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Perspectives on stopping behavior : process analyses of stop-signal inhibition

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Abstract

This study examines the effect of response readiness on the stopping of motor responses. Thirteen subjects performed a primary task requiring a speeded choice reaction on go trials and response inhibition on nogo trials. An occasional cue informed subjects that a nogo trial was imminent but left them uncertain about the number of go trials separating the cue and the upcoming nogo trial. This setup was meant to create test episodes of reduced response readiness (i.e., trial sequences initiated by the cue and terminated by the nogo signal) and control episodes, in which subjects were ready to execute a speeded choice reaction (i.e., trial sequences consisting only of go trials). During both episodes, a visual stop signal could occasionally and unpredictably follow go signal onset, instructing subjects to withhold their response to the go signal. Choice reactions on go trials were delayed during test episodes relative to control episodes. Most importantly, stop reactions were delayed, not facilitated, during test episodes compared to control episodes. These findings were taken to suggest that reduced readiness gives rise to more forceful responses that are then more difficult to inhibit.

2.1 Introduction

Consider the following: you are about to cross a busy shopping street when the traffic sign changes from 'walk' to 'don't walk'. After briefly considering the distance across and the drivers that are lined up, you decide to stop walking. This example illustrates the importance of stopping as an act of control. Stopping is a clear case of executive intervention that can be studied empirically using a relatively simple laboratory analogue, the *stop-signal paradigm*. The stop-signal paradigm consists of a reaction time (RT) task in which the occasional presentation of a stop signal indicates that the prepared or ongoing response must be cancelled. The probability of successful stopping can be manipulated by varying the timing of the stop signal relative to the respond signal. Stopping is easy when the stop signal is presented early, but difficult, or impossible, when it is presented late vis-à-vis the respond signal (e.g., Lappin & Eriksen, 1966; Logan, 1994; Logan & Cowan, 1984).

The performance in the stop-signal paradigm can be conceptualized in terms of a race, in which the stopping process and the go process compete to finish first (see Logan & Cowan, 1984, for an analytic approach). If the stop process finishes before the go process, the response is inhibited. By contrast, if the go process finishes before the stop process, the response is executed. Based on a small set of formal assumptions (i.e., the latency of the go process is not affected by the presence of stop signals, and the latency of stop signal inhibition is assumed to be constant), it is possible to calculate the latency of the covert stop process. The race model provides an excellent account of stopping data obtained using different variations of the stop-signal paradigm (see Logan, 1994, for a review). The stop-signal paradigm has been success-

fully applied in studies using other dependent measures than RT, such as brain potential measures (e.g., De Jong, Coles, Logan, & Gratton, 1990; Van Boxtel, van der Molen, Jennings, & Brunia, 2001), heart rate changes (Jennings, van der Molen, Brock, & Somsen, 1992), and single-cell recordings (e.g., Hanes, Patterson, & Schall, 1998). Investigators used the stop-signal paradigm to examine inhibitory control in various populations, including monkeys (e.g., Hanes et al., 1998) and children diagnosed as hyperactive (e.g., Schachar & Logan, 1990). Others examined age-related changes in stopping latency (e.g., Band, van der Molen, Overtom, & Verbaten, 2000; Kramer, Humphrey, Larish, Logan, & Strayer, 1994; Ridderinkhof, Band, & Logan, 1999; Williams, Ponsse, Schachar, Logan, & Tannock, 1999). Again, others evaluated the detrimental effect of alcohol (Mulvihill, Skilling, & Vogel-Sprott, 1997) or the beneficial influence of methylphenidate (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989).

Although the race model accounts quite well for a wide array of stopping data, it provides little insight into the nature of the stopping process itself (cf. Logan, 1994). To learn more about the stopping process, few studies crossed stopping with a form of inhibition that Logan (1994) dubbed *reactive inhibition*, that is a residual side effect of previous task processing that should be overcome (cf. Logan, 1994). Logan (1981), for example, observed that stopping latency is approximately equal for spatially compatible and incompatible responses (see Logan and Irwin, 2000, for a recent replication). Apparently, stopping does not interact with the ability to resolve the conflict between the prepotent compatible response and the spatially incompatible response (e.g., Kornblum, Hasbroucq, & Osman, 1990). Others crossed stopping with the inhibition of responses to target stimuli flanked by distracters assigned to the same or opposite response (Kramer et al., 1994; Ridderinkhof et al., 1999). These investigators found that responses to targets flanked by incompatible distracters were more difficult to inhibit than responses to compatible displays. This pattern of results was interpreted to suggest that stopping and the need to inhibit the (incorrect) response to incompatible flankers queue up, or compete for execution (cf. Ridderinkhof et al., 1999).

In reactive inhibition, the process producing inhibition may be engaged deliberately but the resulting inhibition that should be overcome, subsequently, is not intended. Contrary to reactive inhibition, stop signal inhibition requires the subject to take deliberate action. The goal of the current study is to examine the interaction of stopping with a form of inhibition that is also intended. This form of inhibition is the reduced readiness to respond that can be elicited by inserting 'nogo' or 'catch' signals into the primary task trial series. It is well known that the insertion of nogo signals delays response latency on go trials. This effect is usually interpreted to suggest that nogo signals reduce the readiness to respond, and thus increase RT, in order to avoid false alarms (e.g., Luce, 1986, for a review). Conceptually, response inhibition to stop signals and inhibitory control of response readiness are considered to represent two varieties of 'intended' inhibition (e.g., Logan, 1994). Empirically, stopping latency has been observed to vary with psychophysiological indicators of response readiness. For example, low levels of response readiness, as indexed by brain potential measures (i.e., the lateralized readiness potential) are associated with successful inhibits (e.g., De Jong et al., 1990; Van Boxtel et al., 2001). Conversely, high levels of response readiness, as indexed by cardiac measures (i.e., heart rate deceleration) are associated with failed inhibits (Jennings, van der Molen, Pelham, Brock, & Hoza, 1997).

Although it has been argued that interaction between tonic inhibitory control of response readiness and phasic inhibitory control of an imminent response would be of consider-

able interest (cf. De Jong, Coles, & Logan, 1995, p. 507), the current report presents the first attempt at a systematic assessment of this issue. The task devised to examine the influence of reduced response readiness on stopping latency is a hybrid go-nogo/choice reaction task. Subjects performed a primary task requiring (a) the execution of a speeded choice reaction on go trials and (b) response inhibition on nogo trials, but only if preceded by an occasional nogo cue. The nogo cue informed subjects that a nogo trial was imminent but left them uncertain about the number of go trials inserted between the nogo cue and the nogo signal. Trial sequences initiated by the nogo cue and terminated by the nogo trial were dubbed *test episodes*. These test episodes are assumed to be associated with reduced response readiness. During both test and control episodes, a visual stop signal could be presented instructing subjects to withhold the response activated by the go signal. Stop signals were presented using a tracking procedure targeted at 50% correct inhibits. Based on the psychophysiological findings reviewed above, it is hypothesized that reduced response readiness associated with the test episodes of the go-nogo/choice reaction task would facilitate stopping on stop-signal trials, indexed by a decrease in stop latency.

2.2 Method

2.2.1 Participants

Thirteen undergraduate students (eight females and five males, mean age 21 years) participated to fulfill course requirements. They also received a monetary reward of DFL. 50,- (approximately \$ 20,-). All subjects were right-handed and had normal or corrected-to-normal vision.

2.2.2 Apparatus and signals

Subjects faced a black computer screen at a distance of 50 cm. The signals for the primary task were the white uppercase vowels A, E, U, or O, and consonants R, S, V, Q, or X. The signals were presented at central location replacing a white fixation square. The visual angle subtended by each signal was approximately $2.30^\circ \times 4^\circ$. Signal duration was 500 ms and the interval between successive signals varied between 1250 and 1750 ms in steps of 125 ms. Left- and right-hand responses were collected from the 'z' and '/' keys on the computer keyboard. Timing was accurate to the nearest five ms. Occasionally, a visual stop signal was presented. The stop signal was indicated by a color change of the letter from white to red.

2.2.3 Task and design

The primary task involved the classification of single letters from the letter set. The vowels A, E, U and O were assigned to one response hand and the consonants R, S, V, and Q were assigned to the other response hand. The letter X served as a nogo cue that informed subjects to withhold their response to the first up-coming O or Q, but kept them uncertain about the num-

ber of letters separating cue and nogo trial. Obviously, subjects were instructed to refrain from responding to the nogo cue. The number of intervening go trials was either 0, 2, 4, or 6. Increments of two letters were used (a) to create episodes varying along a considerable time range, and (b) to limit the number of different episodes. Trial sequences terminated by an O or Q nogo trial, and preceded by the X nogo cue, will be referred to as *test episodes*. Trial sequences terminated by an O or Q, not preceded by an X, were used as *control episodes*. A scheme of test and control episodes is presented in Figure 2.1. Test and control episodes were separated, randomly but equiprobably, by 1, 2, or 3 go trials. This design resulted in equal probabilities of the letters A, E, U, R, S, and V (.125). The letters O and Q were presented with a probability of .094 and the letter X with a probability of .063.

Test Episodes	Intervening trials	Control Episodes
X-O/Q	L	O/Q
X-L-L-O/Q	L-L	L-O/Q
X-L-L-L-O/Q	L-L-L	L-L-O/Q
X-L-L-L-L-O/Q	L	L-L-L-L-O/Q
X-L-L-L-L-L-O/Q	L-L	L-L-L-L-O/Q
X-L-L-L-L-L-L-O/Q	L-L-L	L-L-L-L-O/Q
X-L-L-L-L-L-L-L-O/Q	L	L-L-L-L-L-L-O/Q
X-L-L-L-L-L-L-L-L-O/Q	L-L	L-L-L-L-L-L-O/Q
X-L-L-L-L-L-L-L-L-L-O/Q	L-L-L	L-L-L-L-L-L-O/Q
X-L-L-L-L-L-L-L-L-L-L-O/Q	L	L-L-L-L-L-L-L-L-O/Q
1-2-3-4-5-6	< Letter Position >	1-2-3-4-5-6

Figure 2.1: Schematic representation of test and control episodes. Test episodes were initiated by the nogo cue letter X requiring subjects to refrain from responding and signaling a nogo trial with letter O or Q requiring subjects to withhold their choice reaction. The letters O or Q were also used to terminate control episodes. When terminating control episodes, O and Q required a speeded choice reaction. Episodes included a varying numbers of go trials (indicated by L) preceding the letter O or Q (0, 2, 4, or 6). Episodes were separated randomly but equiprobably by a varying number of go trials (indicated by the letter L; 1, 2, or 3). Episodes were presented in pseudo-random order (see text for further clarification). L indicates a stop trial (i.e., a color change of the letter) instructing subjects to withhold the response to the letter.

Visual stop signals were presented using a single tracking algorithm (Levitt, 1971). This algorithm continuously adjusted stop signal delay to obtain an overall response inhibition percentage of approximately 50%. Thus, upon successful stopping, the stop signal delay on the next stop trial was increased by 50 ms. Failures to inhibit were followed by a 50 ms decrease in stop signal delay. Stop signals were presented only on go trials and at positions 2, 4,

and 6 of an episode for (a) low stop-signal probability to avoid unwanted strategies, and (b) high numbers of stop trials per position. Stop-signal probability was .15 for both test and control episodes.

2.2.4 Procedure

Subjects performed their task in a dimly lit, sound attenuated room. They were instructed to respond as quickly and accurately as possible and to avoid errors of commission on nogo signals. Subjects were told that a stop signal would be presented occasionally, requiring them to refrain from responding to the primary task signal. It was explained to them that stop signal delay would vary across trials so that on some trials stopping would be easy whereas on other trials stopping would be difficult or even impossible.

Subjects received a total of 25 test blocks consisting of 320 trials each. They completed their task in three sessions on consecutive days. On the first day, they received seven test blocks preceded by one hour of practice. On the second and third days, subjects performed on nine test blocks preceded by one practice block. There were short intermissions between test blocks and a longer pause after three blocks. Performance feedback was given after each block. During the experiment, the primacy of the choice/go-nogo task was stressed.

2.2.5 Stop-latency estimation

According to the independence assumption of the race model, the stop process does not affect the latency of the go process. This implies that the left side of the distribution of RTs on no-signal trials (i.e., trials without a stop signal) representing fast RTs, matches the distribution of RTs on stop trials that escape inhibition. The latency of the stop process can be estimated from the start and the finish of the stop process. The start of the stop process is under experimental control by the stop signal delay, but the finish time has to be inferred from the observed no-signal RT distribution. If responses are not stopped on $n\%$ of the stop trials, the finish of the stop process is on average equal to the n -th percentile of the RT distribution on no-signal RTs. Finally, mean stop signal delay is subtracted from this finish time to obtain an estimate of stop latency (see Logan, 1994). Stop-signal tracking based on inhibition rates of 50% provides stop latency estimates that are derived from the center of the no-signal RT distribution (hence the term 'central estimates'), and are relatively insensitive to violations of the assumptions of the horse-race model (e.g., Band, 1997; Logan, Schachar, & Tannock, 1997).

2.3 Results

Mean RTs and error rates were computed after the removal of outliers from the RT distribution (i.e., RTs $> M \pm 2.5 SD$) on a subject-by-subject basis. This resulted in the rejection of 2.0% and 2.1% of the trials for test and control episodes, respectively. Two different sets of analyses were performed on the data. First, mean RTs and error rates on no-signal trials (i.e., trials without a stop signal) were examined to evaluate whether response latency discriminated

between test and control episodes. Secondly, performance on stop trials was examined to determine whether stopping efficiency differed between episodes.

2.3.1 Analysis of performance on no-signal trials

Mean choice RTs, standard deviations, and error rates are presented in Table 2.1. Test episodes including errors of commission to both X, and O or Q were excluded from the present analysis. The percentage of rejected episodes was 9.7%.

Mean RTs were subjected to ANOVA with Episode (test vs. control) and Letter position (1, 2, 3, 4, 5, vs. 6) as within Ss factors. ANOVA yielded significant main effects of Episode, $F(1, 12) = 84.04, p < .001$, and Letter position, $F(5, 60) = 4.56, p < .001$, as well as a significant interaction between Episode and Letter position, $F(5, 60) = 9.91, p < .001$. Letter position did not systematically affect the latency of responding (i.e., test episodes: 481, 469, 467, 476, 481, and 470 ms; control episodes: 454, 447, 451, 445, 439, and 452 ms, for early to late positions, respectively). Follow-up analysis indicated that significant linear and quadratic trends were absent ($F_s < 1$). Thus, the data were collapsed across letter position in the analyses reported below. The outcomes of these analyses are presented in Table 2.1.

Table 2.1: Mean reaction times, error percentages (choice errors, omission errors, and commission errors), and standard deviations (in parentheses) for test and control episodes.

Episode	RT (ms)	Errors (%)		
		Choice	Omission	Commission
Test				
X	395 (42)	-	-	13.4 (11)
L	474 (41)	7.5 (4)	.8 (.6)	-
O/Q	487 (40)	-	-	34.5 (18)
Control				
L	448 (41)	9.7 (4)	.8 (.5)	-
O/Q	473 (45)	9.0 (4)	1.7 (1)	-

Note: L refers to the letters A, E, U, and R, S, V. Subjects were instructed to refrain from responding to the letter X and the letters O and Q when the latter were presented during a test episode. The letters O and Q required a speeded choice reaction when presented during control episodes.

Table 2.1 makes two important points. First, response latency was slower and the percentage of choice errors was lower during test episodes compared to control episodes, $F(1, 12) = 84.04, p < .001$, and $F(1, 12) = 44.85, p < .001$, respectively. This pattern of results demon-

strates the effectiveness of the nogo cue (the letter X) in altering the speed/accuracy tradeoff between episodes. It should be noted, however, that the (nogo) letters O and Q terminating test episodes gave rise to a sizeable number of commission errors. Moreover, response latency to the (go) letters O and Q (i.e., on trials when O and Q were not preceded by the nogo cue X) for control episodes was significantly slower compared to the latency of responding to the other letters, $F(1, 12) = 44.85, p < .001$.¹ These findings suggest that subjects failed to fully comply with the instruction provided by the nogo cue (X). Nonetheless, the speed and error reduction observed for test episodes strongly suggest that subjects adopted a more cautious strategy following the presentation of the cue.

2.3.2 Analysis of stop-signal inhibition

The stopping results are presented in Table 2.2. Preliminary analysis of the tracking algorithm indicated that the overall probability of responding given a stop signal was .509. Response probability was somewhat higher during control episodes compared to test episodes, $F(1, 12) = 36.97, p < .001$. A follow-up analysis indicated that stop signal delay did not differ between episodes, $F(1, 12) = .61, p = .45$. The latency of stop signal inhibition was estimated from the horse-race model using the procedure proposed by Logan and Cowan (1984), and which was described in the Methods. Stop latencies were computed separately for test and control episodes but across trial positions within episodes in order to obtain a sufficient number of observations. Although the difference in stop latency between episodes is small, it is statistically significant, $F(1, 12) = 7.89, p = .016$. Importantly, and contrary to expectations, stop latency during test episodes was slower, not faster, compared to control episodes.

Table 2.2: Probabilities of responding on stop trials, $P(\text{signal-response})$, mean stop-signal delays, mean observed and predicted signal-respond RTs, mean estimated stop latency, and standard deviations for test and control episodes.

Variable	Episode			
	Test		Control	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
P(signal-respond)	.43	.5	.54	.4
Stop-signal delay	160	31	162	31
Signal-respond RT				
observed	448	32	431	37
predicted	404	32	384	30
Estimated stop latency	290	38	283	40

¹ It could be argued that subjects responded slower to O and Q on go trials terminating control episodes because the O and Q are more difficult to discriminate compared to the other members of the letter sets (A, E, U, and R, S, V). This account is rendered less likely in view of approximately equal error rates, 9.0% for O and Q vs. 9.7% for the other letters, $F < 1$).

The race model was also used to predict RTs of responses on stop-signal trials that were not inhibited. As can be seen in Table 2.2, the predicted RTs of inhibition failures were considerably underestimated for both test episodes, $F(1, 12) = 221.14, p < .001$, and control episodes, $F(1, 12) = 256.83, p < .001$. Finally, as predicted by the race model, responses that escaped inhibition on stop-signal trials were significantly faster than responses on no-signal trials, both during test episodes and control episodes, $F(1, 12) = 11.26, p < .01$.

2.4 Discussion

This study set out to examine the interaction between two types of intentional inhibitory control, stop-signal inhibition and inhibition of response readiness. The inhibition of response readiness was manipulated by inserting nogo cues in the series of choice reaction trials. Subjects were required to refrain from responding to the nogo cue and the nogo cue signaled that another nogo trial was imminent. This manipulation created two types of episodes within the trial series; test episodes of reduced response readiness, when subjects were anticipating the nogo signal, and control episodes requiring speeded choice reactions on each trial. The effectiveness of this manipulation was supported by a significant shift in speed/accuracy tradeoff between episodes. Subjects' latency of responding slowed down significantly while they were awaiting a nogo trial and the proportion of choice errors decreased relative to control episodes. The efficacy of the nogo cue was not perfect, however. Subjects committed a sizable number of commission errors to the nogo signals terminating test episodes (O and Q) and responded considerably slower to these signals when part of control episodes (i.e., when Q and O were not signaled by the nogo cue X). Most likely, the relatively high incidence of commission errors on nogo trials is due to the emphasis that was placed in the instructions on the primacy of speeded choice reactions. The small proportion of omission errors to O and Q on go trials during control episodes is consistent with this interpretation.

Stop signals were presented during both episodes and their timing was manipulated using a tracking procedure. The tracking algorithm was targeted at a percentage of 50% correct inhibits. It appeared that the tracking was quite successful (51% correct inhibits), as it was in previous studies (Logan et al., 1997; Osman, Kornblum, & Meyer, 1986, 1990; Ridderinkhof et al., 1999; Williams et al., 1999). The percentages of correct inhibits differed between episodes; 57% vs. 46% for test and control episodes respectively. This finding could be taken to suggest that stop-signal inhibition is more efficient during episodes of reduced response readiness. However, given the approximately equal stop signal delays between episodes, the chance of the stop process winning the race during test episodes was somewhat higher because the go process was slower during test episodes compared to control episodes. Most importantly, stopping latency showed a small (7 ms), but statistically reliable, difference between episodes (the reliability of this difference is supported also by the results of simulations reported in Appendix 2-A). Contrary to expectations, stopping latency was slower, not faster, during test episodes. It was anticipated that stopping would be facilitated during episodes of reduced response readiness. The current results demonstrated the opposite trend, however.

Before interpreting slower stop-signal inhibition for test episodes, the difference between predicted and observed RTs of responses that escaped inhibition on stop trials must be addressed. The latency of stop-signal inhibition was estimated using the race model. This

model also allows one to predict the response latency of failed inhibits. In previous studies, the difference between predicted and observed RTs was used as a test of the independence assumption underlying the race model (De Jong et al., 1990; Jennings et al., 1992; Logan & Cowan, 1984). The difference between predicted and observed RTs is typically in the order of 15 ms or less, suggesting that empirical data fit the model fairly well. The difference observed in the current study was much larger and statistically significant; 44 and 47 ms for test and control episodes, respectively. The use of a visual rather than an auditory stop signal might have contributed to the observed differences in predicted and observed RTs. Stopping experiments with stop signals in the same modality as the primary-task respond signal also reported a statistically significant difference between predicted and observed RTs, albeit of smaller magnitude (Van Boxtel et al., 2001).

At this point, it should be noted, however, that the results of recent studies challenge the use of predicted versus observed RTs as an index of independence violation. As indicated above, Van Boxtel et al. observed a significant difference between observed and predicted RTs. Nonetheless, a detailed examination of psychophysiological response patterns associated with signal-respond and no-signal trials (i.e., central and peripheral indicators of response selection and activation) indicated that these patterns were virtually identical. This finding suggests independence rather than interference, in spite of the difference between observed and predicted RTs that was observed. Simulation studies basically arrived at similar conclusions. Thus, De Jong demonstrated that, rather than dependence, variability of stop latency gives rise to an underestimation of signal-respond RT. This result was replicated by Band (1997) who, in addition, demonstrated that variability in stop latency does not compromise estimates of stop latency when based on tracking procedures. In view of these studies, then, it must be concluded that the currently observed difference between observed and predicted RTs is most likely to result from variability in stop latency, not dependence between stop signal and reaction signal processing. Unfortunately, the current data do not provide a ready explanation for the allegedly high levels of stop signal variability.

Returning to the major finding, a more specific interpretation of why slower stopping should be associated with reduced response readiness can be derived from the analysis of response dynamics reported by Ulrich, Mattes, and Miller (1999). Ulrich et al. examined the magnitude and time course of response force in the Donders a- (simple), b- (choice), and c (go/nogo) tasks. They found that response force was virtually identical for a- and b-reactions. However, c-reactions were more forceful than both a- and b-reactions. Previously, these investigators reported that subjects produced responses that are more forceful when go probability was low than when it was high (Mattes, Ulrich, & Miller, 1997). They interpreted these findings in terms of an extended version of the response readiness model proposed by Niemi and Näätänen (1981). According to their reasoning, it is assumed that subjects reduce their readiness to respond when they perform a c- or go/nogo task in order to avoid commission errors. Additionally, it is assumed that the distance between response readiness and the motor action limit is increased with a decreasing probability of go trials. When response readiness is low, a large increment is needed to exceed the action limit, resulting in slow, but forceful responses. A similar explanation might apply to the present observation that stop latency is slower during inhibitory episodes. Assuming that response readiness is lower during inhibitory episodes, a larger increment is needed to exceed the action threshold. This will result in slower responses on no-signal trials and slower stopping on stop-signal trials, because the more forceful re-

sponses are more difficult to inhibit compared to responses executed when the distance to the action threshold is smaller (i.e., during control episodes).

The current observation that stop-signal inhibition varies as a function of the demands on inhibitory control exerted by the primary task suggests that both are influenced by a single factor, namely response readiness. This type of interaction is referred to as functional dependence (Logan & Cowan, 1984). Functional dependence does not imply stochastic dependence, which affords accurate prediction of trial-by-trial variability in stop latency from trial-by-trial variability in go RT. The race model only assumes stochastic independence between primary task processes and stop processes (Logan & Cowan, 1984; Osman et al., 1986; Ridderinkhof et al., 1999). Functional dependence does not violate the assumptions of the race model or bias the estimation of stop latency based on these assumptions.

In conclusion, it was predicted that reduced response readiness would be associated with a facilitation of stop-signal inhibition. In contrast, the present results indicated that the latency of stop-signal inhibition is prolonged during episodes of reduced response readiness. This pattern of findings is analogous to the results obtained in previous studies examining the interaction between stop-signal inhibition and a variety of reactive inhibition (Kramer et al., 1994; Ridderinkhof et al., 1999). These studies showed that stopping is more difficult when a response conflict has to be resolved. These findings are typically interpreted to suggest that stop-signal inhibition and reactive inhibition associated with the primary task compete for the same resources. The current results, however, point to a positive relation between the latency of stop-signal inhibition and the latency of reactions to the primary task signal. The positive relation may be interpreted to suggest that a state of reduced readiness has similar effects on stopping and primary task processing (see Band, 1997, for a full discussion of this issue). Alternatively, a state of reduced readiness may be associated with more forceful responses that are less easy to inhibit. The latter interpretation is now under investigation using electrical brain and electromyographic measures to assess the temporal and force dynamics of response activation and inhibition.

2-A Appendix

Use of a bad clock does not enhance the detection of differences in SSRT

It could be argued that the determination of the finish RT, and hence the estimation of stop latency, is less accurate when RTs are measured with clocks having suboptimal timing resolution. At the presentation of a respond signal, the counter clock generates a discrete sequence of equally distant time points, starting from zero. The time resolution of the counter clock is defined by the interval between two consecutive time points. The smaller this distance, the more accurate the counter clock. Recording a RT that is generated at a certain moment in time, involves rounding up to the time point counted next (see Ulrich & Giray, 1989 for a study on the effects of bad clocks on reaction time measurement).

The 'bad clock' issue was addressed by a series of simulations investigating the effects of time resolution of the counter clock on the detection of differences between two stop latencies estimated from two primary RT distributions. In the simulations, the numbers of stop trials included were 50, 250, and 600. The *a priori* effect sizes were fixed at 0, 7, and 25 ms. The time resolution of the counter clock was varied from 1, 5, 10, 25, to 50 ms. The values for mean and standard deviation of stop latency were set at 200 ms and 50 ms, respectively. Stop-signal delay was continuously adjusted throughout the simulations in steps of 50 ms according to a staircase-tracking algorithm, depending on the accuracy score on the previous stop trial.

Table 2-A1: Statistical power values that represent the chance of detecting a significant difference in stop latency estimated from two primary-task RT, Normal, distributions with different means ($M_1 = 400$ ms, $M_2 = 420$ ms).

N stop trials	Effect size	Time Resolution of Clock in ms				
		1	5	10	25	50
50	0	0.052	0.058	0.064	0.044	0.052
	7	0.222	0.252	0.218	0.158	0.182
	25	0.928	0.936	0.936	0.914	0.814
250	0	0.056	0.040	0.044	0.064	0.046
	7	0.558	0.618	0.602	0.362	0.144
	25	1.000	1.000	1.000	1.000	0.952
600	0	0.048	0.044	0.052	0.066	0.040
	7	0.924	0.888	0.858	0.490	0.130
	25	1.000	1.000	1.000	1.000	0.954

Note: Each power value is based on 500 replications.

Stop latencies were estimated from two primary RT distributions, the mean of primary RT distribution 1 ($M_1 = 400$) was set 20 ms faster compared to the mean of primary RT distribution 2 ($M_2 = 420$). The data presented in Table 2-A1 to 2-A3 represent power values, or the chance of finding a significant difference in stop latency between two conditions. Each power value was calculated over 500 replications and 13 subjects. Tables 2-A1 to 2-A3 refer to simulations using the Normal, Weibull, and Ex-Gaussian RT distributions, respectively.

It can be seen that the chances of finding a significant stop latency difference with the good clock increase substantially as the *a priori* defined effect size increases from 0 to 7, and to 25 ms. Also, increasing the number of stop trials included in the simulated experiment increases power considerably, especially with moderate effect sizes of 7 ms. Most importantly, the use of a clock of limited time resolution up to 50 ms does not increase the chance of finding significant differences in estimated stop latency compared to a good clock. In most cases, the use of less accurate timing resolution actually masks the detection of significant differences in estimated stop latency. These conclusions hold for primary-task RTs and stop latencies that are characterized by Normal, Weibull, as well as the Ex-Gaussian distributions. The latter finding is consistent with Logan and Cowan (1984) who indicated that the horse-race model does not assume RT distributions of a particular form (p. 313).

Table 2-A2: Statistical power values that represent the chance of detecting a significant difference in stop latency estimated from two primary-task RT, Weibull, distributions with different means ($M_1 = 400$ ms, $M_2 = 420$ ms).

N stop trials	Effect size	Time Resolution of Clock in ms				
		1	5	10	25	50
50	0	0.062	0.054	0.060	0.052	0.040
	7	0.112	0.130	0.122	0.128	0.082
	25	0.502	0.490	0.470	0.458	0.438
250	0	0.072	0.036	0.058	0.058	0.062
	7	0.230	0.246	0.212	0.206	0.110
	25	0.982	0.980	0.988	0.950	0.772
600	0	0.042	0.046	0.034	0.044	0.050
	7	0.502	0.430	0.426	0.312	0.076
	25	1.000	1.000	1.000	1.000	0.856

Note: Each power value is based on 500 replications.

Table 2-A3: Statistical power values that represent the chance of detecting a significant difference in stop latency estimated from two primary-task RT, Ex-Gaussian, distributions with different means ($M_1 = 400$ ms, $M_2 = 420$ ms).

<i>N</i> stop trials	Effect size	Time Resolution of Clock in ms				
		1	5	10	25	50
50	0	0.070	0.050	0.086	0.064	0.042
	7	0.130	0.126	0.128	0.134	0.146
	25	0.632	0.604	0.636	0.600	0.562
250	0	0.050	0.056	0.052	0.060	0.046
	7	0.352	0.378	0.352	0.232	0.148
	25	1.000	0.998	0.998	0.996	0.894
600	0	0.048	0.052	0.076	0.048	0.048
	7	0.644	0.628	0.596	0.396	0.132
	25	1.000	1.000	1.000	1.000	0.968

Note: Each power value is based on 500 replications.

