stop signals are less likely, so it can be distinguished from the goal priority hypothesis by manipulating the percentage of stop signals, as we do in Experiment 1 (see Figure 1d).

The *memory hypothesis* proposes that stimuli that occur on stop trials become associated with stopping, and the association is stronger when inhibition is successful (signal-inhibit trials; Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008). If the stimuli repeat, they retrieve associations with stopping, and that slows RT. If the stimuli do not repeat, they do not retrieve associations with stopping to slow RT. Thus, the memory hypothesis predicts greater post-stop-signal slowing when stimuli repeat after stop trials than when stimuli do not repeat, and this difference should be larger after successful inhibition than after unsuccessful inhibition. In principle, the responses that were supposed to occur on stop trials could become associated with stopping as well, so the memory hypothesis is consistent with slower RT following stop trials when the response repeats. In practice, associations between stimuli and stopping are stronger than associations between responses and stopping; post-stop-signal slowing is greater for stimulus repetitions than for response repetitions (Verbruggen et al., 2008). In the present experiments, we distinguish the memory hypothesis from the other four hypotheses by separating stimulus repetitions, response repetitions, and response alternations. The memory hypothesis predicts greater slowing with stimulus repetitions, and possibly after response repetitions (see Figure 1e). The other hypotheses do not make any predictions about the effects of stimulus repetition. The other hypotheses can be distinguished from each other by examining trials in which stimuli do not repeat.

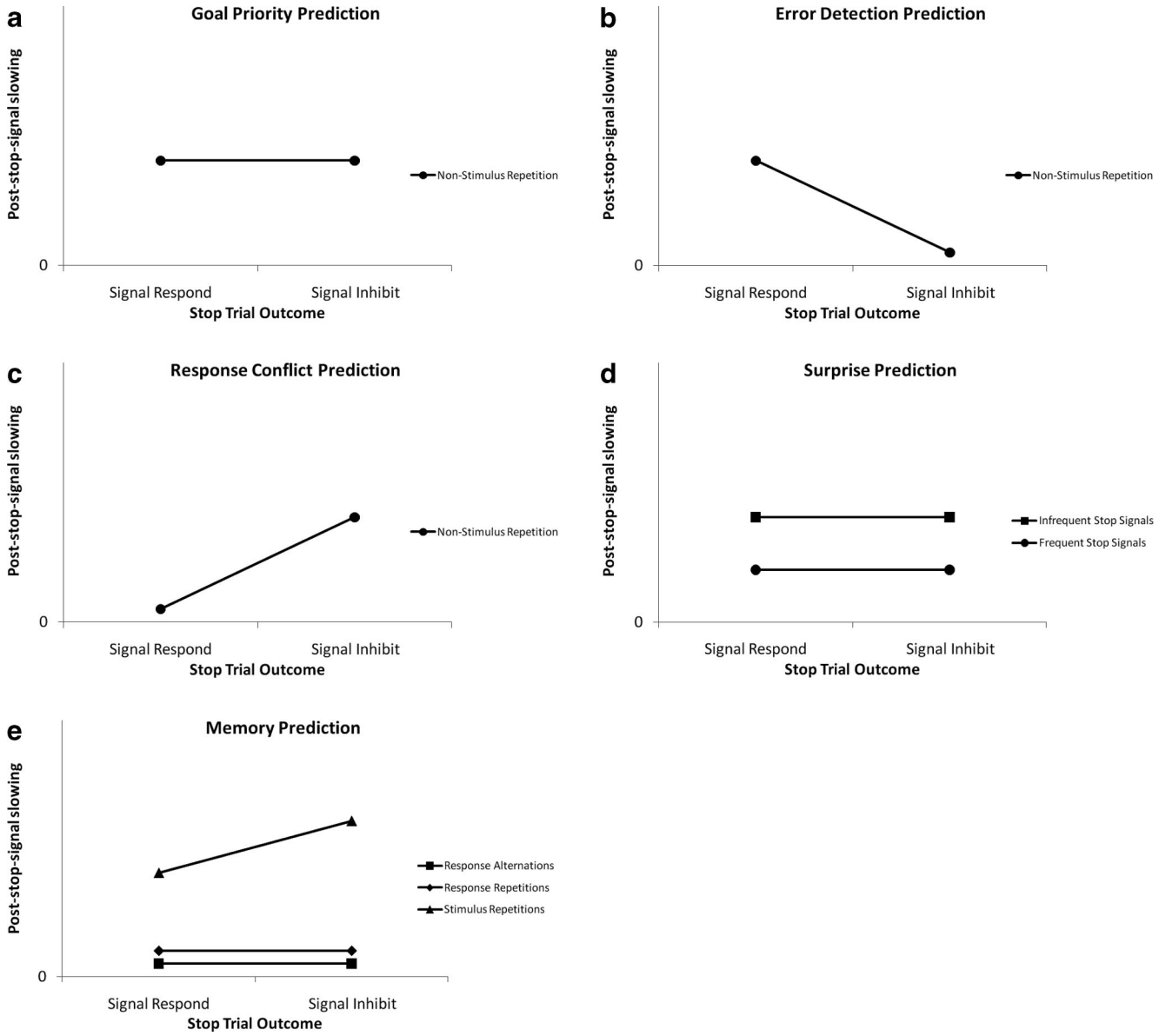

Figure 1. The predictions made by the five hypotheses for post-stop-signal adjustments. Nonstimulus repetitions are trial sequences in which Trial N and Trial $N + 1$ have different go stimuli; stimulus repetitions are instances in which Trial N and Trial $N + 1$ have the same go stimulus; response repetitions are instances in which Trial N and Trial $N + 1$ have the same correct response but a different go stimulus; and response alternations are instances in which the stimulus and the response change from Trial N to Trial $N + 1$.

These hypotheses are logical alternatives, but they are not necessarily mutually exclusive. Some combinations of hypotheses are logically possible and may make similar predictions in some circumstances. Our experiments were designed to allow us to discriminate between hypotheses and between some combinations of hypotheses.

The Present Experiments

Our first goal was to test the five hypotheses for post-stop-signal slowing. Both Experiment 1 and Experiment 2 were designed to address this first goal. Our second goal was to test hypotheses about proactive adjustments in the stop-signal paradigm. In Experiment 1, we manipulated the percentage of stop-signal trials, presenting stop signals on 40% versus 20% of all trials. This allowed us to determine whether slowing when stop signals are more frequent is due to anticipatory proactive adjustments or the accumulation of post-stop-signal adjustments. In Experiment 2, we manipulated the probability of successful inhibition, holding the percentage of stop signals constant, to determine whether stop-signal frequency or probability of success affects post-stop-signal slowing.

Experiment 1

In Experiment 1, subjects made a manual response to one of four shapes in the go task (circle, square, diamond, triangle) and attempted to stop when they heard a stop signal. Subjects completed two sessions: one session in which 40% of trials included a stop signal and another session in which 20% of trials included a stop signal. Mapping four go stimuli onto two responses allowed us to distinguish *stimulus repetition* trials, in which the stimulus and response repeated (e.g., circle→circle), *response repetition* trials, in which the stimulus changed but the response repeated (e.g., square→circle if square and circle were mapped onto the same response), and *response alternation* trials, in which both the stimulus and the response changed (e.g., diamond→circle if diamond and circle were mapped onto different responses). We evaluated the memory hypothesis by comparing stimulus repetition trials and response repetition trials with response alternation trials. We evaluated the goal priority, error detection, conflict, and surprise hypotheses on response repetition and response alternation trials. In both the 40% and 20% conditions, the goal priority hypothesis predicts slowing after both signal-respond and signal-inhibit trials (Figure 1a), the error detection hypothesis predicts slowing only after signal-respond trials (Figure 1b), the conflict hypothesis predicts slowing only after signal-inhibit trials (Figure 1c), and the surprise hypothesis predicts slowing after both signal-inhibit and signal-respond trials. The surprise hypothesis predicts greater slowing in the 20% condition than in the 40% condition (Figure 1d). The memory hypothesis predicts greater slowing after stimulus repetitions, especially after signal-inhibit trials, in both the 40% and 20% conditions, and could accommodate greater slowing after response repetitions than response alternations (Figure 1e).

Manipulating the percentage of stop-signal trials allowed us to determine why mean go RT increases with more frequent stop signals (Emeric et al., 2007; Logan, 1981; Logan & Burkell, 1986). The increase in RT may reflect proactive adjustment in anticipation of frequent stop signals, or it may reflect the accumulation of post-stop-signal slowing. To evaluate these hypotheses, we assessed how quickly RT returned to baseline after a stop signal. If RT returns to baseline quickly, we can rule out accumulated post-stop-signal slowing and support proactive slowing. If RT returns to baseline slowly, then accumulated post-stop-signal slowing is a viable explanation of slower go RT with more frequent stop signals.

Method

Subjects. Twenty-four subjects were recruited from the Nashville area and were compensated $24 for two separate 1-hr sessions on consecutive days. All subjects had normal or corrected-to-normal vision, and all were naive as to the purpose of the experiment. Subjects did not participate in Experiment 2 or any similar studies during the previous two months. We replaced two subjects whose probabilities of successful stopping fell outside the 95% confidence interval of .5 probability of stopping to a stop signal.

Apparatus and stimuli. The experiment was run on a Pentium Dual-Core PC running E-Prime 1 (Psychology Software Tools; www.pstnet.com). The stimuli were presented on a 19-in. (48.3-cm) cathode ray tube monitor. The go task was to respond to a single black shape on a white background presented in the center of the screen. The shape was chosen from a set of four shapes: triangle, circle, square, or diamond. The height of the triangle was 4 cm, the diameter of the circle was 4 cm, each side of the square was 4 cm, and the diamond was 4 cm from point to point. Subjects responded by pressing the "1" or "0" key on the top row of a QWERTY keyboard with the left or right index finger, respectively. The stop signal was an auditory tone (70 dB, 100 ms, 500 Hz) presented through closed headphones (Sennheiser eH 150).

Procedure. Each trial began with a 500-ms fixation display, followed by a single black shape chosen randomly from a set of four shapes. Subjects were instructed to respond as quickly and accurately as possible to the shape according to a specific response mapping. Two of the shapes were mapped onto the keyboard number "1," and two of the shapes were mapped onto the keyboard number "0," and the response mapping was counterbalanced across subjects. The shape stimulus remained on the screen for 850 ms. In order to discourage a strategy of waiting for stop signals to occur, the experimenter instructed subjects to respond while the shape was displayed on the screen. The shape was followed by a 1,000-ms intertrial interval (ITI) during which the screen was left blank.

Between sessions, we manipulated the percentage of trials that included a stop signal. In one session, stop signals occurred on 40% of trials; in the other session, stop signals occurred on 20% of trials. The order of stop-signal percentages was counterbalanced across subjects. The stop signal indicated to subjects that they should withhold their response for that trial. The stop signal was initially presented 250 ms after the presentation of the shape (i.e., SSD = 250 ms). The SSD was continually adjusted according to a tracking procedure, increasing by 50 ms after each signal-inhibit trial and decreasing by 50 ms after each signal-respond trial. This tracking procedure yields a probability of .5 of responding to a stop signal (Levitt, 1971). If the probability of responding is .5, the race between going and stopping is tied, and the stop-signal RT (SSRT) can be estimated by subtracting mean SSD from mean go RT (Logan, Schachar, & Tannock, 1997).

The experiment began with written and verbal instructions. Subjects were instructed to respond quickly and accurately to the shape and to do their best to withhold their response when stop signals occurred. Subjects were instructed not to wait for the stop signal. After the instructions, subjects were given 24 trials of experimenter-supervised practice. At the completion of practice, subjects were given feedback on speed and accuracy. If subjects did not respond with accuracy above 75% and RT below 1,000 ms, the practice was repeated until these thresholds were met. After practice, subjects completed the main task, which included five blocks of 240 trials. At the end of each block, subjects were given feedback on RT and accuracy, as well as the percentage of trials on which they responded while the shape remained on the screen.

Results and Discussion

Mean RTs and accuracies from no-stop-signal trials were calculated for each cell of a 2 (pre or post: Trial N − 1 or Trial N + 1) × 2 (percentage of stop signals: 40% or 20%) × 3 (Trial N: go, signal-respond, or signal-inhibit) × 3 (repetition: response alternation, response repetition, or stimulus repetition) experimental

design (we use N when the trial of interest is either a go or a stop signal, S when the trial is a stop trial, and G when the trial is a go signal). Repeated measures analyses of variance (ANOVAs) were conducted on the mean go RTs and accuracies with this design. Accuracy results did not contradict RTs, and accuracies were high, so we focused on RTs. Summary tables for these ANOVAs appear in Table 1. Mean go RTs and accuracies across subjects appear in Table 2. We included go RTs from correct trials that were shorter than 1,850 ms (shape presentation time plus ITI). To increase the number of observations, we included S − 1 trials that preceded multiple stop signals and S + 1 trials that followed multiple stop signals if the multiple stop signals all had the same outcome (signal-respond or signal-inhibit). To ensure that this inclusion did not affect our conclusions, we compared the ANOVA in Table 1 with a matching ANOVA including trials with only one intervening stop signal between Trials S − 1 and S + 1. The means in the two ANOVA designs were very similar ($r = .95$, $p < .01$), and the ANOVAs led to the same conclusions. Our interpretations of the data are based primarily on planned comparisons instead of ANOVA factors. We conducted the ANOVAs to provide error terms for the planned comparisons.

In addition to traditional null hypothesis significance testing, we computed Bayes factors (*B*s) for our contrasts to compare the likelihood of the data under the null hypothesis of no difference with the likelihood of the data under the alternative hypothesis of some difference. This allowed us to quantify the extent to which the data support the null hypothesis, giving the odds in favor of the null hypothesis (Rouder, Speckman, Sun, Morey, & Iverson, 2009). The maximum possible odds ratio in support of the null hypothesis with 24 subjects is approximately 6.38 (i.e., if the difference is 0), but there is no maximum odds ratio for the alternative hypothesis, because, in principle, the difference is limitless, but if the odds ratio is greater than 10, we present it as >10. In general, odds ratios greater than 3 are considered as substantial support for the favored hypothesis (Rouder et al., 2009). We present Bayes factors as single numbers. Those that occur with significant effects (accompanied by *p* values) should be interpreted as the odds in favor of the alternative hypothesis; those that occur with nonsignificant effects (not accompanied by *p* values) should be interpreted as the odds in favor of the null hypothesis.

Table 2
Experiment 1 Mean Go Reaction Time and Go Accuracy Before (Pre) and After (Post) a Stop Signal

	Go		Signal respond		Signal inhibit	
Measure	Pre	Post	Pre	Post	Pre	Post
40% stop signals						
Response alternations						
RT	516	504	503	522	511	530
ACC	93	94	94	93	92	94
Response repetitions						
RT	511	513	518	539	516	544
ACC	92	90	93	88	93	89
Stimulus repetitions						
RT	515	463	503	511	531	543
ACC	94	96	94	95	92	95
20% stop signals						
Response alternations						
Reaction time	458	461	455	468	466	470
Accuracy	94	94	93	94	93	93
Response repetitions						
Reaction time	458	468	454	477	468	480
Accuracy	93	90	92	87	93	92
Stimulus repetitions						
Reaction time	459	430	458	449	463	466
Accuracy	93	97	94	97	93	98

The percentage of stop signals had no effect on the probability of responding given a stop signal ($M = .50$ for 20% and 40% stop signals), $F(1, 23) < 1$, $B = 5.86$, indicating that the tracking algorithm was successful. Consistent with previous research (Logan, 1981; Logan & Burkell, 1986), there was no significant effect of percentage of stop signals on SSRT (*M*s = 243 and 224

Table 1
Experiment 1 Analysis of Variance Summary Table for Go Reaction Time and Go Accuracy

		Reaction time		Accuracy	
Measure	*df*	*MSE*	*F*	*MSE*	*F*
Pre or post stop signal	1, 23	968	3.93	.001	0.01
Percentage of stop signals	1, 23	62,409	10.37**	.006	0.92
Trial N	2, 46	1,236	21.29**	.004	0.49
Repetition	2, 46	1,823	8.81**	.005	19.20**
Pre or Post × Trial N	2, 46	1,511	12.94**	.004	2.50
Pre or Post × Percentage of Stop Signals	1, 23	366	0.43	.001	1.53
Pre or Post × Repetition	2, 46	2,010	10.19**	.005	15.04**
Percentage of Stop Signals × Trial N	2, 46	612	4.68*	.003	1.37
Percentage of Stop Signals × Repetition	2, 46	714	0.79	.001	0.65
Trial N × Repetition	4, 92	1,221	6.60**	.003	1.96
Pre or Post × Percentage of Stop Signals × Trial N	2, 46	611	7.02**	.002	0.10
Pre or Post × Trial N × Repetition	4, 92	375	6.29**	.002	0.41
Pre or Post × Percentage of Stop Signals × Repetition	2, 46	416	0.03	.002	1.88
Percentage of Stop Signals × Trial N × Repetition	4, 92	1,508	0.04	.002	1.51
Pre or Post × Percentage of Stop Signals × Trial N × Repetition	4, 92	330	1.28	.003	0.88

* $p < .05$. ** $p < .01$.

ms for the 20% and 40% conditions, respectively), $F(1, 23) = 3.07$, $MSE = 1{,}395$, $p < .10$, but the Bayes factor does not suggest substantial support for the null hypothesis ($B = 1.58$).

We conducted planned comparisons to evaluate the effect of stop signals on RT, calculating double difference scores and testing their significance. First, we subtracted RT for go trials preceding stop-signal trials (Trial S − 1) from RT for go trials following stop-signal trials (Trial S + 1) to remove the effect of slow fluctuations in RT over the course of the experiment (Gilden, Thornton, & Mallon, 1995; Wagenmakers, Farrell, & Ratcliff, 2004). Nelson, Boucher, Logan, Palmeri, and Schall (2010) showed that slow fluctuations may contaminate estimates of post-stop-signal slowing. These researchers found that trials preceding signal-inhibit trials tended to be slower than trials preceding signal-respond trials. Ignoring these preceding trials led to an overestimate of post-stop-signal slowing after signal-inhibit trials and an underestimate after signal-respond trials. In order to determine whether our estimates would be affected by slow RT fluctuations, we conducted a 2 (percentage of stop signals: 40% or 20%) × 3 (Trial N: go, signal-respond, or signal-inhibit) × 3 (repetition: response alternation, response repetition, or stimulus repetition) repeated measure ANOVA on Trial N − 1 RT. This ANOVA revealed an effect of Trial N on N − 1 RT, $F(2, 46) = 6.89$, $MSE = 728$, $p < .01$, $B = 2.90$. A planned comparison showed that RT on trials preceding a signal-inhibit trial ($M = 491$ ms) was longer than RT on trials preceding a signal-respond trial ($M = 481$ ms), $F(1, 46) = 10.03$, $MSE = 728$, $p < .01$, $B = 8.81$. This showed that we needed to subtract each condition's N − 1 RT from our estimates of post-stop-signal slowing in order to remove the effects of Trial N − 1. Then we calculated the corresponding difference for go trials preceding (G − 1) and following (G + 1) go trials to control for the effect of repetition on the intervening trial (G in this case; S in the stop-signal case). Finally, we subtracted the (G + 1) − (G − 1) difference from the (S + 1) − (S − 1) difference to isolate the effect of the stop signal. The double difference scores measuring post-stop-signal slowing are presented in Figure 2. Post-stop-signal slowing would be evident as a positive double difference score, which is what we found ($M = 24$ ms), $F(1, 46) = 27.42$, $MSE = 1{,}511$, $p < .01$, $B > 10$.

To evaluate goal priority, error detection, conflict, and surprise explanations of post-stop-signal slowing, we focused on response repetition and response alternation trials, as these hypotheses do not make predictions about the effects of stimulus repetition. We found significant slowing ($M = 17$ ms) collapsed across response repetitions and response alternations, $F(1, 92) = 18.84$, $MSE = 375$, $p < .01$, $B > 10$.

Post-stop-signal slowing was similar for signal-respond trials ($M = 19$ ms) and signal-inhibit trials ($M = 15$ ms), $F(1, 92) < 1$, $MSE = 375$, $B = 4.48$. This result supports the goal priority hypothesis, the surprise hypothesis, or a combination of the error detection and conflict hypotheses. The Bayes factor analysis suggests that it provides substantial evidence against error detection and conflict monitoring alone.

The surprise hypothesis predicts greater slowing in the 20% stop-signals condition than in the 40% stop-signals condition (Notebaert et al., 2009; Núñez Castellar et al., 2010). The results revealed the opposite effect. There was greater slowing in the 40% condition ($M = 29$ ms) than in the 20% condition ($M = 6$ ms), $F(1, 92) = 19.39$, $MSE = 330$, $p < .01$, $B > 10$, disconfirming the surprise hypothesis.

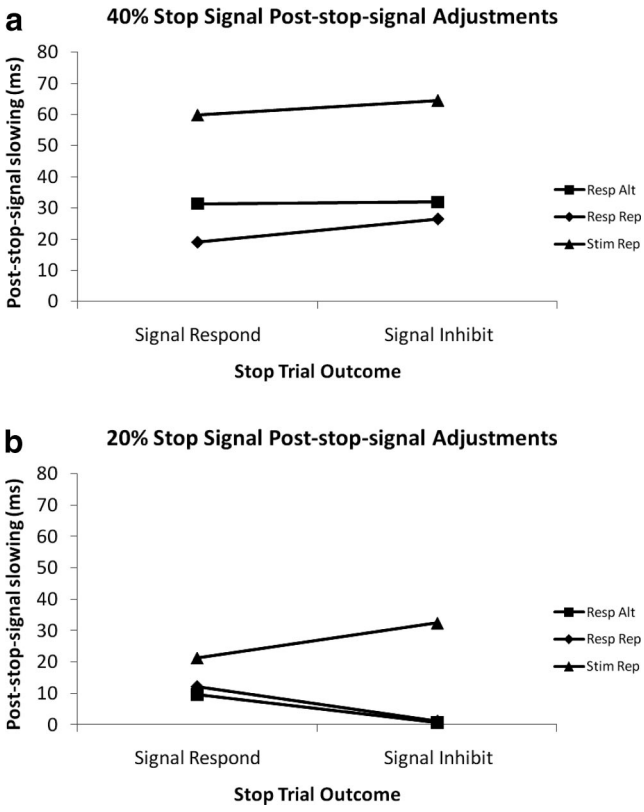

Figure 2. Experiment 1 post-stop-signal adjustment calculated as a double difference score [(S + 1) − (S − 1)] − [(G + 1) − (G − 1)] for 40% (Figure 2a) and 20% (Figure 2b) stop signal sessions. Stimulus repetitions (Stim Rep) are instances in which Trial N and Trial $N + 1$ have the same go stimulus; response repetitions (Resp Rep) are instances in which Trial N and Trial $N + 1$ have the same correct response but a different go stimulus; and response alternations (Resp Alt) are instances in which the stimulus and the response change from Trial N to Trial $N + 1$.

To evaluate the conflict hypothesis further, we compared slowing after signal-inhibit trials with long and short SSDs. Conflict occurs when there is strong concurrent activation of response processes (Botvinick et al., 2001). Stop process activation must be strong on signal-inhibit trials, because the stop process wins the race. Go process activation should be higher when SSD is long because the go process has more time to accumulate activation than when SSD is short. Thus, conflict should be greater for long-SSD signal-inhibit trials than for short-SSD signal-inhibit trials (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000). To test this, we compared signal-inhibit trials with SSDs above and below the median SSD and found no difference in post-stop-signal slowing (14 ms and 12 ms for long and short SSDs, respectively), $F(1, 92) < 1$, $MSE = 375$, $B = 5.93$. This constitutes strong evidence against the conflict hypothesis, and consequently against the combination of the conflict and error detection hypotheses, as an explanation of equal slowing after signal-inhibit and signal-respond trials. Post-stop-signal slowing is best explained by the goal priority hypothesis, as stop signals

appear to shift priority to caution and the stop task independently of success at stopping.

The memory hypothesis predicts substantial slowing when the go stimulus repeats, especially after signal-inhibit trials (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008) and could accommodate greater slowing after response repetitions than after response alternations. We found greater slowing after stimulus repetitions (M = 44 ms) than after the combination of response repetitions and response alternations (M = 17 ms), $F(1, 92)$ = 47.76, MSE = 375, $p < .01$, $B > 10$. There is weak evidence that slowing was greater after stimulus repetition when subjects successfully inhibited (M = 48 ms) than when they failed to inhibit (M = 40 ms) on Trial N, $F(1, 92)$ = 4.06, MSE = 375, $p < .05$, B = .96, consistent with previous research (Verbruggen et al., 2008). There was greater slowing when stimuli repeated in the 40% condition (M = 62 ms) than in the 20% condition (M = 27 ms), $F(1, 92)$ = 45.41, MSE = 330, $p < .01$, $B > 10$. Slowing was the same for response repetitions (M = 15 ms) and response alternations (M = 18 ms), $F(1, 92) < 1.0$, MSE = 375, B = 4.25, suggesting that subjects do not associate responses with stopping. These results suggest that subjects associate stimuli with stopping, which is consistent with previous evidence for the memory hypothesis (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008).

Mean RT was longer in the 40% condition (M = 514 ms) than in the 20% condition (M = 459 ms), $F(1, 23)$ = 10.37 MSE = 62,409, $p < .01$, B = 9.86, which is consistent with proactive slowing (Logan, 1981; Logan & Burkell, 1986; Verbruggen & Logan, 2009b) or the accumulated effects of post-stop-signal slowing. To distinguish between these interpretations, we compared the rate at which RT returned to baseline after a stop signal. We compared RT on the second trial after a stop signal (S + 2) with RT on the trial before (S − 1) and found no residual slowing in Trial S + 2. Trial S + 2 RT was slightly faster than S − 1 in the 40% condition (Ms = 504 ms and 510 ms for S + 2 and S − 1, respectively), $F(1, 23)$ = 6.88, MSE = 64.41, $p < .05$, B = 2.89, and there was no difference in the 20% condition (Ms = 456 ms and 459 ms for S + 2 and S − 1, respectively), $F(1, 23)$ = 1.73, MSE = 46.97, B = 2.84. However, Trial S + 2 is always preceded by a go Trial S + 1, which may result in speeding on Trial S + 2. So we analyzed sequences in which Trial S − 2 was also a go trial, compared Trial S + 2 RT with Trial S − 1 RT, and found no differences in the 40% condition (S + 2: 499 ms, S − 1: 498 ms), $F(1, 23) < 1$, MSE = 135.19, B = 5.33, or the 20% condition (N + 2: 457 ms, N − 1: 456 ms), $F(1, 23) < 1$, MSE = 54.03, B = 4.58. As a further test of the hypothesis that longer RTs in the 40% condition were not due to accumulated post-stop-signal slowing, we calculated mean RT excluding S + 1 trials and found that RT in the 40% condition (M = 501 ms) remained longer than in the 20% condition (M = 456 ms), $F(1, 23)$ = 8.40, MSE = 2,834.26, $p < .01$, B = 5.02. This suggests that accumulated post-stop-signal slowing cannot account for the increase in RT with stop-signal frequency and that proactive adjustments play a key role in the effect.

Conclusions

The results for post-stop-signal support the goal priority hypothesis (Leotti & Wager, 2010; Liddle et al., 2009), and provide evidence against the error detection (Laming, 1968; Rabbitt, 1966), conflict monitoring (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000), and surprise (Notebaert et al., 2009; Núñez Castellar et al., 2010) hypotheses. The results for stimulus repetition trials support the memory hypothesis (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008), but there was no support for responses becoming associated with stopping.

The finding that post-stop-signal slowing in the 40% condition was larger than that in the 20% conditions suggests that global, experiment-wide manipulations can influence the slowing after a stop signal. The hypotheses for post-stop-signal adjustments generally consider only the immediately preceding trial, suggesting a local explanation for slowing. Our results suggest that post-stop-signal slowing is affected by global factors.

Mean RT was longer in the 40% stop signal condition than in the 20% condition. The results suggest that the accumulation of post-stop-signal slowing cannot account for this effect and that proactive adjustments occur in anticipation of stop signals (Logan, 1981; Logan & Burkell, 1986; Verbruggen & Logan, 2009b).

Experiment 2

In Experiment 1, we found more post-stop-signal slowing when stop signals were frequent than when they were infrequent. Our results suggest that global manipulations encompassing many trials affected post-stop-signal adjustments. It is not clear whether this effect was due to the prevalence of stop-signal trials or to the prevalence of failed (or successful) inhibition. To distinguish between these interpretations, in Experiment 2 we manipulated the probability of successful inhibition while holding the percentage of stop signals constant. We used two different tracking procedures for setting SSD that were designed to produce successful inhibition on 30% and 70% of the trials. In a third condition, we intermixed the two tracking procedures to produce an overall success rate of 50%. If post-stop-signal slowing depends on the probability of a stop signal, there should be no differences among the three conditions. If post-stop-signal slowing depends on the probability of successful (or failed) inhibition, then we should see large differences between the three conditions.

Method

Subjects. Twenty-four subjects were recruited from the Nashville area and were compensated $36 for three separate 1-hr sessions on three consecutive days. All subjects had normal or corrected-to-normal vision, and all were naive as to the purpose of the experiment. Subjects did not participate in Experiment 1 or any similar studies during the previous two months. We replaced seven subjects whose probabilities of successful stopping fell outside the 95% confidence interval of the expected probability of stopping in any of the three conditions.

Apparatus and stimuli. The apparatus and stimuli were the same as in Experiment 1.

Design and procedure. The basic trial structure and instructions for Experiment 2 were identical to those of Experiment 1 with the following exceptions: The probability of a stop signal was held constant at .33. We manipulated the tracking algorithm to produce different probabilities of successful inhibition. There were three different conditions involving different tracking procedures

run in different sessions, and the order of tracking conditions was counterbalanced across subjects. In the high-SSD condition, SSD increased by 50 ms each time subjects successfully inhibited their response (signal-inhibit) but decreased by 50 ms when subjects failed to inhibit (signal-respond) on two consecutive stop-signal trials. This tracking procedure should yield a .707 probability of responding to the stop signal (Levitt, 1971). In the low-SSD condition, SSD increased by 50 ms when subjects successfully inhibited their response on two consecutive stop-signal trials but decreased by 50 ms each time subjects failed to inhibit. This tracking procedure should yield a .293 probability of responding to a stop signal (Levitt, 1971). In the mixed-SSD condition, the high- and low-SSD tracking procedures were interleaved within the session. The mixed condition should yield an average probability of responding to the stop signal of 0.5. Because the different tracking procedures yield probabilities of responding that are different from 0.5, we used the integration method to calculate SSRT (Logan & Cowan, 1984).

Results and Discussion

Mean go RTs and accuracies from no-stop-signal trials were calculated for each cell of a 2 (pre or post: Trial N − 1 or Trial N + 1) × 3 (tracking condition: high, mixed, or low) × 3 (Trial N: go, signal-respond, or signal-inhibit) × 3 (Repetition: response alternation, response repetition, or stimulus repetition) experimental design. Summary tables for repeated measures ANOVAs on go RTs and accuracies based on this design are presented in Table 3. Accuracy results did not contradict RTs, and accuracies were high, so we focused on RTs. Mean go RTs and accuracies across subjects appear in Table 4, and post-stop-signal slowing double difference scores are depicted in Figure 3. The inclusion criteria were the same as in Experiment 1. Our interpretations of the data are based primarily on planned comparisons instead of ANOVA factors. We conducted the ANOVAs to provide error terms for the planned comparisons. We also included Bayes factors to quantify the odds in favor of the null or alternative hypothesis, as in Experiment 1.

The main ANOVA presented in Table 3 collapses across the high and low tracking procedures in the mixed condition. This was necessary because we could not separate go Trial N in high- and low-tracking procedures. To test the effect of tracking procedure on post-stop-signal effects in the mixed condition, we conducted a separate 2 (pre or post: Trial S − 1 or Trial S + 1) × 2 (tracking procedure: high or low) × 2 (Trial S: signal-respond or signal-inhibit) × 3 (repetition: stimulus repetition, response repetition, or response alternation) repeated measures ANOVA on go RTs. We found an interaction of Pre or Post × Tracking, $F(1, 23) = 6.41$, $MSE = 221.86$, $p < .05$, $B = 2.43$, but no main effect of tracking or other interactions of tracking with post-stop-signal effects (all ps $> .15$). This significant effect is driven by larger post-stop-signal slowing in the mixed-low condition ($M = 20$ ms) than in the mixed-high condition ($M = 13$ ms). This result does not distinguish post-stop-signal slowing hypotheses, and we do not have a ready explanation, so we do not discuss it further. Complete mixed-condition RT and go accuracy results are presented in Table 4.

As expected, tracking condition had strong effects on the probability of responding given a stop signal, $F(2, 46) = 8{,}477.64$, $MSE = .0001$, $p < .01$. The observed probabilities (Ms = .70, .50, and .31 for high-, mixed-, and low-SSD conditions, respectively) matched the expected probabilities closely, indicating that the tracking procedure worked as we intended (Levitt, 1971). Tracking condition also affected SSRT (Ms = 190 ms, 205 ms, and 236 ms for high-, mixed-, and low-SSD conditions, respectively), $F(2, 46) = 9.97$, $MSE = 1{,}330$, $p < .01$. This is expected from the race model: The stop process should beat the go process more often, the shorter the SSD; therefore, the effective SSRT will include more of the upper tail of the SSRT distribution, the shorter the SSD (Logan & Burkell, 1986).

In order to determine whether there were slow RT fluctuations in these data, we conducted a 3 (tracking condition: high, mixed,

Table 3
Experiment 2 Analysis of Variance Summary Table for Go Reaction Time and Go Accuracy

		Reaction time		Accuracy	
Measure	*df*	*MSE*	*F*	*MSE*	*F*
Pre or post stop signal	1, 23	494	21.76**	.001	5.91*
Tracking condition	2, 46	49,746	2.21	.008	0.52
Trial N	2, 46	1,703	47.31**	.002	4.41*
Repetition	2, 46	1,064	10.89**	.004	15.10**
Pre or Post × Trial N	2, 46	789	57.25**	.002	0.07
Pre or Post × Tracking Condition	2, 46	382	1.59	.001	0.01
Pre or Post × Repetition	2, 46	1,348	18.12**	.004	26.37**
Tracking Condition × Trial N	4, 92	2,871	6.72**	.003	0.55
Tracking Condition × Repetition	4, 92	575	0.55	.002	1.17
Trial N × Repetition	4, 92	492	15.25**	.001	3.56*
Pre or Post × Tracking Condition × Trial N	4, 92	1,058	1.88	.002	0.56
Pre or Post × Trial N × Repetition	4, 92	363	14.28**	.002	5.69**
Pre or Post × Tracking Condition × Repetition	4, 92	565	0.77	.002	1.69
Tracking Condition × Trial N × Repetition	8, 184	697	0.88	.002	1.51
Pre or Post × Tracking Condition × Trial N × Repetition	8, 184	646	0.88	.002	1.01

* $p < .05$. ** $p < .01$.

Table 4
Experiment 2 Mean Go Reaction Time and Go Accuracy Before and After a Stop Signal

	Go		Signal respond		Signal inhibit	
Condition/measure	Pre	Post	Pre	Post	Pre	Post
High-SSD tracking						
Response alternations						
Reaction time	507	498	498	512	523	537
Accuracy	95	94	95	95	96	97
Response repetitions						
Reaction time	504	494	495	534	523	563
Accuracy	94	91	94	90	95	94
Stimulus repetitions						
Reaction time	504	455	501	501	525	554
Accuracy	95	97	95	97	94	95
Mixed-high-SSD tracking						
Response alternations						
Reaction time	488	483	479	484	488	500
Accuracy	95	95	96	94	95	95
Response repetitions						
Reaction time	487	484	473	490	495	524
Accuracy	96	93	95	90	94	95
Stimulus repetitions						
Reaction time	488	446	478	479	483	505
Accuracy	95	98	93	98	95	94
Mixed-low-SSD tracking						
Response alternations						
Reaction time	488	483	472	495	488	501
Accuracy	95	95	96	95	95	97
Response repetitions						
Reaction time	487	484	478	519	486	516
Accuracy	96	93	96	91	96	96
Stimulus repetitions						
Reaction time	488	446	483	481	493	511
Accuracy	95	98	94	96	97	96
Low-SSD tracking						
Response alternations						
Reaction time	487	482	467	485	485	497
Accuracy	95	95	95	95	95	95
Response repetitions						
Reaction time	480	486	475	496	484	497
Accuracy	95	91	95	90	96	92
Stimulus repetitions						
Reaction time	484	440	481	480	490	500
Accuracy	94	97	93	98	95	97

Note. SSD = stop-signal delay.

or low) × 3 (Trial N: go, signal-respond, or signal-inhibit) × 3 (repetition: response alternation, response repetition, or stimulus repetition) repeated measure ANOVA on Trial N − 1 RT. This ANOVA revealed a large effect of trial N on N − 1 RT, $F(2, 46) = 41.34$, $MSE = 508$, $p < .01$. A planned comparison showed that RT on trials preceding a signal-inhibit trial ($M = 499$ ms) was longer than trials preceding a signal-respond trial ($M = 482$ ms), $F(1, 46) = 62.23$, $MSE = 508$, $p < .01$, $B > 10$. As in Experiment 1, this justifies the use of double-difference scores as the measure of post-stop-signal slowing.

Averaged over conditions, there was significant post stop-signal slowing (31 ms), $F(1, 46) = 135.45$, $MSE = 789$, $p < .01$, $B > 10$. To determine whether the magnitude of post-stop-signal slowing depends on the probability of successful stopping, we compared double difference scores in the high-SSD condition (p[inhibit] = .30; slowing = 40 ms) and the low-SSD condition (p[inhibit] = .69; slowing = 24 ms) and found a significant difference overall, $F(1, 92) = 8.43$, $MSE = 1{,}058$, $p < .01$, $B = 5.08$. The difference was significant for stimulus repetitions (64 ms vs. 47 ms, respectively), $F(1, 184) = 5.31$, $MSE = 646$, $p < .05$, $B = 1.58$, and response repetitions (49 ms vs. 11 ms, respectively), $F(1, 184) = 28.03$, $MSE = 646$, $p < .01$, $B > 10$, but not for response

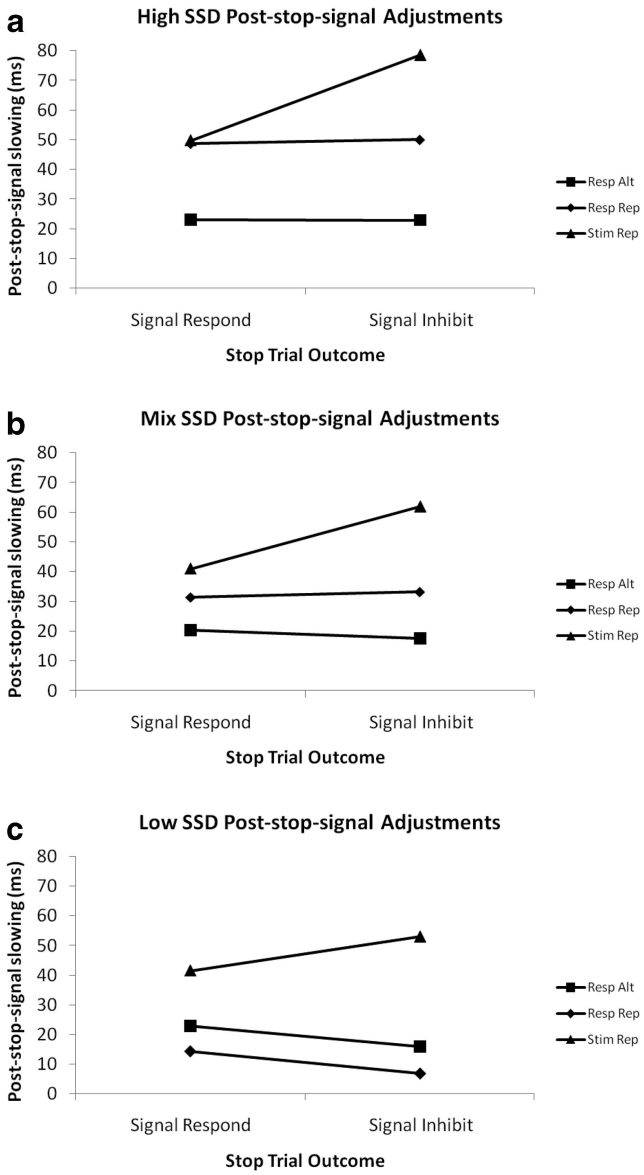

Figure 3. Experiment 2 post-stop-signal adjustment calculated as a double difference score [(S + 1) − (S − 1)] − [(G + 1) − (G − 1)] for (a) the high-stop-signal-delay (SSD) tracking condition, (b) the mixed-SSD tracking condition, and (c) the low-SSD (Figure 3c) tracking condition. Stimulus repetitions (Stim Rep) are instances in which Trial N and Trial $N + 1$ have the same go stimulus; response repetitions (Resp Rep) are instances in which Trial N and Trial $N + 1$ have the same correct response but a different go stimulus; and response alternations (Resp Alt) are instances in which the stimulus and the response change from Trial N to Trial $N + 1$.

alternations (23 ms vs. 19 ms), $F(1, 184) < 1$, $MSE = 646$, $B = 5.65$. The probability of a stop signal was held constant at .33, so these differences suggest that post-stop-signal adjustments are sensitive to the overall probability of failed inhibition.

The response repetition and response alternation conditions allowed additional tests of the goal priority, error detection, and conflict hypotheses. As in Experiment 1, we collapsed across response repetitions and response alternations in evaluating these hypotheses. Post-stop-signal slowing was similar for signal-respond trials and signal-inhibit trials (*M*s = 25 ms and 23 ms, respectively), $F(1, 92) < 1.0$, $MSE = 363$, $B = 4.55$, supporting the goal priority hypothesis or a combination of the error detection and conflict hypotheses. To evaluate the conflict hypothesis, we compared slowing after signal-inhibit trials in the two tracking procedures in the mixed session. The mixed-high SSD procedure yielded longer SSDs than did the mixed-low SSD procedure (*M*s = 324 ms and 211 ms for mixed-high and mixed-low SSD procedures, respectively), $F(1, 23) = 227.26$, $MSE = 674$, $p < .01$, $B > 10$, which means that there should be greater conflict and therefore greater slowing in the mixed-high SSD procedure. Excluding stimulus repetitions, there was no difference in signal-inhibit slowing between mixed-high and mixed-low SSD conditions (*M*s = 32 ms and 33 ms for mixed-high and mixed-low SSD, respectively), $F(1, 23) < 1$, $MSE = 819$, $B = 6.33$, providing strong evidence against the conflict hypothesis, and consequently against the combination of the conflict and error detection hypotheses, as an explanation of equivalent slowing after signal-inhibit and signal-respond trials. Replicating Experiment 1, the post-stop-signal slowing data appear best explained by the goal priority hypothesis, as stop signals appear to shift priority to the stop task independently of stopping success.

The memory hypothesis predicts greater slowing when the go stimulus repeats, especially after signal-inhibit trials, and could accommodate greater slowing when the response repeats. There was greater slowing after stimulus repetitions ($M = 54$ ms) than after nonstimulus repetitions ($M = 26$ ms), $F(1, 92) = 81.84$, $MSE = 363$, $p < .01$, $B > 10$. Within stimulus repetitions, there was greater slowing after signal-inhibit trials ($M = 65$ ms) than after signal-respond trials ($M = 44$ ms), $F(1, 92) = 41.45$, $MSE = 363$, $p < .01$, $B > 10$. There was also greater slowing after response repetitions ($M = 31$ ms) than after response alternations ($M = 20$ ms), $F(1, 92) = 10.55$, $MSE = 363$, $p < .01$, $B > 10$. These results suggest that both stimuli and responses become associated with stopping. However, response repetition slowing did not differ between signal-inhibit trials ($M = 31$ ms) and signal-respond trials ($M = 30$ ms), $F(1, 92) < 1$ $MSE = 363$, $B = 5.77$, suggesting that different processes may underlie response repetition and stimulus repetition effects. Response repetition slowing may reflect an increase in response threshold, but we did not see the corresponding increase in accuracy that usually comes with an increase in threshold (Ratcliff & Smith, 2004).

Conclusions

Experiment 2 tested whether post-stop-signal slowing depends on the probability of a stop signal or the probability of failed inhibition. We held the probability of a stop signal constant and found greater slowing, the higher the probability of failed inhibition. In future research, we plan to manipulate the probability of a stop signal and the probability of failed inhibition independently.

Replicating Experiment 1, the results for post-stop-signal slowing support the goal priority hypothesis (Leotti & Wager, 2010; Liddle et al., 2009) and provide evidence against the error detection (Laming, 1968; Rabbitt, 1966) and conflict monitoring (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000) hypotheses. The results for stimulus repetition trials support the memory hypothesis (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008). In contrast to Experiment 1, we found support for the memory hypothesis working at the level of responses.

General Discussion

Consistent with previous experiments, we found evidence for control adjustments that adapt to the competing demands of stopping and going in the stop-signal paradigm. Subjects slowed go RT after stop trials (Experiments 1 and 2). Their slowing was greater when stop signals were more frequent (Experiment 1) and when failed inhibition was more frequent (Experiment 2). These results allowed us to discriminate among five hypotheses that explain post-stop-signal slowing: We found equal slowing after signal-respond and signal-inhibit trials, which is consistent with the goal priority hypothesis (Leotti & Wager, 2010; Liddle et al., 2009) and inconsistent with the error detection (Laming, 1968; Rabbitt, 1966) and conflict monitoring hypotheses (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000). We found greater slowing with higher percentages of stop signals, which is inconsistent with the surprise hypothesis (Notebaert et al., 2009; Núñez Castellar et al., 2010). We found greater slowing for stimulus repetitions in both experiments, which is consistent with the memory hypothesis (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008), but we found mixed evidence for the memory hypothesis working at the level of responses. In addition, we found evidence against the hypothesis that longer RTs with higher proportions of stop signals (Experiment 1) result from accumulated post-stop-signal slowing. RT returned to pre-stop-signal baseline by the second trial after a stop signal. Thus, longer RT with more stop signals appears to result from proactive adjustments (Verbruggen & Logan, 2009b).

Much of the research on post-stop-signal adjustments focuses on local events from the preceding trial as signals that recruit control processes, but we showed that this is insufficient. We showed that control adjustments in the stop-signal paradigm depend on several factors, including the immediately preceding trial and global factors that extend beyond it. We showed that the magnitude of post-stop-signal adjustment depends on the probability of stop signals and the probability of failed inhibition. Thus, post-stop-signal adjustments are like proactive adjustments in that they depend on aggregates of several trials. However, we showed that post-stop-signal adjustments are different from proactive adjustments, demonstrating that proactive slowing does not result from aggregated post-stop-signal slowing. The mechanisms by which aggregates of several trials influence both reactive and proactive control adjustments are an important topic for future research.

Error Detection in the Stop-Signal Paradigm

The error detection hypothesis suggests that the recruitment of control processes occurs when an error is detected (Rabbitt, 1966).

This hypothesis was developed in the context of choice errors in RT tasks, where posterror slowing is commonly observed (Laming, 1968; Rabbitt, 1966). Recent studies have applied it to the stop-signal paradigm (Schachar et al., 2004; Verbruggen et al., 2008), where it predicts greater slowing after failed inhibition (signal-respond trials) than after successful inhibition (signal-inhibit trials). The data from both experiments contradicted this prediction, suggesting that the error detection hypothesis may not apply to the stop-signal paradigm. One possibility is that stop errors are different in important ways from choice errors. They are typically more frequent: Many stop-signal experiments aim for a 50% success rate (e.g., Logan et al., 1997), whereas most RT experiments aim for success rates (accuracy) above 90%. Subjects may be less concerned with correcting errors when they are more frequent (Gehring, Goss, Coles, Meyer, & Donchin, 1993). Also, stop errors are errors of timing rather than errors of choice. Subjects try to stop but do not stop quickly enough. Choice errors involve confusion among stimuli or responses and may be responded to differently than errors of timing. Another possibility is that the error detection hypothesis is not a valid explanation of posterror slowing in choice RT tasks. Other explanations are available, including surprise (Notebaert et al., 2009; Núñez Castellar et al., 2010) and goal priority (Leotti & Wager, 2010; Liddle et al., 2009). Additional research is necessary to bridge these two literatures.

Conflict Monitoring in the Stop-Signal Paradigm

The conflict-monitoring hypothesis (Botvinick et al., 2001) suggests that the recruitment of control processes occurs as a result of coactivation of competing responses. Evidence from countermanding eye movements in monkeys suggests that coactivation of movement (go) and fixation (stop) neurons occurs only on signal-inhibit trials (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000), so post-stop-signal slowing should be greater on signal-inhibit than on signal-respond trials. Our results showed no greater slowing for signal-inhibit trials, contradicting this version of the conflict monitoring hypothesis (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000). Possibly, monkey results do not generalize to humans or eye movement results do not generalize to keypress responses. Another possibility is that coactivation of fixation and movement neurons does not index response conflict in the stop-signal paradigm. Indeed, monkey data show coactivation of fixation and movement neurons at the beginning and end of every saccade (Hanes, Patterson & Schall, 1998), so coactivation of fixation and movement neurons would not be a valid signal for recruiting control processes.

Another possibility is that conflict occurs on every stop signal trial because the go stimulus and the stop signal both occur and require responses that are incompatible. Indeed, the race model (Logan & Cowan, 1984) assumes that the stop process and the go process are engaged on every stop-signal trial. One or the other may win the race, but both runners are running. Executive processes may detect response conflict whenever two runners compete and produce post-stop-signal slowing regardless of the outcome of the race. The issue is whether response conflict depends on the entire race, as we are suggesting here, or on the competition at the end of the race, as Schall and colleagues (Ito et al., 2003; Stuphorn & Schall, 2006; Stuphorn et al., 2000) suggested. Further research will be required to distinguish these possibilities.

Surprise in the Stop-Signal Paradigm

Núñez Castellar et al. (2010) and Notebaert et al. (2009) showed that subjects slowed their responses after low-probability events, including errors in easy discriminations and correct responses in difficult discriminations, suggesting that surprising events recruit control processes. Our data contradicted the surprise hypothesis, showing greater slowing after frequent than after infrequent events (stop signals). It is possible that the slowing after correct trials observed by Núñez Castellar et al. (2010) and Notebaert et al. (2009) was a response to the feedback presented after each trial in those studies, which may have recruited a control process that raised the threshold for a response in the subsequent trials, increasing both RT and accuracy. We did not present trial-by-trial feedback after go or stop trials, so feedback processing cannot explain our results.

Goal Priority in the Stop-Signal Paradigm

Our results were most consistent with the goal priority hypothesis (Leotti & Wager, 2010; Liddle et al., 2009). We found equal slowing after signal-inhibit and signal-respond trials, suggesting that stop signals recruited control processes that adjusted the balance between stopping and going independently of stop success. These results encourage further exploration of the role of motivation and optimization of performance in the stop-signal task (see also Wong-Lin, Eckhoff, Holmes & Cohen, 2010).

The goal priority hypothesis is not as constrained as the error detection (Laming, 1968; Rabbitt, 1966) and conflict monitoring (Botvinick et al., 2001) hypotheses. Error detection and conflict monitoring attribute post-stop-signal slowing to a specific event (errors and response conflict, respectively) that occurs on the immediately preceding trial. By contrast, goal priority can depend on factors that extend beyond the immediately preceding trial, such as subjects' perception of the percentage of stop signals (Logan, 1981), explicit cues that indicate the importance of the stop signal (Verbruggen & Logan, 2009b), and the magnitude of reward given for stopping and going (Liddle et al., 2009). Thus, goal priority is a more flexible hypothesis, and that flexibility may underlie its ability to account for a broader range of phenomena. Future research should exert stronger constraints on the goal priority hypothesis.

Strategic Adjustment or Automatic Priming?

We suggest that subjects strategically shift their priority toward caution after a stop signal, but an alternative account is that a stop signal on Trial S automatically primes the stop process on Trial S + 1, slowing go RT (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008). Our current design cannot distinguish strategic adjustments from automatic priming after stop signals, and further research will be necessary to distinguish these explanations (McNamara, 2005). One possibility would be to implement a contingency in the sequence of trials that eliminates stop trial repetitions. If post-stop-signal slowing results from the stop Trial S automatically priming the stop goal on Trial S + 1, then

post-stop-signal slowing should not be affected by this contingency. If post-stop-signal slowing results from a strategic shift in task priority after a stop trial, then subjects may incorporate this contingency into their strategy and eliminate or reverse post-stop-signal slowing. If post-stop-signal slowing results from automatic priming, the contingency should not affect it.

Both Experiments 1 and 2 support the memory hypothesis, which involves priming from the go stimulus. The go stimulus on a stop Trial S becomes associated with stopping; when it repeats on Trial S + 1, it retrieves the association, which primes the stop task, increasing S + 1 reaction time (Verbruggen & Logan, 2008a, 2008b; Verbruggen et al., 2008). It is possible that similar priming effects could result from associations between stopping and responses. Indeed, we find greater slowing after response repetitions than response alternations in Experiment 2. However, the response repetition effects may also be explained in terms of strategic adjustments of response criteria, so further research would be required to demonstrate response priming. However, Verbruggen and Logan (2009a) investigated priming in the stop-signal paradigm, presenting the words *GO* and *STOP* as primes for the go process and the stop process, and found effects smaller than 10 ms. The effects in the present experiments were larger than that, so it seems unlikely that our effects were entirely due to automatic priming. Moreover, proactive adjustment of mean RT appears to be strategic, as subjects become more cautious in advance of stop trials (see also Verbruggen & Logan, 2009b). Therefore, we have evidence for both strategic (proactive slowing) and automatic (stimulus priming) adjustments, and additional research is necessary to investigate priming as an explanation for post-stop-signal slowing after trials that are not stimulus repetitions.

What Do Post-Stop-Signal and Proactive Control Processes Adjust?

A theory of cognitive control must specify what is controlled, and that requires a theory of the subordinate processes that are subject to cognitive control (Logan & Gordon, 2001). For choice RT tasks, the subordinate processes that respond to stimuli and generate responses are well described by *stochastic accumulator* models (for a review, see Ratcliff & Smith, 2004). In these models, a choice response is generated when evidence accumulates to a threshold. Evidence is stochastic, so the accumulation process produces distributions of finishing times that correspond well to RT distributions. Stochastic accumulator models include three basic parameters that could be adjusted in response to control signals, such as errors, response conflict, or the occurrence of stop signals: the *onset* of accumulation, the *rate* of accumulation, and the *threshold* of accumulation (Ratcliff & Smith, 2004). Delaying the onset, reducing the rate, or raising the threshold could all produce longer RTs. Fortunately, changes in these parameters have different consequences for RT and accuracy that can be used to discriminate among them: Delaying the onset increases RT without affecting accuracy; reducing the rate increases RT and reduces accuracy; and increasing the threshold increases RT and accuracy. Indeed, Rabbitt (1966) and Laming (1968) interpreted posterror slowing as an adjustment of response threshold that increased accuracy (i.e., a change in the speed–accuracy trade-off).

It is difficult to interpret the post-stop-signal slowing in our experiments in terms of changes in onset, rate, or threshold because there were complex interactions involving pre versus post and trial type (stimulus repetition, response repetition, and response alternation). An experiment with more trials would be required to provide enough data to fit a stochastic accumulator model to RT distributions for correct and error responses in each condition (Ratcliff & Smith, 2004). This is an important goal for future research.

The proactive slowing observed in Experiment 1 is easier to interpret because percentage of stop signals had no main effect on accuracy and did not produce any significant interactions. Thus, the proactive slowing we observed is most likely due to a delay in the onset of accumulation. A reduction in accumulation rate is implausible because control adjustments should increase cognitive control and therefore increase accumulation rate (Botvinick et al., 2001). An increase in threshold would produce an increase in accuracy, which we did not observe. Verbruggen and Logan (2009b) fit the diffusion model to RT and found that proactive adjustments delayed onset but also increased threshold (also see Logan, 1981). Further investigation of the mechanisms that mediate control is an important goal for future research.

Conclusions

We have shown that control adjustments in the stop-signal paradigm are sensitive to the immediately preceding trial and to aggregates of trials beyond the immediately preceding one. The results allow us to rule out three of five hypotheses that account for slowing after stop-signal trials, leaving goal priority and memory as viable explanations. The evidence that global factors influence post-stop-signal slowing sets the stage for new research that addresses the nature of the signals to the control processes, how the signals are processed, and how the results of executive processing adjust subordinate processes to balance competing demands.

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