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**PREPARATION, ADJUSTMENT, AND
INHIBITION OF RESPONSES**

ACADEMISCH PROEFSCHRIFT

ter verkrijging van de graad van doctor
aan de Universiteit van Amsterdam
op gezag van de Rector Magnificus
prof. dr. J. J. M. Franse
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Contents

Acknowledgements	5
Chapter 1: Introduction	7
Historical Background	7
Chronopsychophysiology	8
Control Processes	9
Development	10
Organization of the Dissertation	11
Chapter 2: The Ability to Inhibit and Activate Speeded Responses: Multifaceted Developmental Trends	15
Experimental Overview	18
Experimental Tasks	22
General Discussion	37
Chapter 3: Top-down Effects in a Probabilistic-Priming Paradigm: Strategic Preparation and Response Inhibition	45
Introduction	45
Top-down effects: Preparation and Inhibition	46
Dependent Variables and Predictions	48
Preparation Effects	55
Inhibition Effect	65
General Discussion	69
Chapter 4: Mental Rotation Interferes With Response Preparation	75
Experiment 1	80
Experiment 2	90
Experiment 3	97
General Discussion	101
Chapter 5: Optimizing the Design and Interpretation of the Stop-Signal Paradigm	107
The Paradigm	109
Scope of the Article	120
General Method	121
Study 1	126
Study 2	142
Study 3	144
General Discussion	149
Chapter 6a: General Discussion and Summary (English)	157
Chapter 6b: Algemene Discussie en Samenvatting (Dutch)	163

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1 Introduction

Information-processing psychology is a relatively new science with fuzzy boundaries. And just as it happens in other new sciences, the scope of information-processing psychology is frequently widened. For example, an old-fashioned definition of the field might read:

Information-processing psychology investigates how a stimulus enters the mind and is translated into a response.

Although this definition would still apply to most of the research that takes place in the field, it ignores a number of important features of the mind, that are often summarized under the term *executive control*. In this dissertation these aspects will be referred to as top-down effects, to contrast them to the bottom-up effects that only represent stimulus-driven processes.

Historical Background

A historical overview of reaction-time experiments should begin with the work of Von Helmholtz (1850), who first performed measurements of neural conduction time in the frog. Donders (1868/1969) extended the scope of speed measurements to psychological processes. He introduced a method that was aimed at estimating the duration of component mental processes. He made a comparison between three classical task situations that were thought to differ in only one processing requirement. In Donders' A task or simple reaction time (RT) task, subjects were instructed to make a fast response to a known sound. This RT was compared with the RT on Donders' B task (choice RT task), where one of two possible responses was made. The difference between these two measures was considered as an estimation of the duration of stimulus discrimination and the response choice. Donders' C task (disjunctive RT task or go/no-go task) consisted of the requirement to respond to one sound, but not to another. The difference between the C and A was interpreted to be the duration of the stimulus discrimination, and the difference between C and B was interpreted as the duration of response selection.

This revolutionary approach to mental processes was soon criticized by Külpe (1893/1909) for invalid assumptions. Donders' (1868/1969) Subtraction Method was based on the idea that the difference between tasks can be expressed in terms of an added requirement. Criticism focused on the assumption that the reaction process is built up in a serial architecture, and that the speed and communication of stages that are not manipulated are not affected by the insertion or deletion of an extra process. This point applies also to the subjects' strategy during task performance. For example, while the A- and C-task allow preparation of the response, the B-task requires a more conservative strategy. In other words, the mind can be set to the performance on a task, and the subjects' expectations are likely to play an important role in speeded responses.

Despite the criticism on the Subtraction Method, the seminal contribution of Donders was still appreciated a century later, when Sternberg (1969) proposed a modern version of a mental chronometric procedure. His Additive Factors Method does not involve the addition or deletion of processing stages, but concerns manipulations of the duration of processing stages. The pattern of interactions between manipulations is used as a tool to investigate the locus of effects, and thus elucidate the model of cognitive processes.

For example, adding random noise to the stimulus display delays RT due to a perceptual effect, and the requirement to make a more complicated stimulus-response (SR) translation delays

RT due to a response selection effect. These two manipulations have additive effects because they affect two independent stages. If, however, two manipulations affect the duration of the same stage, the effects typically interact. The logic of the Additive Factors Method can be reversed by saying that the locus of the effect of an unknown factor, e.g., a new psychoactive drug, can be derived from the pattern of interactions of this factor with other, calibrated factors. Likewise, the specific nature of cognitive development and aging has been assessed with the Additive Factors Method (e.g., Ridderinkhof, 1993; Smulders, 1993).

The model that underlies the Additive Factors Method has been criticized for assuming independent stages and a serial architecture. These assumptions have been challenged by experiments that showed signs of separate analysis of stimulus dimensions (Miller, 1982) and the effects of irrelevant stimulus element on motor processes (see Coles, Smid, Scheffers, & Otten, 1995, and Van der Molen, Bashore, Halliday and Callaway, 1991, for reviews). These findings suggest that 1) partial output of a processing level can be transmitted to later processing levels before the process is finished and that 2) later processing levels can be active in parallel with earlier levels.

Chronopsychophysiology

An important role in the discussion about the serial versus parallel architecture of the information processing system is played by psychophysiological indices (see Van der Molen et al., 1991). During the reaction process, the steps that lead to the response have a reasonably consistent timing relative to the onset of the stimulus. Because some of these processes are accompanied by electrophysiological contributions to the electroencephalogram (EEG), their activity can be investigated by averaging event-locked time series of the EEG.

The clearest wave in the thus acquired event-related potentials (ERP) is a positive potential over parietal and central leads that reaches its maximum 300 ms or more after stimulus onset, and is therefore called the P300 or P3. The peak latency of the P3 is shown to be sensitive to manipulations of the duration of perceptual processes, whereas it is insensitive to motor processes (Donchin, 1981). This has led researchers of the cognitive architecture to use the P3 as an additional measure besides RT to identify the locus of speed effects. The amplitude of the P3 has been used as an index of the amount of information that is extracted from the stimulus, and is known to be rather sensitive to task-relevance and stimulus probability.

An interesting derivative of the ERP is the lateralized readiness potential (LRP, see Coles, 1988). This waveform describes the development over time of a response preference for one hand over the other. Because hemispheric differences and lateralizations that are not related to the side of the response are removed by the averaging procedure, the LRP is considered to be a real-time index of the preference for a response. The onset of the LRP is used as another chronopsychophysiological measure that reflects the timing of response activation.

Just before the response, the electromyogram (EMG) indicates that a motor command arrives at the muscles. The timing of EMG and RT have a high correlation. However, EMG activity is not always followed by an overt response (e.g., Jennings, Van der Molen, Brock, & Somsen, 1992). This fact makes it possible to treat trials with EMG for an incorrect response as a category between correct and incorrect (e.g. Coles, Scheffers, & Fournier, 1995)

The contribution of psychophysiological measures to the debate about continuous parallel-processing models versus discrete serial-processing models can be illustrated with two experiments. It has been shown with the LRP that irrelevant elements in a stimulus array can first cause preparation for the incorrect hand, before the relevant element leads to preparation for the correct hand (e.g., Gratton, Coles, & Donchin, 1992). This result can not be explained with a model in which perceptual analysis of the stimulus leads to constant output about the identity of the relevant

stimulus element. Instead, it suggests that the responses that belong to irrelevant elements are primed at the level of motor processes. This response priming interferes with the execution of the correct response if the priming is invalid, but facilitates the execution if the priming is valid.

Other experiments with the LRP suggested that the perceptual analysis of a multidimensional stimulus can deliver partial output about one dimension (Miller & Hackley, 1992; Osman, Bashore, Coles, Donchin, & Meyer, 1992). In these experiments, an easy-to-analyze stimulus dimension was mapped onto one of two response hands, and a hard-to-analyze stimulus dimension instructed subject whether that response should be executed or not. In this experiment, LRPs showed preparation for the “correct” hand in the condition where no response should be executed. These results have shown that motor processes can start on the basis of partial output about the correct hand before the perceptual processes are finished.

Consensus is growing for the view that the serial stage model is not valid to describe the response process to multi-element stimuli or stimuli of which dimensions are easy to separate (e.g., Coles et al., 1995; Sanders, 1990). However, the experiments that support the existence of parallel processing have primarily been based on relatively simple stimulus-response translation processes, and the overlap has been shown primarily for perceptual processes in combination with response activation.

In summary, the output of a stage is not constant, processing stages do not follow a strict serial sequence, and the duration of later stages can be affected by a failure to make the proper selection on earlier stages. This holds that the RT is not just an addition of the duration of stages, but results from the length of a variable route along subprocesses (e.g. Miller, 1993; Townsend & Schweickert, 1989). Thus, RT effects may differ from the sum of effects on separate processing levels. However, influences on separate levels do not easily interact in models that allow for parallel processing either (Roberts & Sternberg, 1992).

Control Processes

Another criticism of stage models is that they do not accommodate changes to the speed of processing as a result of strategic, or top-down influences. For example, the stage model can not explain how subjects can trade the accuracy of responses for a gain in speed, because the duration of stages is considered to depend on stimulus information alone. This stimulus-driven or bottom-up approach to performance is in sharp contrast with the observations of Rabbitt (1966, 1968) that errors are frequently followed by slower responses that are more often correct. That is, subjects change their performance on the basis of observed results. Rabbitt (1979) argued that serial stage models ignore a critical feature of human performance. Subjects use information about the structure of a task to actively control their momentary perceptual selectivity and their choice of responses. Thus, the criticism that applied to the Subtraction Method can be used in a related form against the Additive Factors Method: Manipulations that affect the speed of one stage cannot be assumed to have a local effect. The activity of other stages is adjusted to changes in overall performance. These views indicate that information-processing models need to be supplemented with subjects' ability to monitor behavior and exert some type of top-down control.

An explicit model of top-down control of the response process has been proposed to explain inhibitory and adaptive behavior. Logan and Cowan (1984) investigated response inhibition with the stop-signal paradigm (cf. Lappin & Eriksen, 1966; Logan, 1994). In this paradigm an RT task is occasionally interrupted by a stop signal with a variable onset asynchrony relative to the response signal. The instruction is to respond as fast as possible on all trials, but to try to stop the response if the stop signal occurs. Although several authors (Lappin & Eriksen, 1966; Logan, 1981; Osman, Kornblum, & Meyer, 1986; Vince, 1948) have suggested some sort of a race between a stop and a response process, a formal race model for the interpretation of stop-signal results was developed by

Logan and Cowan (1984). The model simply holds that the onset of a stop-signal is the start of a stop process that engages in an independent race against the response that is in preparation. Although there has been some controversy about the locus of the finish line of this race (De Jong, Coles, Logan, & Gratton, 1990; Osman et al., 1986; 1990), the general idea is that the process that finishes first determines whether a response is withheld or not. Assuming that the race is truly independent holds that 1) the duration of the go and stop process are not correlated, and 2) the go process has the same properties, regardless whether or not the stop signal is presented. Based on these assumptions, the model provides methods for estimating the speed of the stop process, among other measures.

Logan and Cowan (1984) described inhibition as a form of executive control, and argued that control processes have an hierarchical relationship to the response process. In hierarchical theories the executive system monitors the consequences of its commands to subordinate systems. An act of control from the executive system has privileged access to the response process and does not suffer from the type of capacity limitations that are typically found for the processes in subordinate systems. Because subordinate systems would stop if the orders from the executive system are countermanded, the response can be stopped at any point in the reaction process. In conclusion, Logan and Cowan's (1984) theory of control implies that the response process and the stop process run in an independent race, and that the point of no return lies close before the execution of the response.

Although the stop command is assumed to have privileged access, adaptation of responses requires more than just a countermanding order from the executive system. A delay of adaptation may occur if a response has to be replaced by a sufficiently different response. De Jong, Coles and Logan (1995; see also De Jong et al., 1990) employed the stop paradigm to investigate inhibitory efficiency in stop conditions and in conditions where a response was to be suppressed in favor of an alternative response. They found that the speed of stop-all conditions was higher than the speed of stop-change conditions. Based on psychophysiological indices of central selective preparation for the response hand (LRP), and of response execution (EMG), they concluded that nonselective inhibition can be exerted with high speed at a level close to response execution, whereas selective inhibition, as required in the change condition, needs to be exerted at a central level, and is therefore much slower.

Development

One of the easiest RT effects to replicate, is that speed increases during childhood, reaches a peak for young adults, and gradually decreases for the elderly. This consistent pattern can be found for a variety of RT tasks, ranging from the easiest two-choice RT tasks to tasks that require complicated mental acts, such as mental rotation. It has been found in meta-analyses across task domains (e.g. Hale, 1990), that in a scatter plot with RTs of children as a function of the RTs of young adults on the same tasks, the data points follow an almost linear pattern. Linear regression functions through these data reach R^2 's of at least .9. It has been concluded from this strong relationship that the development of speed is the same for all mental subprocesses. This view implies that one global mechanism underlies all age changes, and that there are no subprocesses (cf. stages) that deviate from the generalized change in speed by developing faster or slower than others. Kail and Salthouse (1994) proposed a clock-speed metaphor, derived from computer technology, as a model for a global mechanism that changes with development, and that affects the speed of all subprocesses to the same extent. In this model, a given subprocess requires a number of processing steps, and the speed of each step is determined by the clock speed. As the clock speed increases with age, the duration of task performance becomes shorter. However, the relationship between

child RT and the adult RT remains the same, because the same architecture underlies the performance of both groups.

In apparent conflict with the global-development hypothesis, there is a body of literature about single cognitive functions that show deficiencies for children relative to adults. Most striking are reports of RT effects due to changes with age in selective attention (Enns, 1990; Lane & Pearson, 1982), the ability to resolve a response conflict (Ridderinkhof, 1993), and resistance to interference (Dempster, 1992). Although the findings suggest that there are processes that escape the generalized pattern of speed changes with age, the difference between these findings may not be as large as it seems. If the domain of tasks with specific age effects is considered, it can be argued that they are all part of the ability to select what is relevant and suppress what is irrelevant. In that case, the idea of a global mechanism could be maintained under the hypothesis that this mechanism consists of the ability to inhibit irrelevant information.

Bjorklund and Harnishfeger (1990) reviewed some of the more prevailing cognitive developments and accommodated them in a larger theory about the efficiency of inhibition. They argued that the age-related changes in the efficiency of inhibition are responsible for findings about attentional deficiencies, because inhibition serves to keep task-irrelevant information out of the working memory. Dempster (1992) showed that there is a large similarity between children and patients with frontal-lobe lesions in the pattern of performance deficits on interference-sensitive tasks. He combined information about frontal-lobe functions with the view on resistance to interference, and argued that this synthesis is “a step toward a unitary theoretical explanation of diverse expressions of cognitive development and aging” (p. 65).

The idea that inhibition affects the speed on a variety of tasks is consistent with neuropsychological insights (Fuster, 1989; Welsh & Pennington, 1988). During the development of the brain, some areas are faster to mature than others. One of the areas that mature the slowest, is the prefrontal cortex. The myelinization in this area, which supports the speed of information transmission, continues until adolescence. Three important functions are ascribed to the prefrontal cortex. First of all, the prefrontal cortex provides a representation of the strategies and input to a task in the form of a working memory. But in order to keep up the efficiency of the working memory, two other functions are required. Relevant information needs to be recruited as part of the preparatory set, and irrelevant information needs to be removed or suppressed in order to prevent interference.

The theory that a general inhibitory efficiency changes with age and affects performance on interference-sensitive tasks is tested in this dissertation. The alternative view is that the performance on some inhibition tasks develops by a different time course than the performance on other tasks.

Organization of the Dissertation

In this dissertation, the response process is primarily investigated with choice-RT tasks. Response bias, interfering information and changes in the instruction are added to the basic choice-RT task, to challenge subjects to exert top-down control on the response process. A comparison of RTs is then made to illustrate how subjects deal with such error-prone situations, and to investigate which factors determine the success or failure of top-down control.

Chapter 2 discusses four experiments performed by 5-year-olds, 8-year-olds, 11-year-olds and young adults. The unifying property of these tasks is that a primary visual stimulus calls for a response, and a secondary auditory stimulus interferes with the normal response process. Inhibition is required for the correct response in all of these tasks. Manipulation of the direction of a tone causes bias for a response with the ipsilateral hand. This bias is not supposed to cause errors, and should therefore be held in check. In an immediate-arousal experiment, the intensity of the tone stimulates subjects to respond immediately. Again, a reasonable accuracy can only be maintained if

incorrect preparation is controlled. In the third and fourth experiment the tone served as signal to stop the response that was in preparation, or replace it by the opposite response. The speed of stopping was compared between age groups. The theory of Dempster (1992) leads to the prediction that young children are less efficient in these response-inhibition requirements than are older children and adults.

Chapter 3 deals with the question to what extent subjects can apply an instruction to exert top-down modulation on the response process. It describes how an increased probability that response primes are valid can be taken into account for the strategic preparation of a response. The inhibition of incorrect preparation is investigated in two conditions that are thought to require different inhibitory processes. Based on De Jong et al.'s (1995) distinction between selective and nonselective stopping, the inhibition on no-go condition is predicted to take place until just before the response, whereas inhibition on change trials is expected to take place at a central level preceding or during the production of LRP.

In Chapter 4, the structural limitations to response selection and preparation were investigated. Subjects were instructed to base performance on a combination of two dimensions of a character. The identity mapped onto hands, and a go/no-go decision was based on whether the character was a normal or mirror image. Because the characters were rotated over a variable angle relative to the upright position, the mirror-normal discrimination could only be made after subjects mentally rotated the character. In the meantime, the identity of the character could be translated into a code of the correct response hand. Because the mental rotation requirement is a heavy mental load, parallel activation of the response was predicted to be hampered.

In Chapter 5 we investigated the performance of the horse-race model as a tool for the interpretation of stop-signal results under normal and deviating characteristics of the response and stop speed. Because it is not possible to study this in detail by empirical measurements, we performed extensive computerized simulations of stop-signal behavior. The simulations were used to test the consequences of violating the independence assumption and the consequences of differences in speed characteristics for the observed measures that are commonly used in the stop-signal paradigm.

Finally, Chapter 6 provides a summary of the data and concludes that top-down effects can be exerted on several points in the response process, as is suggested by Logan and Cowan (1984). In contrast to popular views about the importance of inhibitory control for cognitive development, several forms inhibition are concluded to be quite efficient early in life.

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2 The Ability to Inhibit and Activate Speeded Responses: Multifaceted Developmental Trends¹

Abstract

Inhibitory control and response activation were tested as candidate concepts for a global mechanism that influences the speed of information processing on a variety of tasks, and that changes in efficiency with age. Children aged 5, 8, or 11, and young adults were tested on four reaction-time tasks with largely overlapping designs. Children were hardly more sensitive than adults to interference from irrelevant direction cues and intensive auditory stimulation. The speed of withholding responses was only slightly slower for young children than for older children and adults. These results argue against an important role for inhibitory efficiency in determining the speed of responding. In contrast, the development of response activation has more explanatory power for age-related changes in reaction time. The prediction from the global-mechanism view, that reaction times on a variety of tasks follow the same decrease with age, was not supported.

Some time ago, Howe and Pasnak (1993) edited a book identifying shifts in our thinking about cognitive development. Two themes that seem to emerge in recent investigations of children's cognitive development are concerned with complementary aspects of speeded information processing: developmental changes in the ability to inhibit (Harnishfeger & Bjorklund, 1993) or to resist interference (Dempster, 1993) and the growing capacity to activate information more quickly (Kail, 1993). The view that inhibition is a major determinant of cognitive development holds that effective performance on many tasks requires the capability to deal with competing information or responses (Dempster, 1993) or, more specifically, to keep task-irrelevant information out of working memory, leaving more mental space available for the processing of task-relevant information (Harnishfeger & Bjorklund, 1993). Furthermore, it is argued that the developmental increase in the ability to inhibit is mediated by maturational changes in the frontal lobes (Dempster, 1993; Harnishfeger & Bjorklund, 1993). The complementary view that the increasing ability to activate information quickly is central to cognitive development is derived from the ubiquitousness of age-related changes in response speed across widely different tasks. Although it is argued that a global mechanism must be the driving force for developmental changes in the speed of responding, the currently available evidence does not seem to allow a compelling conclusion as to the nature of this global mechanism (cf. Kail, 1993; p.112)

Obviously, cognitive development is propelled both by changes in the ability to inhibit task-irrelevant information and the ability to activate task-relevant information. The present study will be concerned with both aspects of development operating in speeded information-processing tasks. Broadly defined, the term inhibition has a long history (MacMillan, 1996), may refer to a great variety of phenomena (e.g., Dempster, 1993), and seems to apply to many levels of functioning (e.g., Clark, 1996; Logan, 1994). The hypothesis that inhibition, loosely defined, strengthens during development is primarily based on reviews of children's performance on interference sensitive tasks. Harnishfeger and Bjorklund (1993), for example, pointed to the growing ability to inhibit motor responses revealed by tasks inducing some sort of response competition (e.g., Diamond,

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1988), the decrease in the susceptibility to external distractors present in selective-attention tasks (e.g., Lane & Pearson, 1982), and the capability to suppress internal distractors activated during the performance of tasks placing a demand on working memory (e.g., Harnishfeger & Bjorklund, 1993). It is important to ask whether the developmental improvements on these interference-sensitive tasks are all mediated by a unitary mechanism or must be due to a variety of distinctive processes, each of which is specialized to deal with different aspects of interference. The position adopted by Harnishfeger and Bjorklund (1993) seems to implicate a single mechanism responsible for screening out irrelevant information from working memory. In this vein, these authors reframed mental-resource theory by suggesting that developmental changes in the amount of processing capacity that can be allotted to task performance varies with the ability to inhibit task-irrelevant information. Dempster's (1993) analysis of the literature led him to propose that developmental changes in inhibitory ability reflects a common underlying mechanism that is mediated by the frontal lobes. But this author is not willing to reject the notion of inhibition as a multifaceted process. He argued that inhibition may well vary along several dimensions and suggested that changes in the ability to inhibit follow different developmental trajectories depending on the dimension involved in the interference-sensitive task. Thus, the ability to inhibit responses was assumed to develop rapidly during early childhood whereas the resistance to perceptual interference was thought to follow a prolonged developmental course into adolescence. At this point, Dempster (1993) concluded that current evidence cannot decide between a single and a multiple-mechanisms interpretation of the ability to resist interfering information.

The first central focus of the present study is whether increased resistance to interference is the manifestation of a general inhibitory mechanism, as suggested by Harnishfeger and Bjorklund (1993) or whether developmental changes in the ability to inhibit should be better explained in terms of a subset of related inhibitory mechanisms that may or may not change in parallel, a possibility submitted by Dempster (1993). This question has been addressed by adopting an experimental strategy derived from Kramer and colleagues who raised the same issue in the study of cognitive aging (Kramer, Humprey, Larish, & Logan, 1994). These authors reviewed the literature suggesting that the cognitive decline in later life is the results of a progressive and generalized slowing of information processing. More specifically, they pointed to the hypothesis proposed by Hasher and Zacks (1988) suggesting that the age-related decrease in the speed of information processing is due to the decline of inhibitory ability during aging. Incidentally, the Hasher and Zacks (1988) hypothesis laid the foundation for Harnishfeger and Bjorklund's (1993) interpretation of developmental changes in information processing during infancy and childhood. The issue of a general mechanism versus a subset of inhibitory processes arose also in the cognitive-aging literature, and Kramer and co-workers decided to address this question by examining the performance of the same group of young and elderly adults using a variety of different paradigms that are supposedly revealing different inhibitory mechanisms. They obtained inhibitory deficits on some tasks (Wisconsin card sorting task and stopping tasks) but not on others (negative priming task, Eriksen flanker task and cognitive failures questionnaire). Moreover, there was little evidence for associations among inhibitory indices acquired in the different tasks. These findings led Kramer and colleagues to conclude that age-related inhibitory failures are specific rather than general in nature (cf. Kramer et al., p. 47).

The strategy used by Kramer et al. (1994) was adopted in the current study to perform an even more stringent test of the hypothesis that developmental changes in the ability to inhibit are manifestations of a single underlying mechanism. Four age-groups, rather than two groups of subjects, participated in the study to obtain a larger resolution of developmental changes. Furthermore, subjects performed in one standard paradigm, rather than on widely different tasks, in order to create optimal conditions for a single inhibitory mechanism to reveal itself. The standard

task was a visual two-choice reaction task in which on some or all trials an auditory accessory was presented at various times shortly after the onset of the respond signal. The major difference between tasks was the instruction to the subjects. In two tasks, subjects were instructed to ignore the task-irrelevant auditory accessories. In one task, the auditory accessory varied in intensity from soft to loud tone bursts. It was anticipated that loud tone bursts would be more disruptive to the visual choice process compared to soft tones and that young children's performance would be hurt more by the distractors compared to young adults. In the other task, the auditory accessories were presented either monaurally or to both ears so as to communicate a spatial location. It was predicted that the processing of the location of the tone would interfere with the directional information provided by the visual reaction stimulus, and that young children would be more sensitive than adults to the task-irrelevant spatial information associated with the accessory. In the two other tasks, the auditory accessory was task-relevant. In one task, the tone required the subject to refrain from responding to the visual reaction stimulus whereas in the other task the tone required changing from the compatible to the incompatible response to the visual reaction stimulus. It was assumed that the task-relevant auditory accessories would require an inhibitory mechanism that is supposedly less efficient in children compared to adults. Within the scheme proposed by Harnishfeger and Bjorklund (1993), the former tasks would belong to the category "Inhibition to External Distractors" whereas the latter tasks are instances of the category "Inhibition of Motor Behavior". For both schemes, Harnishfeger and Bjorklund (1993) amassed evidence to suggest progressive development of inhibitory control.

The second major focus of the present study is concerned with developmental changes in response activation. There is an abundant literature suggesting developmental improvement in response speed (for a review Cerella & Hale, 1994). Most interesting, it seems that the growth functions are similar across a wide variety of speeded response tasks. For tasks such as same-different decisions, two or four choice reactions, multiplication, mental rotation, word recognition, and visual search, the results are the same: a progressive increase in the speed of responding. These observations seem to implicate some global mechanism that limits the speed of all processes activated in speeded response tasks. As this mechanism matures, all processes are executed more quickly. The suggestion that developmental changes in the speed of information processing are mediated by a single, global mechanism receives strong support from meta-analytic studies. Kail (1993) reviewed the literature and found that the relation between children's and adults' reaction times (RTs) could be expressed as $Y=mX$, where Y and X are children's and adults' RTs and m is a slowing coefficient. He demonstrated that, across a wide variety of tasks, children's RTs can be expressed as a multiple of adults' RTs and that the m parameter changes exponentially with age. Kail used the clock speed of a microcomputer as a metaphor for the global mechanism underlying age-changes in the speed of information processing (e.g., Kail & Salthouse, 1994). Following this reasoning, individuals who differ in 'clock speed' do not differ in terms of the structural components that constitute the reaction process but only with regard to the pace at which the components are operating. Thus, as young children are growing older their clock speed will be gradually increasing and this will lead to a more rapid execution of all processes that can be activated during speeded response tasks.

It should be noted, however, that the notion of a global mechanism has been criticized on many counts, ranging from methodological issues to demonstrations of differential age changes (for a review see Bashore, 1994). It seems that the regression-analytic procedures, commonly used in the meta-analytic literature, may conceal task-dependent and process-specific age changes in processing speed that can be revealed using ANOVA techniques (for an example see Van der Molen & Ridderinkhof, in press). Moreover, simulation studies have shown that models invoked to explain age-related changes in processing speed may not be able to distinguish between global and local

effects on the reaction process (Molenaar & Van der Molen, 1994). The second aim of the present study, then, is to carefully compare age changes in the speed of responding across the tasks selected for examining inhibitory control. Uneven developmental trends would provide strong evidence for differential growth given the paradigmatic similarity of the reaction tasks.

Experimental Overview

The primary aim of the current study was to determine whether developmental changes in the ability to inhibit are general or specific in nature. In order to provide a strong test of the hypothesis that inhibitory control refers to a general mechanism, the same subjects performed on four interference tasks having a considerable paradigmatic similarity. The subjects were from four age groups ranging from early childhood to young adulthood. The four tasks were all signalled two choice tasks in which a left-appearing stimulus required a left-hand button press and a right-appearing stimulus a right-hand button press. On some or all tasks, a tone was presented with short delays after the onset of the visual stimulus. The stimulus onset asynchronies (SOAs) were determined individually based on the performance on two additional tasks; a visual two-choice task without auditory accessories and an auditory simple reaction task in which the subject responded with the preferred hand.

The first task selected to examine the ability to inhibit involved a manipulation of immediate arousal. In the context of the study of the psychological refractory period, it has been observed that auditory accessories may exert an immediate arousing effect on response preparation (e.g., Bertelson & Tisseyre, 1969). More recently, it has been established that strong auditory accessories (> 80 dB) facilitate response speed in simple reaction tasks but not in choice reaction tasks (for a review see Sanders, 1990). In choice reaction tasks, strong auditory accessories have been found to exert a detrimental effect on choice RT together with an increase in error rates (e.g., Van der Molen & Keuss, 1979; 1981). This pattern has been interpreted to suggest that the auditory stimulus elicits an immediate arousing effect on motor activation (e.g., Sanders, 1977). Immediate arousal has a positive effect on performance whenever response preparation is low (e.g., when time uncertainty is high) and response-selection requirements are minimal (e.g., in simple or highly compatible choice tasks). The immediate-arousal effect must be inhibited, however, when response activation is already high (e.g., when warnings precede respond signals) and response selection is more difficult (e.g., when signals require incompatible responses). It is predicted that the immediate arousing effect of loud accessories will facilitate response speed in children at the cost of higher error rates. In contrast, adults will make an attempt at inhibiting immediate arousing effect to reduce the obvious danger of committing errors. In other words, the slope of the function relating the intensity of the auditory accessory to visual choice RT will be steeper in children compared to adults. This prediction is based on (i) response preparation is lower in children than adults (e.g., Wickens, 1974) providing more room for immediate arousal to exert its facilitating effect on motor activation, and (ii) the ability to inhibit the immediate-arousal effect to reduce error rates is less developed in children than adults.

The second task is a lateral-interference paradigm, popularized by Simon (1990; for a review). In this task, subjects are asked to perform the same signalled two-choice task and again, auditory accessories are presented shortly after the onset of the visual reaction signal. But now the accessory is presented monaurally to the left or right ear or binaurally. It is commonly observed that response speed is prolonged when the location of the tone does not correspond to the location of the responding hand (e.g., left-ear accessory while the visual stimulus requires a right-hand response) relative to corresponding (e.g., left-ear tone and left-hand response) or neutral (i.e., binaural presentation) trials. This pattern has been interpreted to suggest that the accessory might elicit a

natural tendency to respond to the auditory stimulus source that may interfere with responding to the visual stimulus (e.g., Simon & Craft, 1970). The locus of interference seems to be at the level of response selection (e.g., Stoffels, Van der Molen, & Keuss, 1985; see also Hommel, 1995) or just prior to that stage of the choice reaction process (e.g., Stoffels & Van der Molen, 1989). Given that children are more sensitive to interference, it is predicted that they experience greater difficulty in suppressing the tendency to respond to the auditory stimulus source. Hence, the RT difference between noncorresponding and corresponding or neutral trials will be more pronounced than in adults.

The third task differed from the preceding immediate arousal and lateral-interference tasks in two respects. First, the auditory accessory is presented on only a proportion of trials – not on all trials. Second, and more importantly, the auditory accessory is now task-relevant by instructing the subject to withhold the overt response to the visual stimulus. Obviously, when the auditory stop-signal is presented early, it is very easy to refrain from responding but when it is presented late, it might be impossible to countermand the reaction to the visual reaction signal. The stopping task has been well researched by Logan and colleagues who devised a methodology for examining the latency of the internal response to the stop signal (e.g., Logan & Cowan, 1984). The methodology is based on a simple horse-race model of auditory stop-signal and visual reaction-signal processing. If the stopping process to the auditory signal finishes earlier than the visual reaction process, the response is inhibited, but if the visual reaction process is faster, then the response will be executed. It appears that the horse-race model accounts for the data very well (for a review see Logan, 1994). All that is assumed is that the stopping and visual reaction processes are independent. For a convenient calculation of the stop latency, it is helpful to assume that this speed is constant. The latency of the internal stopping process can be computed quite easily.² For young adults, stop-signal RT is approximately 200 ms across a wide variety of tasks (for a review see Logan & Cowan, 1984; Logan, 1994). Stop-signal RT is longer in older adults (Kramer et al., 1994) and hyperactive children (for a review Oosterlaan, 1996; see also Schachar, Tannock, Marriott, & Logan, 1995). Stopping is also slower in children but Schachar and Logan (1990) failed to obtain statistical support for developmental change and the age range (8 to 12 years) studied by Oosterlaan (1996) might have been too limited. The purpose of the current study is to re-examine developmental changes in stopping RT by using a wider age range (5 to 20 years) and more subjects to increase power.

In addition to the latency of the internal response to the stop signal, the stopping paradigm provides an inhibition function, of which the slope has been taken as an index of inhibitory control. The inhibition function relates the probability of inhibition to the time interval extending between the presentation of the stop signal and the overt response to the visual reaction stimulus. Differences in the ability to inhibit will affect the slope of the inhibition function. In general, the slope of the inhibition function will be steeper when the inhibition mechanism is more efficient. If the inhibition mechanism of young children is slower or less likely to be triggered, the inhibition function will be

² Given the assumption that the processing of the stop-signal and visual reaction signal are independent, the RT distribution of the visual reaction process obtained for trials on which no stop-signal was presented can be considered as the RT distribution of the visual reaction process from trials on which a stop-signal was presented. Assuming also that the latency of the stopping process is constant, the internal RT to the stop-signal can be seen as a point on the time axis of the RT distribution. At a given finishing time of the stop-signal, all responses to the right of this point are inhibited because the stopping processes finished first and all responses to the left of this point are executed because the visual reaction process finished first. The proportion of trials on which the response escaped inhibition is equal to 1 minus the probability of inhibition. The internal RT to the stop-signal can now be computed by rank ordering all visual RTs to determine the n th fastest value, where n is the number of RTs in the distribution multiplied by 1 minus the inhibition probability. The obtained value provides an estimate of the finishing time of the stop-signal, relative to the onset of the visual reaction signal. Stop-signal RT is then estimated by subtracting out the delay of the stop-signal (for details see Logan & Cowan, 1984).

flatter than those of older age groups (cf. Schachar & Logan, 1990; p. 711). It should be noted, however, that the slope of the inhibition function also depends on the mean and variability of the visual choice reaction process. The model of Logan and Cowan (1984) provides a method for correcting inhibition functions for differences in stopping and the visual choice reaction process. Basically, the method implies plotting the probability of inhibition as a function of the Z-score that represents the interval between stopping completion and overt responding in standard-deviation units of the visual choice reaction process. If the inhibition functions from different age groups cannot be aligned after this correction, it is assumed that flatter functions indicate less efficient inhibitory control (cf. Schachar & Logan, 1990; p. 712).

A final task that has been included is referred to as the stop-change paradigm in the literature (Logan, 1994). In the stop-change task, the auditory accessory requires the subject to inhibit the visual reaction process and to make an overt response to the stop signal. Logan and Burkell (1986), for example, asked their subjects to respond with the index and middle fingers of their right hand to indicate that the visual reaction stimulus was respectively an A or a B versus a C or a D. The stop signal required them to inhibit their response to the letters and to respond as quickly as possible to the tone with the index finger of their left hand. They observed that stop-change RTs were considerably longer than stopping RTs. Logan and co-workers tested hyperactive children on the stop-change paradigm and replicated their previous findings showing that stopping was slower than that of control children. Interestingly, they observed that hyperactive children experienced greater difficulty in changing to an alternate action after the inhibition of the visual choice-reaction process (Schachar et al., 1995). Similar findings have been reported by Oosterlaan and Sergeant (1996). The developmental study by Oosterlaan (1996) indicated that stopping in the stop-change task did not improve with age from 8- to 12-years of age but response re-engagement did: With age, children were better to execute an overt response to the stop signal.

For the present discussion, the results obtained in a psychophysiological study conducted by De Jong and colleagues are very important (De Jong, Coles, & Logan, 1995). In this study, subjects performed on stopping and stop-change tasks and brain-potential measures were taken to provide an index of preferential response activation. The results of the stopping task were consistent with a previous study (De Jong, Coles, Logan, & Gratton, 1990) in showing that subjects were able to successfully inhibit the overt response to the visual reaction stimulus while response activation had reached full criterion at central level as indexed by the brain-potential measures. This finding was interpreted to suggest the existence of two inhibition mechanisms: a relatively slow mechanism located at the central level and a rapid mechanism at a more peripheral level. If responses escape inhibition at the central level (as suggested by full-blown brain-potential amplitudes), they might be stopped by the inhibition mechanism located downstream (see also Jennings, Van der Molen, Brock, & Somsen, 1992). In the stopping task, inhibition might rely on the fast peripheral mechanism but in the stop-change task subjects are required to inhibit selectively responses to the visual reaction stimulus without impeding the response to the stop signal. The additional requirement to execute an alternate response should prevent the use of the rapid (stop-all) peripheral inhibition mechanism and thus requires the recruitment of the slower (stop-selective) central mechanism. Indeed, De Jong and colleagues observed longer latencies to the stop signal in the stop-change task in addition to reduced brain-potential amplitudes. Both findings are suggestive of the action of the central inhibition mechanism. Given the existence of two inhibition mechanism, one slow but selective and located at the central brain level and the other rapid but indiscriminate located at the midbrain level, these mechanisms develop at different rates. More specifically, it is hypothesized here that the crude, peripheral inhibition mechanism matures at a more rapid pace than the refined, central inhibition mechanism. The stop-change task employed in the current study was somewhat different from the tasks commonly used in that the stop signal required a change in

the response to the visual reaction stimulus rather than a response to the tone. It is predicted that stop-change RTs are slower than stopping RTs and, most importantly, that the developmental trend observed for re-engagement is more pronounced than the age-related change in stopping.

Method

Subjects

Three groups of primary school children and one adult age group participated in the study. The youngest age group consisted of 16 five-year-old children (M age = 5 years and 5 months, SD = 5 months), the middle age group consisted of 16 eight-year-old children (M age = 8 years and 6 months, SD = 5 months), and the oldest age group of children consisted of 16 eleven-year-olds (M age = 11 years and 8 months, SD = 7 months). The groups of children were half male and half female. The children, who were predominantly suburban and middle class, were recruited with the help of their teachers and participated with parental consent. They received a small gift for their participation. The adult group consisted of 21 psychology students (M age = 21 years and 7 months, SD = 2.4 years). Nine students were male and 12 were female. They received course credits for their services.

Apparatus and Stimuli

All tasks were performed on a Macintosh SE/30 computer with a black-on-white screen. Subjects responded using a response panel with two high-precision push buttons (travel times < 1 ms) connected to the Macintosh computer. Subjects were seated in a small, comfortably lit room at a distance of approximately 80 cm in front of the computer screen. In all paradigms, a trial was started with the onset of a warning stimulus consisting of two squares, sized 24 mm in width and presented 42 mm apart. The warning stimulus was followed by the respond stimulus with intervals randomly varying between 800 and 1200 ms. In the simple reaction task, the respond stimulus consisted of a binaurally presented white-noise stimulus with an intensity of 85 dB(A) and 400 ms duration. In the choice-reaction paradigms, the respond stimulus consisted of the appearance of a line-drawing of a clown's head just above one of the squares. The head of the clown was 38 mm in width and 45 mm in height (i.e., subtended over three degrees of visual angle). The visual reaction signal appeared within one screen refresh-cycle and was response terminated or, in case no response was emitted, disappeared after 2300 ms. This maximum duration was reduced to mean RT plus five standard deviations after the first measurements of these values on a visual choice task. The onset of the visual reaction signal could be followed by a secondary, auditory signal of 400 ms duration and with a stimulus onset asynchrony (SOA) determined on the subject's speed of responding and an estimate of the individual's processing time of the auditory signal. The auditory signals were presented by size-adjusted, padded headphones. The subject's response (or its inhibition) was followed by a feedback stimulus consisting of a smiling (correct response) or a mocking (incorrect) face of the clown.

Procedure

Subjects performed their task in two sessions of two hours on consecutive days. The first session began with the performance of the spatial choice and simple reaction time tasks to provide an estimate of the processing speed of the auditory signal used to determine the SOAs of the secondary signal in the immediate arousal, lateral interference, and stopping paradigms. Following the auditory simple, and the visual choice tasks, subjects received practice and test blocks of one of the other paradigms. The first block of trials and the first five trials of each test block were for practice and were discarded from data analysis. After each block of trials, subjects received detailed information on their performance and were instructed to maintain optimal speed and accuracy levels for the remainder of the session. During the second session, subjects performed on the remaining paradigms. The order of the paradigms was counterbalanced across subjects. The details concerning the simple reaction, choice reaction, and other paradigms will be provided below.

Experimental Tasks

Auditory simple reaction task.

In this task, the visual warning stimulus was followed by an auditory respond stimulus. Catch trials were included to prevent unacceptable levels of anticipations. These trials occurred with a probability of 10% and consisted of warning stimuli *not* followed by the respond signal requiring subjects to inhibit their motor response at the time of predicted stimulus occurrence. Subjects responded to the auditory signal with their preferred hand and received three blocks consisting of 55 trials each. The first block was practice and the first five trials of the second and third blocks were for warm-up. There was a short intermission between trial blocks. Subjects were instructed to go as fast as they could but prevent commission errors and premature responses. If subjects responded on catch trials or prematurely on go trials they received negative feedback; a traffic stop sign.

Visual choice reaction task.

In this task, the visual warning stimulus was followed by a visual reaction stimulus – the clown's head was presented either above the left or the right square. Subjects were told that they would see two boxes first (i.e., the two squares presented in the warning screen) and then a clown would jump out of one of the boxes (i.e., the appearance of the clown's head in the respond screen). Their task was to press the response button corresponding to the location of box where the clown appeared. Both speed and accuracy were emphasized. Three blocks were presented, each consisting of 55 trials. The first block was practice and the initial five trials of the test blocks were for warm-up.

Immediate-arousal task.

Binaural noise accompanied the visual reaction stimulus on all trials. The noise had a fixed duration of 400 ms but varied in intensity from trial to trial on a pseudo random basis; 65, 85, and 105 dB(A). There were four SOAs; zero and three SOAs determined on the subject's performance on the auditory simple and visual choice tasks. The SOAs were the 20th, 50th, and 80th percentile times of the visual choice RT distribution minus the mean of the auditory simple RT distribution. Lower bounds were set on 17, 33, and 50 ms for SOA 2, 3, and 4, respectively. In each trial block, individual combinations of the four SOAs and three trial types were presented six times in a pseudo-random order. Subjects received six blocks of 72 trials. The first trial block was for practice and the first six trials of each test block were for warm-up. An additional practice block was given when accuracy fell below 90% or when RT was consistently above the mean obtained in the visual choice reaction task. Instructions emphasized speed and accuracy. The duration of trial blocks was approximately 5 min and there were short rests between trial blocks. Subjects were instructed to respond as quickly and accurately as possible to the visual reaction signal and to ignore the auditory noise.

Lateral interference task.

The onset of the visual reaction signal was followed by monaurally or binaurally presented white noise with a 400 ms duration and 85 dB(A) intensity. The presentation (binaural or monaural) and location (left or right ear) of the noise was varied from trial to trial to create three trial types: Congruent trials on which the location of the noise corresponded to the location of the button press (e.g., left-ear stimulation and left-hand button press or right-ear stimulation and right-hand button press), incongruent trials on which the location of the noise and the location of the response was at contralateral sides of the body (e.g., left-ear stimulation and right-hand button press or right-ear stimulation and left-hand button press), or neutral trials on which the noise was presented to both ears. The auditory noise was presented using four different SOAs according to the same procedure as in the immediate-arousal task. In each block, individual combinations of the four SOAs and three trial types were presented six times in a pseudo-random order. Subjects received six blocks of 72 trials. The first trial block was for practice and the first six trials of each test block were for warm-

up. An additional practice block was given when accuracy fell below 90% or when RT was consistently above the mean obtained in the visual choice reaction task. The duration of trial blocks was approximately 5 min and there were short rests between trial blocks. Subjects were instructed to respond as quickly and accurately as possible to the visual reaction signal and to ignore the auditory noise.

Stopping task.

Subjects performed the visual choice reaction task described above. On a small proportion of the trials, however, an auditory stop signal followed the onset of the visual reaction stimulus requiring the inhibition of the motor response to the visual stimulus. The auditory stop signal was white noise with a duration of 400 ms and an intensity of 85 dB(A). It was presented on 30% of the trials using three different SOA conditions – early, medium, and late – determined using a staircase tracking algorithm (Levitt, 1970). This technique allows to set stop-signal delays by tracking subjects' inhibition performance. Thus, stop-signal delay was increased for early SOA trials by 17 ms every time the subject inhibited and decreased by 33 ms every time the subject responded. On average, subjects should respond on 33% of the short SOA trials and respond on 67% of the trials. The middle SOA increased by 17 ms when subjects inhibited and decreased by 17 ms when they responded. For middle SOA trials, subjects should respond on half of the trials and inhibit on half of the trials. The late SOA increased by 33 ms when subjects inhibited and decreased by 17 ms when they responded. Subjects should respond on 67% of the time and inhibit 33% of the time to delays set by this rule. In previous studies, this method has been used successfully to obtain delays at which the probability of responding is between zero and 1 for each subject, and to avoid that subjects prolong their RT to increase the probability of inhibiting (e.g., Osman, Kornblum, & Meyer, 1986; De Jong et al., 1990). In the current study, SOAs were limited to a range of 500 ms preceding and 1500 ms following the onset of the visual reaction stimulus.

Subjects were instructed to respond as quickly and accurately as possible and not to delay their responses awaiting the occurrence of the stop signal. Inhibition failures on stop-signal trials were followed by negative feedback; i.e., the frowning of the clown's face. Subjects received 10 test blocks consisting of 60 trials each, with the first five trials for warm-up. This resulted in 385 nonsignal and 165 stop-signal trials, 55 trials for each SOA. Prior to testing, subjects received a practice block which was repeated until the SOA tracking approached balance.

Stop-change task.

Subjects performed the stopping task described above but the instruction was now to respond on the opposite side of the visual reaction stimulus whenever the auditory signal was presented. The procedures of the stop-change task were identical to the stopping task with the exception that the tracking algorithm was now targeted to the probability of responding on the opposite side.

Finally, it must be noted that the order of the immediate arousal, lateral interference, stopping and stop-change tasks were counterbalanced across subjects. The older subjects performed their tasks in one experimental session, the 5- and 8-year-olds needed two experimental sessions on subsequent days. Their performance on nonsignal trials did not discriminate between sessions.

Results and discussion

The results will be presented in two sections. First, we will discuss each of the tasks separately, focusing on age-related trends in inhibitory function. Second, we examine age-related changes in response speed to assess whether the developmental increase in processing speed is global, affecting all elements of the reaction process indiscriminately, or selective, affecting some processes more than others.

Auditory simple reaction task.

Outliers were removed ($RT < M - 2.5 SD$ and $RT > M + 2.5 SD$) and mean RTs were computed for each subject. In Table 1 we present the mean simple RT for each age group and the slowing coefficients relating children's RT to the speed of responding in the young adults. As anticipated, RT decreased considerably with age, $F(3, 65) = 50.95$; $p < .001$. Post-hoc contrasts indicated that all groups differed significantly from each other. The RT/Age function corresponds closely to the findings reported previously in the literature (e.g., Woodworth, 1938). The slowing coefficients correspond well with the values obtained by Kail (1993) in a series of meta-analytic studies. Across a great variety of studies and tasks, he observed that the slowing coefficients change exponentially with age; from about 3 in 5-year-olds to approximately 1.25 for 12- to 14-year-olds (c.f. Kail, 1993, p. 99). These findings have been interpreted to implicate some global factor in age-related changes in the speed of responding (see also Cerella & Hale, 1994).

Table 1: RTs to Respond Signals. Slowing coefficients are presented between brackets (see text for explanation).

Age Group	5-yrs	8-yrs	11-yrs	adults
Simple RT	414 (2.59)	295 (1.84)	206 (1.29)	160
Choice RT	690 (2.51)	465 (1.71)	335 (1.28)	272
Choice RT (arousal)	693 (2.62)	414 (1.60)	281 (1.11)	245
Choice RT (interference)	664 (2.82)	405 (1.69)	282 (1.15)	253
Choice RT (stop)	742 (2.46)	498 (1.65)	364 (1.21)	302
Choice RT (stop-change)	753 (2.44)	528 (1.71)	361 (1.17)	308
Change RT	1114 (3.12)	667 (1.48)	425 (1.19)	356

Note: All times are in ms. RT is Reaction Time.

The number of premature reactions did not differentiate significantly between age groups, $F(3, 65) = 1.79$, $p > .1$. Mean accuracy was 96.7%. The finding that age groups did not differ in the number of premature responses is rather surprising vis-à-vis notions of inhibition as a major developmental dimension (e.g., Dempster, 1993). The performance of a simple reaction has been described as the execution of a "prepared reflex" (e.g., Woodworth, 1938). The primary demand of the simple reaction task is then the inhibitory control of this prepared reflex. Following inhibition notions of developmental changes in the speed of responding, one would expect that young children are less able to control response preparation than adults and thus commit more premature responses. An alternative interpretation providing a unified account of the simple RT data would be that preparation in young children is less efficient than in adult subjects. Possibly, young children prepare less because they are less able to protect the stimulus-response channel against irrelevant stimuli and/or responses (e.g., Fuster, 1989). Psychophysiological measures of response preparation may provide a more definite test of this interpretation (e.g., Ridderinkhof & Van der Molen, 1995).

Visual choice reaction task.

Outliers were removed and mean choice RT of correct responses is presented in Table 1. The ANOVA with Age Group as between-Ss factor yielded a significant main effect of Age Group, $F(3, 65) = 120.45$; $p < .001$. Post hoc analysis indicated that the differences between age groups were all significant. The developmental decrease in visual choice RT is remarkably similar to the RT/Age function obtained for simple reactions. The slowing coefficients presented in Table 1 are very close to the values obtained for the simple task. This finding is highly compatible with the hypothesis that a global mechanism accounts for developmental changes in processing efficiency across a wide array of speeded performance tasks (e.g., Hale, 1990; Kail, 1991; 1993).

Subjects made few errors; average accuracy was 97.8%. Most likely, the low error rate was due to the highly compatible S-R mapping rule relating the spatial positions of stimuli and responses. Nonetheless, the ANOVA revealed a significant main effect of Age-group, $F(3, 65) = 17.93$; $p < .01$. Response accuracy increased from 96.4% in the 5-year-olds, to 97.8% and 98.4% in respectively the 8- and 11-year-olds, and to 98.8% in adult subjects. Post-hoc analyses indicated that all group differences were significant. The current findings indicate that even 5-year-olds are well able to perform a speeded choice reaction task, albeit less efficient than older children and adults.

Immediate-arousal Task

Error rates exceeded 40% for four subjects; one 6-year-old, two 11-year-olds, and one adult. The data from these subjects were excluded from further analysis. Outliers were removed from the data of the remaining subjects and their mean RTs were subjected to ANOVA with Age Groups (4) as between-Ss factor and SOA (4) and Intensity (3) as within-Ss factor. The ANOVA yielded a significant main effect of Age Group, $F(3, 61) = 81.61$, $p < .0001$. Mean RT for each age group and children's slowing coefficients are presented in Table 1. It can be seen that the RT/Age function is close to the RT gains obtained in the auditory simple and the visual choice reaction tasks.

Response speed increased with the intensity of the auditory noise, $F(2, 122) = 18.50$, $p < .0001$. Visual RT associated with soft tones was 413 ms and decreased to 396 and 385 ms for medium intensity and loud tones. Post-hoc analysis indicated that all intensity differences were significant. This finding is consistent with previous research reporting a negative gradient of the RT/Intensity function when S-R mappings are highly compatible (e.g., Sanders, 1980; Van der Molen & Keuss, 1981). Although the intensity effect was stronger for the 5-year-olds compared to the other age groups (52 ms compared to 22 ms, 27 ms, and 18 ms for, respectively, the 8- and 11-year-olds and the young adults), the interaction between Intensity and Age Group failed to reach significance, $F(6, 122) = 1.39$, $p > .24$.

Response speed decreased with increasing SOAs, $F(3, 183) = 98.38$, $p < .0001$, from 353, to 379, 409, and 450 ms. This finding suggests that subjects timed their response to the visual reaction stimulus vis-à-vis the occurrence of the auditory noise. Young children prolonged their response more than older subjects did, $F(9, 183) = 21.54$, $p < .0001$. The 5-year-olds delayed their response by more than 200 ms whereas the delay was only 30 ms for the young adults. There was a significant interaction between the effects of SOA and Intensity, $F(6, 366) = 5.00$, $p < .001$, but the higher-order interaction with Age failed to reach significance, $F(18, 366) = .61$, $p > .8$. The significant two-way interaction is plotted in the upper panel of Figure 1. It can be seen that the intensity effect is gradually decreasing when the auditory noise is presented with larger delays following the onset of the visual choice stimulus. This finding seems to suggest that the immediate arousal elicited by the auditory noise exerts its effect relatively early during the processing of the visual choice stimulus when response readiness is still progressing towards the motor action limit (Niemi & Näätänen, 1981).

The ANOVA performed on response accuracy yielded significant main effects and interactions. Accuracy increased with Age, $F(3, 61) = 4.73$, $p < .005$, and SOA, $F(3, 183) = 44.45$, $p < .0001$, and decreased with Intensity, $F(2, 122) = 22.04$, $p < .0001$. The accuracy differences between noise intensities and age groups decreased with SOA, $F(6, 366) = 222.09$, $p < .0001$, and $F(9, 183) = 3.97$, $p < .0005$, respectively. The interaction between Age group and Intensity reached significance, $F(6, 122) = 2.33$, $p < .05$. Finally, the higher order interaction between the effects of Age Group, SOA, and Intensity, $F(18, 366) = 11.61$, $p < .0001$, revealed that age differences in accuracy occurred primarily when loud noise was presented at the onset of the visual stimulus. The differences in accuracy between loud and soft noise are plotted in the lower panel of Figure 1 as a

function of SOA and Age Group. It can be seen that the immediate-arousal effect on response accuracy is considerable for young adults and the 11-year-olds when the noise was presented at the onset of the reaction stimulus. The immediate-arousal effect on accuracy progressively diminishes with increasing SOAs. Finally, it can be seen that the immediate-arousal effect is virtually absent for the youngest age group.

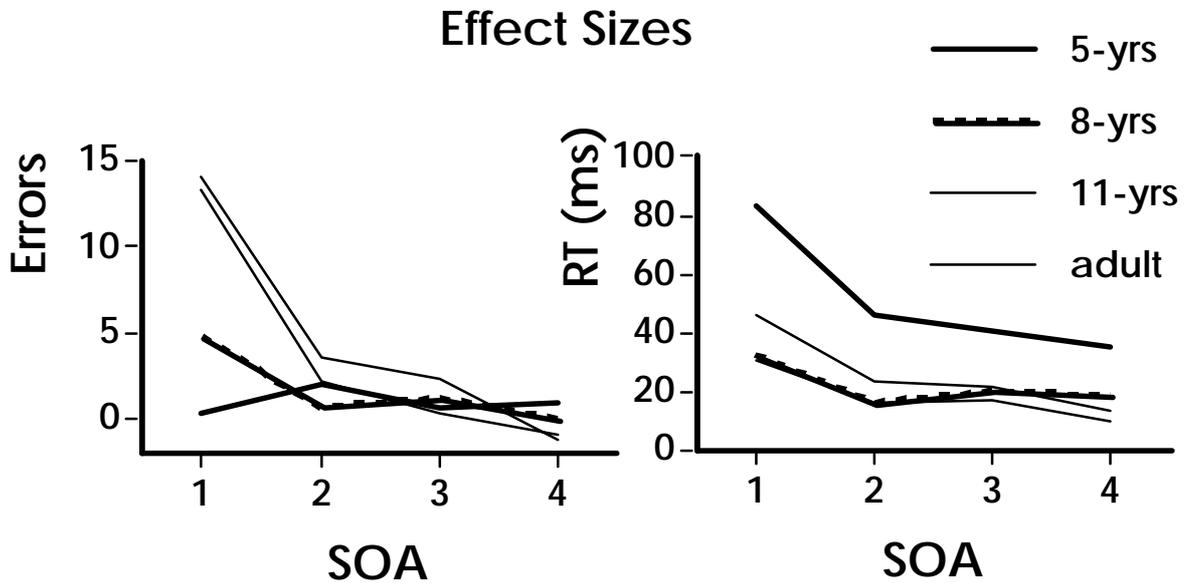


Figure 1: The effect size of sound intensity on error rate (loud minus soft, left) and mean reaction time (soft minus loud, right) as a function of Age and Stimulus Onset Asynchrony (SOA).

The current findings are consistent with previous reports of immediate-arousal effects on response speed and accuracy in adult subjects. Loud auditory accessories are assumed to exert an immediate-arousal effect resulting in increased readiness to respond (e.g., Sanders, 1977; 1980). The increased readiness to respond will accelerate the speed choice reactions at the risk of committing more errors. Indeed, adult subjects responded faster and committed more errors when loud noise accompanied the onset of the visual reaction stimulus. It was predicted that children would be more sensitive to the immediate-arousal effect elicited by loud noise. More specifically, they were assumed to show steeper RT/intensity functions and increased error rates relative to the adults. The results were only partly consistent with this prediction. The RT/Intensity function was somewhat steeper for young children but this effect did not reach significance. Furthermore, for this age group the immediate-arousal effect on response accuracy was virtually absent. A possible explanation of this unexpected pattern of results refers to age-related changes in response preparation. It is long known that young children experience difficulty in predicting stimulus occurrence in speeded information-processing tasks (e.g., Wickens, 1974). Moreover, psychophysiological studies seem to suggest that young children are just waiting for something to happen rather than making an attempt at predicting actively the occurrence of task-relevant stimuli (e.g., Weber, Van der Molen, & Molenaar, 1994). Given that response preparation was relatively low in the youngest children, there was simply more room for loud noise to increase response readiness and less danger for making errors. In this vein, the immediate-arousal findings suggest that the need to inhibit rather than the ability to inhibit is less in young children compared to older subjects.

Lateral-interference task

Five subjects did not perform well on this task. Although their response speed did not deviate significantly from their peers, their error rates approached chance level. Thus, the data from these subjects, two 5- and three 12-year-olds, were excluded from further analysis. Outliers were removed from the data of the remaining subjects and mean RTs were then submitted to ANOVA with Age Groups as between-Ss factor and SOA (4) and Congruence (3) as within-Ss factors.

All main effects were significant. Mean RT for each age-group and the children's slowing coefficients are presented in Table 1. Response speed increased with age, $F(3, 60) = 59.83, p < .0001$. Post hoc analysis indicated that all group differences were significant with the exception of the RT difference between the 11-year-olds and the adult subjects. The slowing coefficients indicate that the developmental gain in the speed of responding is similar to the age-related change obtained in the previous simple and choice RT tasks. This finding is adding to the hypothesis that a global mechanism is involved in age-related changes in processing speed.

As in the immediate-arousal task, RT increased with SOA from 353 ms for the zero delay to 377, 397, and 436 ms for the longer SOAs, $F(3, 180) = 84.79, p < .0001$. Post hoc analyses indicated that all RT differences between SOAs were significant. Finally, responses were slower when the auditory accessory was presented at the ear opposite to the response side (399 ms) compared to presentation at the lateral ear (387 ms) or at both ears (386 ms), $F(2, 120) = 12.25, p < .0001$. Post-hoc analysis indicated a significant RT difference between incongruent versus neutral or congruent trials but not between neutral and congruent trials. The detrimental effect of auditory accessories presented at the opposite ear is consistent with the literature on the lateral interference effect (e.g., Simon, 1990). The failure of monaural tones to produce a facilitatory effect on visual RT when presented at the lateral ear is also a typical finding (e.g., Stoffels, Van der Molen, & Keuss, 1989).

The ANOVA yielded significant interactions between SOA and Age Group, $F(9, 180) = 25.90, p < .001$, and between SOA and Congruence, $F(6, 360) = 8.89, p < .0001$. The higher-order interaction between the effects of Age Group, SOA, and Congruence reached significance, $F(18, 360) = 1.82, p < .05$. The interference effect (i.e., the RT difference between incongruent and congruent trials) is plotted in the upper panel of Figure 2 as a function of SOA and Age Group. It can be seen that interference occurs only when the auditory noise is presented at the onset of the visual stimulus for all but the youngest age group. For the 5-year-olds, interference is also found when the noise is presented with a longer delay. This pattern of results does not necessarily imply that the 5-year-olds were more sensitive to lateral interference. Previous reports have shown that the detrimental effects of irrelevant spatial cues depend on the timing of the processes activated by the spatial cue relative to primary-task processing (e.g., Hommel, 1995). The current findings are consistent with the literature in suggesting that irrelevant spatial cues associated with auditory accessories interfere at a relatively early stage of visual information processing. Given the stronger tendency in the 5-year-olds to prolong primary-task processing there was simply more operating space for irrelevant spatial cues to exert their negative effect on processing speed.

Subjects made few errors; less than 5%. The ANOVA on response accuracy with Age Group (4) as between-Ss factor, and SOA (4) and Congruence (3) as within-Ss factors yielded significant main effects of SOA, $F(3, 180) = 75.43, p < .0001$, and Congruence, $F(2, 120) = 55.50, p < .0001$, but the effect of Age Group failed to reach significance, $F(3, 60) = 1.62, p > .1$. Error rate was higher when the auditory noise coincided with the onset of the visual stimulus and was presented at the contralateral ear. The ANOVA showed significant interactions of SOA and Congruence, $F(6, 360) = 25.51, p < .0001$, Age Group and SOA, $F(9, 180) = 6.99, p < .0001$, and Age Group and Congruence, $F(6, 120) = 2.49, p < .03$. The higher-order interaction between the effects of Age Group, SOA, and Congruence, $F(18, 360) = 3.25, p < .0001$, reached significance. The interference effect on response accuracy (i.e., the difference between incongruent and congruent trials) is plotted

in the lower panel of Figure 2 as a function of SOA and Age Group. For the older subjects, the contralateral noise seems to exert its negative effect on response accuracy when it is presented at the onset of the visual reaction stimulus. Response accuracy of the youngest children seem to be less rather than more affected by the auditory noise.

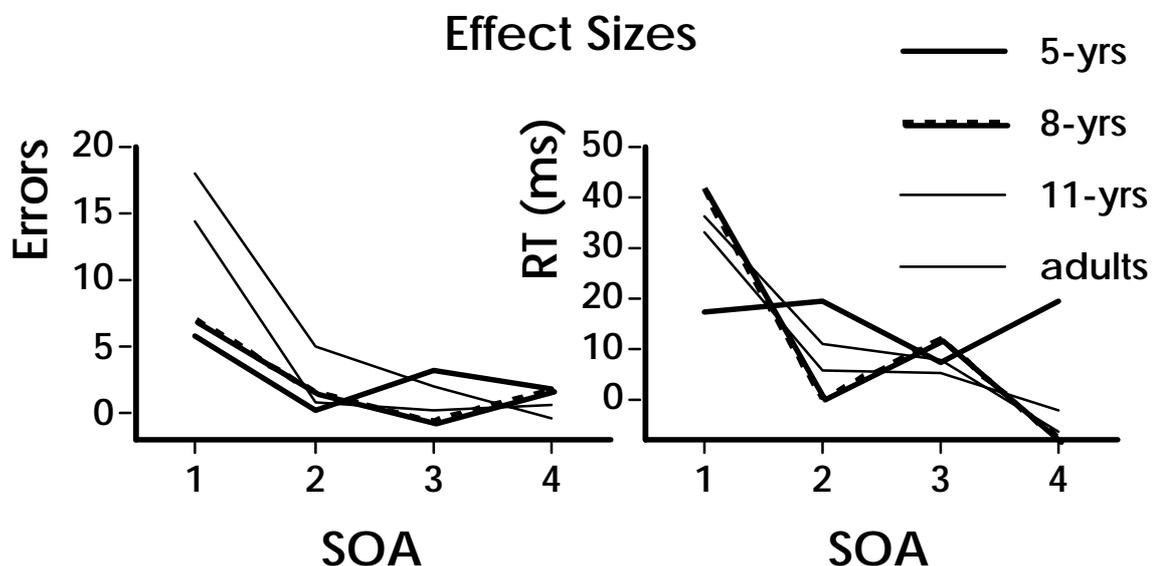


Figure 2: The effect size of congruence of sound location and response side (incongruent minus congruent) on error rate (left) and mean reaction time (right) as a function of Age and Stimulus Onset Asynchrony (SOA).

To summarize, the current findings are consistent with previous reports showing detrimental effects of contralateral auditory accessories on the speed and accuracy of visual choice processing (e.g., Simon, 1990). The findings provide support for the hypothesis that auditory accessories interfere at a relatively early level of visual information processing, most likely prior to the activation of the motor system (Hommel, 1995; Stoffels et al., 1989). If anything, older subjects made more errors than younger subjects when visual stimuli were accompanied by contralateral noise. This finding seems inconsistent with the notion that young children are more susceptible to interference compared to older children and adults (e.g., Lane & Pearson, 1982). Possibly, the reduced risk of making errors is related to a lower readiness to respond in young children (see the reasoning above related to the immediate-arousal effect).

Stopping task

The results of two 11-year-olds were discarded from data-analysis because of their deviant performance. In one boy, omission rate on nonsignal trials was 17.1%, while the group average was only 2.9%, suggesting a strong bias to wait for the stop signal. In another boy, the rate of false-hand errors was 15.2% for late stop signals which is highly deviant from the rest of the group.

An ANOVA performed on the RTs from nonsignal trials yielded a significant effect of Age Group, $F(3, 63) = 76.30, p < .0001$. Mean RT for each age group and children's slowing coefficients are presented in Table 1. Although RTs were somewhat longer, the age-related increase in response speed is similar to the trends observed in the preceding tasks. An ANOVA examining the effects of time-on-task revealed that responses were faster during the second compared to the first half of the experiment, $F(1, 63) = 23.86, p < .001$; 27 ms averaged across age groups. This finding indicates that subjects did not adopt the deliberate strategy to wait for the occurrence of the stop signal. Such

a strategy would be damaging to the assumptions of the horse-race model (e.g., Logan & Cowan, 1984). Error rates were small but the ANOVA yielded a significant effect of Age group, $F(3, 63) = 3.71, p < .003$. Mean error rate was .36% for the young adults and increased to 1.33%, 1.25%, and 1.08% for, respectively, the 11-, 8-, and 5-year-olds.

Table 2. Stop-signal delays, Proportion of Successful Inhibits, Estimated Stop-Signal RTs, and Mean Observed and Predicted RTs for Inhibition Failures.

Age Group	5-yrs	8-yrs	11-yrs	adults
	SOA1			
Stop-signal delay	290	105	37	24
Inhibits (%)	75.1	68.3	66.9	68.6
Stop-signal RT	284	312	283	251
Predicted RT	496	365	280	248
Observed RT	646	462	307	261
	SOA2			
Stop-signal delay	428	199	115	73
Inhibits (%)	56.3	50.6	49.8	51.9
Stop-signal RT	245	280	241	223
Predicted RT	549	396	299	261
Observed RT	627	438	307	265
	SOA3			
Stop-signal delay	459	266	169	123
Inhibits (%)	38.5	33.9	32.9	33.3
Stop-signal RT	336	281	231	201
Predicted RT	601	426	318	274
Observed RT	653	437	326	274

Note: All times are in ms. RT is Reaction Time.

Table 2 presents the results obtained from stop-signal trials. It can be seen that stop-signal delays decrease with Age, $F(3, 63) = 37.43, p < .001$, and increase with SOA, $F(2, 126) = 137.69, p < .001$. The interaction between these effects reached significance, $F(6, 126) = 3.26, p < .01$. The differences in stop-signal delays between SOAs were larger for young compared to older subjects. It should be noted that this interaction is only a consequence of the tracking procedure (see Methods).

Obviously, the proportion of successful inhibits decreased with stop-signal delays, $F(2, 63) = 1299.98, p < .0001$, from 70% for the shortest SOA to 52% and 35% for the medium and longest SOA, respectively. It should be noted that these values are very close to the criteria used in the tracking procedure. The proportion of successful inhibits varied somewhat between age groups, $F(3, 63) = 5.29, p < .05$. The average proportion of inhibits was 57% for the youngest children compared 51% for older subjects. In the upper-left panel of Figure 3, inhibit proportions are plotted against mean RT minus stop-signal delay. It can be seen that with Age, stop signals can be presented closer to the response to yield comparable proportions of inhibition. The analysis of the slopes of the regression lines fitted to the probability of inhibition data revealed an age-related increase, $F(3, 63) = 3.50, p < .02$. The slopes increased with age from 229, 240, 320, to 408 %/s.

Evaluating the success of inhibition is complicated by the fact that the speed of inhibition and the speed and variability of the choice reaction process may differ between age groups. A lower probability of inhibition can be due to a slower stopping process simply because it will lose the race more often than a faster stopping process. Thus, it is important to obtain an estimate of the speed of the stopping process (see Experimental Overview). The stop-signal RTs and children's slowing coefficients are presented in Table 2. Overall, inhibition times are slightly longer in younger children but this effect just failed to reach significance, $F(3, 63) = 2.53, p < .07$. The slowing coefficients were 1.28, 1.29, and 1.12 indicating that the age-related gain in stopping speed is much smaller than the developmental change in response speed. Overall, stop-signal RT decreased with delay, $F(2, 63) = 6.74, p < .004$, but the ANOVA revealed a significant interaction with Age group, $F(6, 126) = 4.61, p < .001$. The 5-year-olds showed shortest inhibition times for medium rather than late delays as in the other age groups.

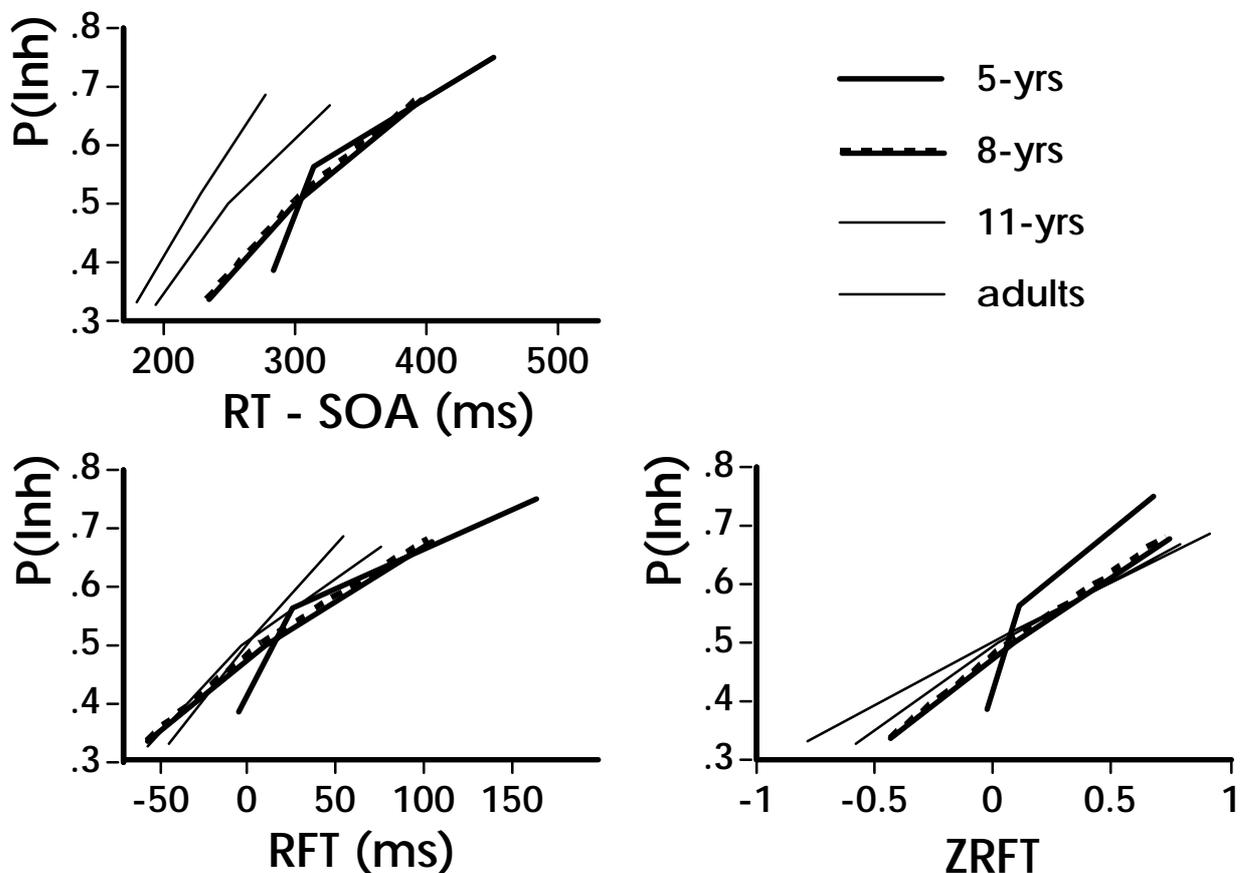


Figure 3: The inhibition function for four age groups in three subsequent stages of correction. In the upper left panel, the SOA is adjusted to differences in mean RT. In the lower left panel, the influence of mean stop speed (SSRT) is removed, resulting in a relative finishing time (RFT) of the stop- and go-process. Finally, in the lower right panel, the influence of primary-task speed variability is corrected for. (see text for explanation)

The inhibition functions, corrected for age-related changes in the speed of inhibition, are plotted in the lower-left panel of Figure 3. In this plot, the time from the overt response is defined relative to the completion of the stopping process. The slopes of the corrected inhibition functions are still steeper when subjects are getting older, $F(3, 63) = 39.67, p < .0001$. The slopes of the corrected inhibition functions are now 190, 296, 519 and 767 %/s with increasing age. These data

indicate that the apparent age-related changes in the success of inhibition cannot be reduced to group differences in stopping speed.

The slopes the inhibition function were then corrected for group differences in the variability of the choice reactions. RT variability differed widely between age groups; from 236, 138, 91, to 53 ms in adults. In order to examine whether the inhibition functions can be aligned when correcting for RT variability, the inhibition functions were plotted in terms of a Z-score that represents the relative finishing times of the stop and choice reaction process in standard deviation units derived from the choice RT distribution (see Experimental Overview). In the lower-right panel of Figure 3, inhibition functions are plotted against Z-relative finishing times (ZRFT). The ZRFT correction resulted in steeper slopes for younger children. Surprisingly, the slopes are showing an age-related decrease after correction rather than being aligned; 52, 34, 26, and 23 %/Z. The ANOVA yielded a significant developmental trend, $F(3, 63) = 3.92$; $p < .012$, but contrast analysis revealed that the only significant difference was between the youngest children and the adult subjects, $F(1, 63) = 10.59$, $p < .002$. It should be asked, however, whether this group difference in slope is more apparent than real. There is no theoretical reason to assume that the efficiency of inhibitory control decreases during middle childhood. Possibly, the steeper slope observed for the youngest children is due to "over-correction" resulting from specific features of the horse-race model. Obviously, this should be demonstrated by formal analysis or numerical simulation (Band, see Chapter 5).

The horse-race model provides a check to verify whether the data are consistent with the model. If the model is correct, the RTs of responses that escaped inhibition on stop-signal trials (inhibition failures) can be predicted by using the corresponding part of the RT distribution obtained when the visual reaction stimulus was not accompanied by the stop signal (e.g., Logan, Cowan, & Davis, 1984). The predicted and observed RTs are presented in Table 1. The predicted RTs are the means derived from the fast part of the RT distribution for nonsignal trials; that is, RTs shorter than the sum of stop-signal delay and stop-signal RT. The observed RTs are the means from trials where inhibition failed. Predicted and observed RTs should be close and, obviously, these latencies should increase with stop-signal delay. For the adult subjects, predicted and observed RTs correspond well. Differences are less than 13 ms which is within the appropriate range (e.g., De Jong et al., 1990). For all age groups, predicted and observed RTs increase with stop-signal delay as predicted by the horse-race model. Additionally, the differences between predicted and observed RTs decrease with stop-signal delay. Most importantly, deviations from the model seem to increase for younger children; from 5 ms for adult subjects to 14 ms, 50 ms, and 93 ms for the 5-year-olds. These observations were statistically verified by ANOVA examining the differences between predicted and observed RTs. The ANOVA indicated that observed RTs were significantly longer than predicted RTs, $F(1, 63) = 38.63$, $p < .0001$. The discrepancy decreased with stop-signal delay, $F(2, 126) = 22.73$, $p < .0001$, and age, $F(3, 63) = 9.70$, $p < .0001$. Finally, the between group differences were more pronounced for short compared to longer stop-signal delays, $F(6, 126) = 4.00$, $p < .004$.

A substantial discrepancy between observed and predicted RT has been reported previously by Jennings et al. (1992), using adults subjects. These authors obtained a 34 ms difference for adult subjects when stop signals were presented with a 50-ms delay and only 17 ms when stop delay was 150 ms. This finding is consistent with the present observation that the difference between observed and predicted RTs decreases with stop-signal delay. Jennings et al. (1992) raised and rejected the possibility that a bad fit might be due to the smaller amount of trials for short compared to late stop-signal delays. A re-analysis including equal trial numbers for both conditions resulted still in a better fit for late compared to short delays (Jennings et al., 1992; p. 428). De Jong et al. (1995) reported a 27 ms difference between observed and predicted RTs when adult subjects performed a selective-stopping task in which the stop signal required only the inhibition of responses with one

hand but not with the other hand. The authors pointed to a previous simulation study (De Jong et al., 1990) indicating that a poor fit might be due to a positive correlation between the duration of stopping and the choice reaction process which would be a violation of the independence assumption of the horse-race model. This interpretation is weakened by the observation of a relatively prolonged stopping duration while, as De Jong et al. (1995) rightfully argued, their simulations would predict under- rather than overestimation. The simulations by De Jong et al. (1990) suggest that the current pattern is best explained by assuming large variability of the stopping process and, possibly, a positive correlation between stopping and the choice reaction process. The bad fit between the predictions and the data of the younger children is most likely to result from a larger variability of the stopping process (see also Band, Chapter 5).

In conclusion, the adult findings are consistent with previous studies on inhibitory control using the stop-signal paradigm (e.g., Logan & Cowan, 1984; De Jong et al., 1990; Jennings et al., 1992). There was an excellent fit between the data and predictions and the estimated duration of the stopping process was within the appropriate range. The children, even the youngest age group, performed the task reasonably well. They did not adopt a strategy to wait for the stop signal and their error rates were well below acceptable limits. In contrast to the adult findings, however, the children's data did not fit the horse-race model very well. The pattern of findings was suggestive of increased stopping variability for younger children. The simulation studies performed by De Jong et al. (1990) indicate that the horse-race model may tolerate some variability of the stopping process in spite of the assumption of constant stop-signal RT. Apparently, the variability of children's stopping is more than the model can accommodate. Given the outcomes of the simulation studies performed by De Jong et al. (1990), a likely consequence would be an underestimation of stop-signal RTs, in particular for younger children and later SOAs. The conclusion emerging from the current findings would then be that children's stopping is both slower and more variable compared to adults.

On a methodological note, it was surprising that the ZRFT correction of the inhibition functions resulted in an age-related decrease in slope. On the basis of the analytical work of Logan and Cowan (1984) and a series of empirical studies by Logan and colleagues (see review in Logan, 1994), it was anticipated that correction of group differences in the variability of choice RT would result in reduced slope differences between groups or even the alignment of inhibition functions rather than steeper slopes for younger children. Recent simulation studies manipulating choice RT variability along a wide range yielded "over-correction" for increased variability (Band, Chapter 5). This observation seems to present a serious challenge to the use of ZFRT-corrected inhibition functions as the prime index of between-group differences in inhibitory control. Over-correction may obscure true inhibition deficits. The current findings suggest that larger stopping variability is the principal difference between age groups. Unfortunately, stopping distributions are difficult to evaluate (Logan, 1994; Band, Chapter 5; but see Colonius, 1990).

Stop-change task

The data from 14 subjects were excluded from data-analysis; one 5-year-old, three 8-year-olds, seven 11-year-olds, and three adults. Nine subjects had unacceptable error rates (>15%) suggesting a heightened readiness to execute the opposite response to the visual choice reaction stimulus. Such a strategy would be a violation of the requirement to give priority to the primary task. Additionally, five subjects were excluded because they responded either too quickly to early stop-change signals (> 10% anticipations) or too slowly to late stop-change signals (> 15% omissions). On average, the probability of change did not discriminate significantly between excluded subjects and their peers.

The speed of responding on nonsignal trials increased with Age, $F(2, 51) = 77.50, p < .0001$. The mean RT for each age group and the children's slowing coefficients are given in Table 1.

Response speed on nonsignal trials corresponds well with the performance in the stopping task and the error rates were very low. Error rates were low but discriminated between age groups, $F(3, 51) = 35.49$, $p < .002$. The 5-year-olds made somewhat less errors ($< 3\%$) than the older subjects ($> 4.5\%$). The speed of responding on change-signal trials increased with age, $F(3, 51) = 78.33$, $p < .0001$, but more rapidly than the choice responses on nonsignal trials. Mean stop-change RT is presented in Table 1 together with the children's slowing coefficients. The disparate developmental rates in the speed of choice and change responses present a serious challenge to the hypothesis of a single, global mechanism mediating age-related changes in speeded information processing (e.g., Cerella & Hale, 1994, for a review).

Table 3. Change-signal delays, Proportion of Successful Changes, Change RTs, Estimated Change-Signal RTs, and Mean Observed and Predicted RTs for Change Failures.

Age Group	5-yrs	8-yrs	11-yrs	adults
	SOA1			
Change-signal delay	71	-83	-128	-76
Changes (%)	68.7	55.2	58.6	65
Change-signal RT	536	562	450	356
Change RT	996	599	388	330
Predicted RT	503	403	280	250
Observed RT	685	439	260	254
	SOA2			
Change-signal delay	247	70	-19	15
Changes (%)	56.3	46.7	48.1	48.8
Change-signal RT	431	443	360	288
Change RT	1120	671	408	359
Predicted RT	543	419	290	264
Observed RT	669	475	293	262
	SOA3			
Change-signal delay	383	196	90	74
Changes (%)	40.3	31.8	31.6	33.4
Change-signal RT	402	389	301	253
Change RT	1224	731	480	379
Predicted RT	594	446	309	275
Observed RT	678	467	308	269

Note: All times are in ms. RT is Reaction Time.

The further results obtained from change-signal trials are presented in Table 3. Change-signal delay differed between age groups, $F(3, 51) = 9.61$, $p < .0001$, and, obviously, between SOAs, $F(2, 51) = 118.67$, $p < .0001$. The interaction between these effects reached significance, $F(6, 102) = 3.33$, $p < .008$. The increase in change-signal delay with SOA is much more pronounced for younger children compared to older subjects. Note that change-signal delays for the early SOA are negative with the exception for the youngest children. For the older subjects change signals were presented prior to rather than after the onset of the visual reaction signal to obtain the desired

proportion of successful changes. Thus on these trials the change signal might have acted as a warning in addition to providing response information.

The procedure of tracking the change process appeared to be successful. The change proportions were close to the values set by the tracking procedure. The obtained change proportions were 62.6%, 50.2%, and 34.6% respectively for early, middle, and late SOAs, $F(2, 51) = 453.83$, $p < .0001$. The 5-year-olds seemed somewhat more successful in response change whereas the 8-year olds did slightly worse than adult subjects, $F(3, 51) = 8.42$, $p < .0001$. The differences between age groups were more pronounced for the early SOA compared to the middle and late SOAs, $F(6, 102) = 2.62$, $p < .02$. These findings are similar to the results observed for the stopping task and should not be interpreted to suggest that either stopping or change is more efficient in the youngest children compared to older subjects.

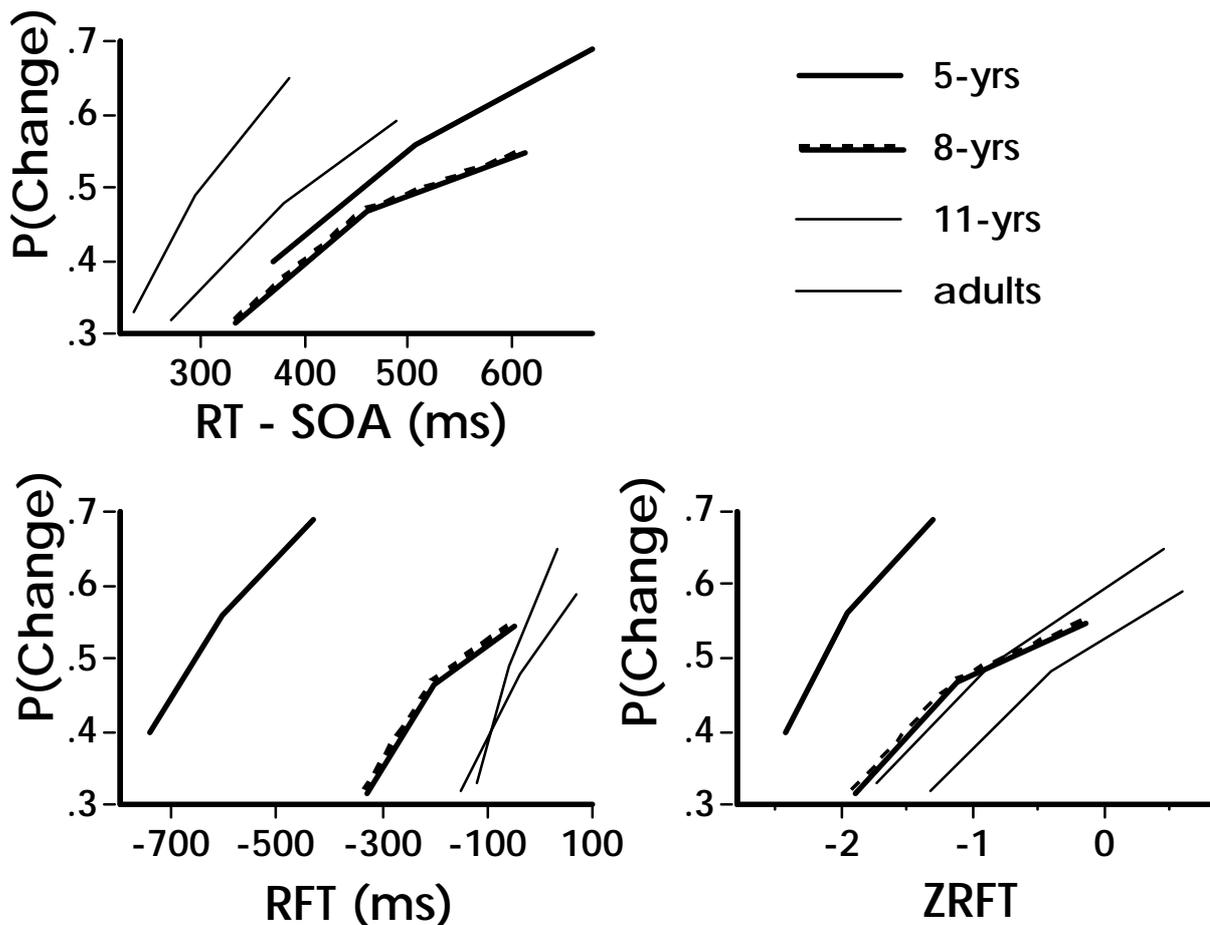


Figure 4: The stop-change function for four age groups in three subsequent stages of correction. In the upper left panel, the SOA is adjusted to differences in mean RT. In the lower left panel, the influence of mean stop-change speed (SSRT) is removed, resulting in a relative finishing time (RFT) of the go- and stop-process. Finally, in the lower right panel, the influence of primary-task speed variability is corrected for. (see text for explanation)

The change proportions are plotted in the upper-left panel of Figure 4 as a function of subjects' response times minus the corresponding change-signal delays. The slope of the change function is steepest for adult subjects and slopes decrease with age. The analysis of the slopes of the regression lines fitted to the probability of change data revealed an age-related increase, $F(3, 51) = 6.22$, $p < .001$. The slopes increased with age from 110, 120, 136 to 279 %/s. The change function obtained for the 5-year-olds differs somewhat from the other functions. The tracking procedure resulted in higher change proportions in this age group compared to the other groups. One possible

explanation might be that, in spite of instructions, the 5-year-olds delayed their response to the visual choice stimulus waiting for the change signal to occur. This strategy should result in progressively slower responses to the visual choice stimulus during the course of task performance. However, an ANOVA evaluating the effect of time-on-task failed to reveal a systematic decrease in the speed of responding. If anything, responses were faster during the second half compared to the first half of the task, $F(1, 51) = 13.59, p < .001$, and this effect was even more pronounced for the youngest age group.

The change-signal RTs derived from the horse-race model (see Stopping-task section) presented in Table 3 show that the speed of stopping to the change signal increases with change-signal delays, $F(2, 51) = 33.65, p < .0001$. This finding is similar to the stop-signal RT data and may suggest that there was some variability in the speed of stopping (cf. Logan & Burkell, 1986). Change-signal RTs decreased significantly with age, $F(3, 51) = 4.87, p < .005$, and this trend was not altered by the change-signal's Delay, $F(6, 102) > 0.8$. The estimated change times were used to correct the change functions. The corrected change functions are presented in the lower-left panel of Figure 4. The slopes of the corrected change functions increase with age, $F(3, 51) = 8.34; p < .0001$. The slopes were 60, 81, 91, and 191 %/s from young to old. These data indicate that the apparent age-related changes in change efficiency cannot be reduced to age-differences in change speed.

In order to assess whether the change functions can be aligned when correcting for age-related changes in the variability of the visual choice reaction process the RT values were normalized using the single-subject standard deviations of RT. The standard deviations differed considerably between age groups, 305, 172, 113, and 71 ms with increasing age. The ZRFT correction resulted again in the steepest inhibition function for the youngest children, $F(3, 51) = 2.40, p < .04$. The slopes were 35, 21, 14, 20 %/Z from young children to adult subjects. Post-hoc contrast analysis indicated that the 5-years olds differed significantly from the adult subjects, $F(1, 51) = 5.54, p < .02$. All other group differences were not significant. The steeper slope for the youngest children might be due to overcorrecting as suggested previously (see Stopping-task section).

In addition to the time needed to respond to the change signal, it can be examined to which extent the need to change to an alternate action influenced the activation of this response. This can be done by comparing mean change RTs to the mean of the corresponding part of the nonsignal RT distribution. In principle, change RTs should equal nonsignal RTs slower than the percentile value corresponding to the change probability. Overall, change RTs were somewhat slower than the nonsignal RTs, 645 versus 619 ms, but this difference did not reach significance, $F(1, 51) = 1.75, p > .1$. Change RTs were slower than nonsignal RTs for the 5-year-olds, 150 ms, but for the other age groups nonsignal RTs were slower than change RTs, 21, 38 and 5 ms with age, $F(3, 51) = 9.06, p < .0001$. Finally, the increase in response speed with SOA was more pronounced for change RTs, 581, 646, and 666 ms, compared to nonsignal RTs, 580, 661, and 667 ms, $F(2, 102) = 9.41 < .0003$. This interaction was not altered by age. It should be noted, however, that this comparison is not completely fair. Faster change RTs for the older age groups might be due to change signals presented prior to rather than following the onset of the visual reaction stimulus. Change signals presented prior to the reaction stimulus might have acted as a warning signal facilitating response activation. This potential confound was eliminated by comparing nonsignal RTs with change RTs obtained from trials on which the change signal was presented after the onset of the visual reaction stimulus. This comparison showed that change RTs were significantly slower than nonsignal RT, 707 vs. 639 ms, $F(1, 37) = 18.89, p < .0001$. This difference was more pronounced for younger subjects; with age 226, 11, 20, and 6 ms, $F(3, 37) = 14.65, p < .0001$. Basically, however, the conclusion remains the same. In contrast to the other subjects, the youngest children experience great difficulty in recruiting the alternate response. This finding may suggest a strategy difference between age groups. The 5-year-olds may respond to the change signal by pulling the emergency

break whereas the older subjects are able to selectively inhibit the compatible response and activate the incompatible response. In other words, the youngest children may tend to use the all-or-none inhibition mechanism while the other age groups exercise a dynamic control over their response options.

Finally, it was evaluated whether the horse-race model fitted the data by comparing observed versus predicted RTs (see Stopping task section for procedure). Predicted and observed RTs are presented in Table 3. Predicted RTs were considerably faster than observed RTs, 383 vs. 426, $F(1, 51) = 22.13, p < .0001$. This difference decreased with Age, 130, 37, 5, 1 ms, $F(3, 51) = 15.21, p < .0001$, and the age-related decrease was more pronounced for early compared to later change signals, $F(6, 102) = 2.69, p < .04$. These findings were not substantially different from the results of an analysis including only trials on which change signals followed the onset of the visual reaction stimulus. Moreover, the results are virtually identical to the findings obtained for the stopping task. The match between data and the race model is very close for the adult subjects and reasonably well for the 11-year-olds. But the horse-race model fitted the data of the younger age groups rather poorly. The model significantly underestimated mean RT for change-signal trials, in particular for the youngest children. Note that in contrast to the stop-change task employed by Logan and Burkell (1986), the correct change response in this experiment was based on the primary task. This could have introduced the risk of dependence between the primary and stop process. However, the adult data did not support this assertion, and based on the similarity between stop-all and stop-change results, there is no reason to doubt the interpretability of the present change findings.

To conclude, the adult data are in line with previous studies requiring subjects to inhibit their response to the visual reaction stimulus but to respond to the auditory accessory. There was a close correspondence between observed and predicted RT suggesting that the horse race model fit the data quite well (De Jong et al., 1995). The change-signal RTs were considerably longer than the stop-signal RTs (e.g., De Jong et al., 1995), and they tended to decrease with change-signal delay (Logan & Burkell, 1986). The difference between change- and stop-signal RTs has been taken to suggest that inhibition in the change task invokes a slower central inhibition mechanism whereas inhibition in the stopping tasks may involve a faster peripheral inhibition mechanism (cf. De Jong et al., 1995). Finally, the response to the change signal was considerably slower than simple auditory RT. This difference has been attributed by Logan and Burkell (1986) to concurrence costs from preparing to respond to the visual reaction stimulus.

Basically, the data obtained from the children yielded a similar pattern with the exception that the fit of the data and predictions was rather poor for the 5- and 8-year-olds. In addition, the problems encountered in the ZRFT correction of inhibition functions were virtually similar for the change and stopping tasks, in that ZRFT correction resulted in steeper slopes for the youngest children. All children showed longer change- than stop-signal RTs, like adult subjects, and change-signal RTs decreased with SOA. Across delays, change-signal RTs were somewhat longer relative to the latencies reported by previous studies using comparable age groups (e.g., Schachar et al., 1995; Oosterlaan & Sergeant, 1996). Change-signal RT decreased with age. This finding may suggest that the central inhibition mechanism would be more sensitive to developmental change than the peripheral inhibition mechanism. Finally, the overt response to the change signal showed the most impressive differences between age groups. Change RTs to the auditory accessory were much longer than the RTs to the tones in the simple reaction task (641 vs. 269 ms), and the age-related decrease was much more pronounced (see Table 1). Schachar et al. (1995) interpreted change RT as a manifestation of the efficiency of response re-engagement; that is, the ability to shift rapidly to an alternate action (see also Oosterlaan & Sergeant, 1996). In this vein, the current results would suggest that young children experience great difficulty to shift from one to another response; a deficit in the control of competing actions.

Response Speed

In order to assess the hypothesis that developmental change in the speed of responding is conditioned by a single factor modulating response latencies across a wide range of tasks, the age-related changes in the RTs obtained in the current study were fitted by growth functions that Cerella and Hale (1994) derived from the work of Kail. Basically, the growth function is a negative exponential function with a rate parameter and a starting value. The growth function is reduced to the multiplicative function referred to above ($Y=mX$) when age is fixed. Thus, the growth function simply modulates the slowing coefficient as the child is getting older. First, an exponential growth function was fitted to all latency data yielding a rate parameter of .29 and explaining 81.6% of the variance. Growth rates were then extracted for four types of reaction processes separately, namely simple RTs, choice RTs, change RTs, and RTs to the stop- or change signals. The growth parameters obtained for these processes were .25, .31, .33, and .11 respectively. The exponential functions fitted the data quite well for the observable (i.e., simple, choice and change) RTs but considerably less for the internal RTs (i.e., to stop- and change signals). The variance of the observable RTs accounted for by the growth functions was more than 98.1% whereas it was only 77.1% for the internal RTs. The current divergence in growth rates is in sharp contrast with the findings reported by Kail (1993) who obtained a consistent growing rate of .21 across a series of studies. The major conclusion to be drawn from the present data is that the ability to activate a response develops at a slower pace than the ability to inhibit a response.

General Discussion

This study set out to examine two related hypotheses. First, it was examined whether age-related changes in the ability to inhibit were mediated by a general or specific mechanism. This issue was researched by asking different age groups to perform on a set of two-stimulus paradigms in which one stimulus required a two-choice reaction and the other stimulus interfered in some way with the reaction process. In two tasks, the interfering stimulus was task-irrelevant and in two other tasks, the interfering stimulus was task-relevant. It was predicted that all four tasks would reveal an age-related increase in the ability to inhibit and that this developmental trend would be more pronounced for the two latter compared to the two former tasks. Across tasks, there was relatively little evidence for a pronounced developmental change in inhibitory capability. It was anticipated that the immediate-arousal effect of loud auditory accessories would be more disruptive for young children compared to adult subjects and that they would have greater difficulty in resisting the tendency to respond to the source of auditory stimulation. However, both tasks yielded only marginal evidence for inhibition failures in children. These findings seem inconsistent with convincing demonstrations of children's failures to resist interference. In a previous study, for example, Ridderinkhof and Van der Molen (1995) required subjects in a similar age range to perform on an Eriksen flanker task and observed a considerable developmental deficit in the ability to ignore flanking noise while responding to the central stimulus of the array. Moreover, the current findings are difficult to explain vis-à-vis common notions that children are more sensitive than adults to external distractors (for reviews Dempster, 1993; Harnishfeger & Bjorklund, 1993). It should be noted, however, that most of this literature is concerned with tasks in which the interfering information is task-relevant and presented in the same modality. In the Eriksen flanker task, for example, flanking noise and central stimuli are usually assigned to competing responses and both are presented in the same stimulus array. A possible explanation of the current findings would then be that adult levels of the ability to resist task-irrelevant information presented in a different modality is acquired prior to age five.

A similar pattern was observed for the tasks in which the auditory accessory was task-relevant. Both the stopping and change task should place a clear demand on inhibitory control. The stopping task, however, yielded little evidence for a clear inhibitory deficit in young children. The developmental increase in the speed of responding to the stop signal failed to reach significance. This finding is consistent with the results reported previously by Schachar and Logan (1990) and Oosterlaan (1996) and is adding to these findings by extending the age-range to 5-year-olds. Variability rather than latency seems to be the discriminating feature distinguishing between age groups. But this finding could be established only indirectly. The data fitted the horse-race model very well for adult subjects but rather poorly for young children. The specific pattern of results seemed most consistent with the interpretation, derived from simulation studies (De Jong et al., 1990; Band, Chapter 5), that the stopping process is more variable in children than adults. It might well be that in young children the inhibitory mechanism is triggered less consistently or that young children sometimes fail to detect the stop signal. Ideally, these interpretations should receive additional support from age-group differences in the slopes of the inhibition functions. Unfortunately, the procedures recommended by Logan and Cowan (1984) resulted in the steepest slope for the youngest children; a finding that cannot be accommodated by existing hypotheses of inhibitory development.

The results obtained for the change task revealed an interesting pattern. The latency of the stopping process to the change stimulus decreased with age and was systematically longer than the latencies obtained in the stopping task. Previously, De Jong and colleagues (1995) amassed performance and psychophysiological evidence to suggest that stop- and change-requirements involve different inhibition mechanisms. In the stopping task, subjects may rely on a rapid inhibition mechanism located at a peripheral level. In the change task, however, subjects must use a central mechanism that is slower because it has to be selective rather than to exert an all-or-none influence. The differential decline in stop latencies obtained for the stopping and change tasks provides support for the multifaceted nature of inhibition. It would be of interest to adopt the psychophysiological procedures employed by De Jong et al. (1995) to further examine differential developmental trends in stopping and change.

Furthermore, the change task yielded interesting data pertaining to the ability to execute an alternate response after the inhibition of current action. Response re-engagement appeared to be more difficult for young compared to older subjects. An important question to ask is why young children need excessively long to change from one response to another. One possibility interpretation is that children experience simply greater difficulty in changing from one to another response set. This possibility would be consistent with observations of young children performing on the Wisconsin Card Sorting Test. It is a recurrent finding that young children need more time to adopt a new response set after the experimenter changed the response rule (e.g., Welsh & Pennington, 1988). However, developmental deficits on this task might be due to a host of factors including the failure to inhibit the current response set. Another possibility might be that young children are more sensitive to S-R compatibility. The change task required them to inhibit a compatible response and re-engage into an incompatible response. This interpretation would be consistent with observations indicating that the costs of making an incompatible response are larger for young children compared to adult subjects (e.g., Ridderinkhof, Van der Molen, Band, & Bashore, 1997). A third, and more specific possibility, can be derived from a suggestion made by De Jong et al. (1995). These authors suggested that the hands might be functionally coupled effector systems, so that the need to inhibit one really implies inhibiting two. In this vein, the requirement to inhibit one hand and the need to relief inhibition of the other hand for executing the alternate response may ask for elaborate procedures that are more time-consuming for younger subjects. As suggested by De Jong et al. (1995), this interpretation can be examined using a change task in

which the alternate response is not functionally coupled to the primary response, such as foot and hand.

The other issue that was examined in the present research concerned the hypothesis that developmental changes in processing speed are mediated by a global mechanism (e.g., Kail, 1993). This hypothesis would predict that the developmental rates would not differentiate between tasks. However, the results did not support the global view of developmental changes in processing speed but seemed to be in favor of a selective view. Response speed to stop and change signals showed relatively little developmental change in contrast to the speed of choice reactions or response re-engagement. Simple RT developed at a rate in between stopping and choice reactions and was closest to the values reported previously by Kail (1993). Recent findings supporting selective changes in response speed during development or cognitive aging indicate that response-related processes are more sensitive to advancing age than stimulus-related processes (e.g., Bashore & Smulders, 1995; Bashore, Osman, & Heffley, 1989; Van der Molen & Ridderinkhof, in press). However, the number of studies showing differential growth or decline is too small to permit any strong conclusions. The current findings seem to add to this limited database and it would be interesting to apply the psychophysiological procedures adopted in those studies to determine the locus of selective age effects on the speed of information processing (see also Bashore, Ridderinkhof, Van der Molen, & Wylie, 1997).

Finally, it should be asked how the pattern of age-related changes in the inhibition and activation of speeded responses obtained in the present study relates to theoretical frameworks suggested to explain developmental changes in the ability to inhibit or activate motor responses. The current pattern of findings consists of relatively strong developmental trends for response selection and re-engagement and less pronounced trends for the inhibition of ongoing action. The diversity in developmental trends are clearly inconsistent with notions that speeded information processing is mediated by a single global mechanisms of the sort proposed by Kail (1993) or Cerella (1994). The present findings might be consistent with the Harnishfeger and Bjorklund's (1993) inefficient inhibition notion but their position is basically a reconceptualisation of the mental-capacity theory which has been criticized for lacking explanatory power (e.g., Navon, 1984). Moreover, Brainerd and Reyna (1989) argued convincingly that most of the performance evidence recruited in support of inefficient inhibition theory can be explained in terms of developmental changes in output interference. These authors suggested that as children grow older, their response-selection mechanism becomes more articulated resulting in a richer assortment of responses, and thus less interference. The current findings are also compatible with Dempster's (1993) frontal-lobe interpretation. There is abundant evidence to suggest that the frontal lobes are critically involved in maintaining goals, the selection of responses, and the protection against interference (e.g., Grafman, Holyoak, & Boller, 1995). Unfortunately, the frontal-lobe notion of developmental changes in performance provides only a loose framework that may accommodate most findings but does not permit strong predictions.

An important question for future research is whether developmental changes in the inhibition and activation of motor responses can be integrated within emerging theoretical notions emphasizing the regulation of action by inhibition rather than capacity limitations. Recently, Jennings and Van der Molen (1996) provided the outlines of a Coordination for Action (CAT) perspective derived from the theorizing of Allport (1980) and, in particular Neumann (1987; 1996). This perspective assumes that coherent processing and acting require the assembly of a perception/action linkage establishing a time/priority schedule. The perception/action linkage is a functional concept that is akin to pathway activation by Posner (1978) and the construction of a response channel by Eriksen and Schultz (1979). The execution of the linkage plan will necessarily reduce the number of tasks that can be performed concurrently and thus will be one determinant of

the apparent 'capacity limitation' imposed by speeded processing tasks. The execution of the linkage plan requires temporal co-ordination. That is, the scheduling of successively greater inhibition of distracting inputs and responses that are inconsistent with planned action. Furthermore, the execution of the linkage plan requires process co-ordination. That is, efficient performance is only possible by maintaining parallel actions without losing the coherence of action or unduly delaying the temporal execution of the attended action.

Developmental changes may occur along the lines suggested by this CAT perspective. Developmental deficits in the construction of a perception/action linkage should be revealed by the effects of factors such as the compliance with instructions, the use of prior information, and practice. The current data seem to provide only indirect evidence for developmental changes in the construction of a linkage plan by suggesting that younger children are less prepared when the reaction stimulus arrives. Developmental deficits in the maintenance of linkage plans should be indicated by greater sensitivity to distracting stimuli, especially when distractors are novel, similar to reaction stimuli, or induce competing response tendencies. The current data suggest that the ability to maintain the linkage plan in the presence of distracting task-irrelevant stimuli seems to mature relatively early. Developmental changes in the ability to co-ordinate smooth performance by co-ordinating perception-action structures should be revealed most clearly in dual-task paradigms. Although the current data do not directly bear on dual-task interference, the pronounced delay in response re-engagement suggests that young children may experience considerable difficulty in co-ordinating perception-action sequences. Future research will address this issue more thoroughly.

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3 Top-down Effects in a Probabilistic-Priming Paradigm: Strategic Preparation and Response Inhibition³

Abstract

Response-priming effects and psychophysiological methods were used to investigate where in the processing system priming effects are localized and whether the prime is used for deliberately preparing a response. There were clear signs of top-down modulation of the amount of response preparation at early and central levels. Inhibition and adaptation to changes in the response requirement relative to the expectation were investigated to test whether selective inhibition (change) and nonselective inhibition (stop-all) of the primed response are exerted on the same levels of preparation. Incorrect preparation could be interrupted at a cortical level as well as after the level of the motor cortex. Inhibition of muscle activation was almost confined to stop-all conditions.

Introduction

The speed of responses is sensitive to priming effects. Information about the probability of a stimulus or the probability of a response changes the way a person processes new stimuli. This may be the result of effects beyond the subjects control, for example the use of automatized processing paths. However, it may also be a consequence of strategic changes somewhere in the chain of processes between stimulus presentation and the response. Recent models of information processing increasingly emphasize the role of these top-down effects on the speed of choice reactions.

In this paper, the contribution of top-down effects to the response process is investigated. Expectations about the correct response to the following stimulus are evoked in conditions that usually provide valid information, and in conditions that provide cues without a predictive value. Top-down influences to the response process have two complementary aspects. On the one hand, probability information can be used to facilitate processing of information. On the other hand, if conflicting instructions are processed, the subject has to resolve interference.

A large number of studies has used priming in combination with probability manipulations (e.g., Gehring, Gratton, Coles, & Donchin, 1992; Gratton, Bosco, Kramer, Coles, Wickens, and Donchin, 1990; Gratton, Coles, & Donchin, 1992; Miller, Schäffer, & Hackley, 1991; Posner & Snyder, 1975). In a probabilistic-priming paradigm, information about the probability of a stimulus or response is provided in advance of the imperative stimulus. After a time interval, an imperative stimulus is presented that does or does not confirm the prediction of the prime. In order to stimulate subjects to use the information conveyed by the prime, the probability of a valid prime is often increased. Gehring et al., for instance, showed that subjects can actively control the degree to which they prepare for the primed stimulus or response, based on knowledge about the reliability of priming information. Hence, probabilistic primes result in a faster response if the information is valid.

At the same time, a higher probability of valid priming carries the obvious danger of erroneous responses on invalid trials (Gratton et al., 1992). Some authors contend that the effect of

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invalid priming is counteracted through active suppression of incorrect preparation (e.g., Kornblum, Hasbroucq, & Osman, 1990), whereas others (Coles, Gehring, Gratton, & Donchin, 1992) argue that active control is not necessary to resolve the conflict between conflicting response tendencies. For example, adaptation to new input may involve a resetting process that requires time, but not necessarily suppression of invalid priming (cf. Sanders, 1980). However, as the preparation of a response is more extensive, the subject can put more effort in the inhibition of the incorrect response, as is illustrated with subjects' ability to stop or adjust responses that are already in a far state of preparation (e.g., Logan & Burkell, 1986).

In the present experiment, the probabilistic-priming paradigm is integrated with a go/no-go paradigm. This results in three conditions that are physically similar, but that differ in the type of top-down effects that can or should be exerted on the response process. In a noninformative priming condition the probability of prime validity is at chance level. In an informative priming condition primes are valid on 80 percent of all trials and invalid on 20 percent. In the disjunctive priming condition the response hand is always correctly primed, but 80 percent of the imperative stimuli indicate that the response should be executed, whereas responses should be withheld in the remaining 20 percent.

The preparation of responses and inhibition of incorrect preparation may take at least two forms in the priming paradigm (e.g., Hackley, Schäffer, & Miller, 1990). Priming of a response in a choice reaction time task does not take away the uncertainty about *which* response to use (Rosenbaum, 1980), whereas in a disjunctive or go/no-go task (e.g., Jennings, Van der Molen, Somsen, & Terezis, 1990), the prime makes it possible to prepare for one specific response, and to execute it or not on the basis of the target stimulus. Likewise, if the incorrect response is on change trials, at least part of the preparation for the correct response needs to be initiated yet, so that the inhibition has to be selectively exerted on the incorrect preparation. In contrast, provided that there is a strong tendency to respond at the time that a no-go stimulus is presented, inhibition of the response can be exerted without concern about other responses that would need to be initiated. De Jong, Coles and Logan (1995) argued that a response can be inhibited in a nonselective way if it is not necessary to execute a different response.

Top-Down Effects: Preparation and Inhibition

Preparation

It is complicated to conclude from priming studies, whether and where top-down influences can be exerted on the mode of information processing. Valid primes can facilitate perceptual analysis, especially if the prime is identical to the target (e.g., Gratton et al., 1990). Likewise, priming of a specific response leads to faster motor processing (Rosenbaum, 1980). One problem in the interpretation of probability studies is that they contain very different types of probability information. Some studies vary the probability of a stimulus, others vary the probability of a response, but often the stimulus and response probability covary (see Sanders, 1980). This implies that the probability of a stimulus can be translated to the probability of a response and possibly vice versa. Moreover, some studies employ the same stimulus as a prime and as a target, so that the task can be performed by deciding whether the two stimuli match or not, rather than really analyzing the stimulus. It is not hard to account for priming effects in such tasks in a model without top-down influences. This was a first reason for us to employ a response prime, rather than a perceptual prime. As a second reason, we wanted to be able to investigate response inhibition processes. To maximize the need for inhibition, we have chosen prime stimuli that have a direct relationship to one of the response alternatives. The target stimuli are color patches that have no natural relationship to the response. Two colors are mapped to one response and two to another; a fifth color constitutes a no-go stimulus. Third, we wanted to manipulate the duration of the preparation on the basis of the

prime, and chose to do this by a perceptual manipulation, which would be harder to combine with a perceptual prime.

Another problem for the interpretation of probability studies is that it is hard to distinguish top-down application of probability information from nonstrategic changes in the utilization of the prime. For example, a low error rate in an informative priming condition may enhance the use of primes, either through strategic factors or through nonstrategic reinforcement of contingencies. In the present experiment we tried to distinguish between these two possibilities by presenting the informative and noninformative priming conditions not only in separate blocks, but also intermixed. It can be argued that the nonstrategic effect of priming occurs only when informative primes are not intermixed with noninformative primes, because mixing would break the contingency. In contrast, top-down modulation can occur both in pure and mixed blocks, because it involves an active interpretation and application of the prime information. The comparison between the pure and mixed blocks can thus reveal to what extent subjects change the degree of preparation strategically on the basis of information about the usefulness of the prime.

In conclusion, we have selected two indices that can enhance the plausibility of top-down modulation of preparation. First, although it is easy to recognize the primed response during the foreperiod, preparation for a probable target stimulus requires a processing step from the direction of the prime to the probability of two colors relative to two others. While a bottom-up model would be able to account for probability-dependent preparation effects on response-related processes; top-down influences are plausible if the preparation for a stimulus is modulated on the basis of probabilistic response primes. Second, application of a probabilistic prime for stronger preparation in one condition than in another is possible as a result of changes beyond the subjects' voluntary involvement, but differences in the amount of preparation can not be explained by bottom-up effects if the probabilistic information conveyed in the prime is changed from trial to trial.

Change and Inhibition

If subjects prepare for the primed response, error rates on invalid trials are higher than in a nonprepared situation. Still, after a disconfirmation of the primed information, subjects are often able to inhibit the execution of the primed response. The present experiment investigates two types of inhibition to test the hypothesis that they require different mechanisms (De Jong et al., 1995). Response inhibition is more time consuming when at the same time another response is executed, compared to when this additional requirement is absent. De Jong et al. suggested that withholding responses in favor of an alternative response requires a more specific inhibitory mechanism. They proposed a distinction between nonselective and selective inhibition. The latter is thought to require active suppression of the incorrect preparation at a central motor level. In contrast, suppression of all responses that are in preparation is thought to be possible at a later level, through the inhibition of a 'go-signal' (Bullock & Grossberg, 1991) just before response execution.

The main difference between the stop-signal paradigm that was used in De Jong et al.'s (1990, 1995) studies and the priming that is used in the present experiment, is that subjects receive the inhibition information separately from the go-information in the stop-signal and stop-change paradigm; and in a somewhat more integrated form in the priming paradigm. The consequence of this difference is that subjects may need an extra incentive to use primes for the fast preparation and activation of responses. In the design of the present experiment, the response tendency was maximized by using a fixed foreperiod so that the target onset can be estimated with high certainty (cf. Gottsdanker & Shragg, 1985), and by emphasizing speed over accuracy.

The present experiment tries to replicate the support for a difference in loci of nonselective and selective inhibition. Nonselective inhibition is tested with a go/no-go task, in which the primed response should be executed on 80% of all trials, but should be withheld on the remaining 20%.

Selective inhibition is tested with pure blocks of informative trials, which differ from the go/no-go task in the requirement in 20% of the trials to execute a response that was different from the primed response. The locus of inhibition effects is determined with performance scores and the help of psychophysiological measures that are selectively sensitive to response activation and to response execution. The relationship between selective and nonselective inhibition is assessed on the basis of the similarity between the physiological indices of inhibition in these two conditions.

It is argued by De Jong et al. (1995) that the two inhibitory mechanisms differ in speed. This difference can become more evident if the onset of inhibition processes is varied. In the present experiment, the relative timing of the preparation and inhibition of the primed response was manipulated by a contrast manipulation. In trials with a high-contrast stimulus, the relatively slow mode of inhibition may still be effective. On high-contrast conditions the information that disconfirms the primed response becomes available short after the target onset, when the primed response is not yet far in preparation. However, on low-contrast trials the onset of the inhibition process is delayed, while the response is prepared during an extended period. This implies that a slow mode of inhibition would have a smaller chance of being successful on low-contrast trials. If the fast mode is accessible for the change condition, there is a strong incentive to use it.

It is better not to use the same target colors that are mapped onto left- and right-hand as no-go stimuli, because this would disturb the pattern of response preparation that we were interested in as a test of nonselective inhibition. Therefore we chose to use one separate no-go color, which implies that there was no contrast manipulation on no-go trials.

Dependent Variables and Predictions

Behavioral measures

The costs and benefits of priming and preparation are in the first place expressed in terms of observed speed and accuracy. The difference between the RT of valid and invalid conditions reflects the amount of preparation and the difficulty to execute a response that was not primed. The speed and accuracy of noninformative priming conditions can be used to compare whether there is a relative gain on valid or loss on invalid conditions with informative priming. Noninformative conditions are neutral because they contain invalid information just as often as valid information, but nonetheless they contain directional information that allows a direct translation to a response.

The existence of top-down effects on the response process is expected to be visible from a larger validity effect on informative priming than on noninformative priming conditions. However, to substantiate the hypothesis that a difference between the validity effect in informative and in noninformative conditions is the result of a top-down mediation of preparation, the same effects should be found in pure and in mixed blocks of informative and noninformative trials. In addition, the effects of invalid priming are expected to be larger for low-contrast trials, because they provide more opportunity to prepare the primed response than high-contrast trials.

Successful inhibition of primed responses does not yield RTs, but the accuracy on no-go and change trials provides an index of the efficiency by which inhibition is exerted. Based on De Jong et al.'s (1995) observation that selective inhibition is slower than nonselective inhibition, it is expected that there are more errors on the change condition than on the no-go condition. Furthermore, because the fast inhibitory mechanism is expected to be inaccessible on change trials, a much higher error rate is expected on low-contrast change trials than on high-contrast change trials.

Unfortunately, speed and accuracy do not give a detailed view of the processes between stimulus presentation and the response. For example, RT effects do not differentiate between the results of preparation during the foreperiod and the results of preparation at the time of target processing. Also, RT effects do not specify to what extent incorrect preparation on invalid trials continues before inhibition becomes effective. These questions can be investigated in more detail

with the help of event-related potentials (ERPs). A number of components in the brain potentials have been validated as markers for the duration or intensity of specific phases in the reaction process.

P3

The P3 (Sutton, Braren, Zubin, & John, 1965) is a positive peak with a maximum amplitude over the parietal and central cortical areas. It has been interpreted as a manifestation of the revision of schemata (Donchin, 1981) or of resetting short-term memory (Grossberg, 1984). There is substantial evidence that the peak latency of the P3 (in this context also referred to as P300 or P3b) is sensitive to factors that affect the duration of processes preceding and including stimulus evaluation, while it is insensitive to changes in response-execution processes (Duncan-Johnson, & Donchin, 1982; Kutas, McCarthy, & Donchin, 1977; McCarthy & Donchin, 1981). Effects of probabilistic priming on the duration of stimulus evaluation are reflected in the latency of P3 (see e.g., Gehring et al., 1992), whereas preparation of motor processes is predicted not to affect P3 latency.

The P3 latency pattern that arises when the probability effect has a response-selection locus (see Sternberg, 1969) is harder to predict. The stimulus-evaluation interpretation of Donchin and co-writers would suggest that there is no effect of response-selection manipulations on P3 latency. However, a number of studies have shown P3 effects that are hard to interpret as (strictly) perceptual (e.g., Ragot & Fiori, 1994). This has led some authors to the hypothesis that there are two overlapping waveforms that contribute to the observed P3 wave (see Verleger, *in press*, for a meta-analysis). The first waveform is associated with perceptual processing in a way that is comparable to Donchin's (1981) interpretation. The second waveform, however, is thought to be associated with response selection, and occurs only when the stimulus code is translated into a response code along a slow route (*cf.*, Frith & Done, 1986). As a result, the P3 latency in conditions where a slow route is in effect is expected to be sensitive to all processes preceding response initiation.

Other interesting effects of stimulus probability and validity have been found on P3 amplitude. The situation that typically evokes the largest P3 is a stimulus with low probability, but with a high task-relevance (Duncan-Johnson & Donchin, 1977). On the basis of these findings, the P3 amplitude is thought to be an index of the amount of information that is extracted from the stimulus (Ruchkin & Sutton, 1978; Tueting, Sutton, & Zubin, 1970) or to reflect the updating of subjects' strategies and expectancies (Donchin & Coles, 1988). More generally, Kok (1990) showed that the P3 amplitude is sensitive to the allocation of resources. In Kok's model a rise of the P3 amplitude is associated with facilitatory processes like the orienting response, whereas a fronto-central attenuation of P3 amplitude is caused by selection processes associated with attention, difficult mental operations, or motor preparation. In line with this model, Kopp, Rist, and Mattler (1996) found an effect on P3 of the congruence between a target stimulus and its flankers, which they interpreted to reflect a difference in the amount of information that needed to be evaluated. Congruent stimulus arrays can be evaluated on the basis of any piece of information, whereas incongruent arrays require a more effortful evaluation.

In the present experiment disconfirming stimuli have low probability and have high task relevance if there are expectations about the stimulus on the basis of the prime. However, if the prime is not translated to the probability of stimuli, there is no reason to expect a difference between valid and invalid conditions.

The P3 amplitude is also sensitive to the requirement to inhibit a response. De Jong, Coles, Logan, and Gratton (1990) found a positive deflection on stop-trials. They did not interpret this difference as a classic P3 to the stop signal, because it reached a maximum over Cz instead of Pz. Instead they explained the positivity as a reflection of inhibition. Several studies have likewise revealed a positivity that overlaps the P3, and that has a more anterior maximum than Pz, in conditions where responses were correctly inhibited (Eimer, 1993; Jodo & Kayama, 1992; Kok, 1986; Naito & Matsumura, 1994; Simson, Vaughan, & Ritter, 1977; but see Pfefferbaum, Ford,

Weller, & Kopell, 1985 for an example of a reduction of P3 on no-go trials). There is some dispute about whether a P3-enhancement on inhibition trials is the result of the presence of inhibition-related activity, the absence of response-related activity, or both (e.g., Kok, 1986). The present experiment compares inhibition conditions with and without an overt response. If there is also a P3 enhancement on change trials, this can not be assigned to the absence of movement-related potentials.

LRP

Response preparation is reflected in the ERP by a negative slow wave preceding responses, called the readiness potential or *Bereitschaftspotential* (Kornhuber & Deecke, 1965), with a maximum over the motor cortex. Kutas and Donchin (1974) noted that this negativity was larger contralateral than ipsilateral to the responding hand. Others have shown that this asymmetry (called lateralized readiness potential, LRP) can be used to extract a real-time measure that shows when one hand is prepared more than the other (Coles, 1988; De Jong, Wierda, Mulder, & Mulder, 1988). In the probabilistic-priming paradigm, the LRP has been used to follow the amount of preparation for the primed hand as a function of the reliability of the prime (Gehring et al., 1992; Gratton et al., 1990, 1992). From the findings that there were different amounts of preparation for the primed hand as a function of prime reliability in the interval between prime and target, and early after target presentation, it was concluded that strategic preparation can take place at an activation level. Three indices of the LRP deserve mentioning for the present experiment (see Coles, 1988, for a review).

First, the direction of LRP is indicative of the hand that is prepared most at a given moment. In situations where early information or primes suggest a response with the incorrect hand, positive lateralization precedes the negative lateralization (Coles, 1988). It is predicted that subjects prepare the primed response during the foreperiod in informative conditions, but not in noninformative conditions. This should be manifested by differential lateralization of the LRP. After target onset, additional lateralization for the primed hand is expected to occur, preceding lateralization for the correct hand. This additional lateralization is also thought to be larger for informative than for noninformative conditions. If the preparation effect continues to have its effect after target onset, the lateralization for the invalidly primed hand should be larger for low- than for high-contrast condition.

Second, the amplitude of the LRP reflects the degree of selective preparation for one hand in comparison with the other. Several studies have shown that in no-go conditions partial information can be used to prepare a response to a degree that can be observed on the LRP (Miller & Hackley, 1992; Osman, Bashore, Coles, Donchin, & Meyer, 1992). However, these studies found an amplitude difference between no-go trials and trials with a response. Gratton et al. (1988) have even hypothesized that a response is triggered at a fixed criterion amplitude of LRP. The amplitude of the LRP has been used also to differentiate between selective and nonselective inhibition. In a stop-signal paradigm, LRPs almost reached the same amplitude for trials with correct inhibition as on response trials (De Jong et al., 1995). With reference to the hypothesis that a fixed level of LRP is associated with central triggering of the response (Gratton et al., 1988), De Jong et al. argued that in this stop-all condition, the inhibition took place at a locus after the central response command. In contrast, in a change version of the stop-signal paradigm (Logan & Burkell, 1986), where one response should be withheld while a different response should be generated, the LRP for the first response was considerably less pronounced. The main difference between the stop-change and stop-all condition was that, in the former, the alternative response was to be executed, whereas, in the latter, inhibition could be nonspecific, i.e., all motor processes could be frozen. It was concluded from the LRP attenuation that inhibition of a subset of responses required a mode of inhibition that occurred at a locus before or during the production of LRP.

In line with De Jong et al.'s (1995) findings, a large LRP amplitude for the primed hand is expected on no-go trials, because the inhibition on these trials can be exerted downstream from the source of LRP. On correct change trials, however, the LRP amplitude is expected to be smaller. Because the same mechanism is thought to be required for early and late onsets of the inhibition process, a difference in LRP amplitude for the primed hand is not expected between high- and low-contrast change trials.

Third, the onset of the lateralization is considered to be a real-time index of the latency at which selective activation for one hand starts (Miller, Patterson, & Ulrich, in press; Smulders, Kenemans, & Kok, 1996). If the prime leads to activation of the incorrect response, the onset of the lateralization for the correct hand is affected by the time that is required for resolving the conflict between two opposing response tendencies. After the onset of activation for the correct hand, the time between LRP onset and RT is an indication of the speed by which the response is activated.

In the present experiment faster activation of the correct response in valid than in invalid conditions would be expected. Additional validity effects during activation of the correct response should be manifested by an increase of the validity effect between LRP onset and RT. The contrast manipulation is likely to have a main effect on LRP onset. In addition, because the contrast manipulation affects the duration of preparation for the primed response, the validity effect is expected to be larger in low-contrast than in high-contrast informative trials; whereas the noninformative trials are not likely to show a large validity effect on LRP.

EMG

Muscle activity is used as an index of peripheral motor activation that can be observed prior to the overt response and even when the response is not executed.. For the measurement of validity effects, the mean EMG-onset latency is a variable that reflects all preceding effects in the response process, but does not include the validity effects that are present in the speed of execution. That is, subjects may execute a nonprimed response with more hesitation than a primed response.

For the investigation of inhibition, the EMG on no-error trials is an index of whether the activity of the primed hand can be suppressed close before the response, or has to be inhibited before the start of EMG to be successful. Based on the distinction between a central locus of selective inhibition and a later locus of noncentral inhibition, no correct trials with EMG are expected in the change conditions, whereas some EMG trials are expected in the no-go condition.

The Present Experiment

To summarize, the present experiment combines two approaches to top-down effects on the reaction process that are interdependent. An informative prime about the response can support preparation, but an invalidly primed response needs to be suppressed. While other studies compared either noninformative with informative priming of response hands (e.g., Gehring et al., 1992), or informative priming of response hands with disjunctive priming (e.g., Hackley et al., 1990), we compared the three types of priming in one experiment. Each trial in the experiment consists of a prime that informs the subject about a response direction and the probability that response will need to be executed. After 1500 ms, a target stimulus, consisting of a color patch, is presented. Each of four possible colors is mapped to one of two response hands.

In the choice-RT part of the experiment, the most important information is the validity effect. The difference between valid and invalid conditions is used as a measure of the amount of preparation for the primed response, and possibly the associated stimuli. Preparation in the informative conditions is compared with preparation in noninformative priming conditions to test whether probability information is used to modulate the degree of preparation, either intentionally or not. To differentiate whether differences in preparation are the result of top-down modulation,

the validity effects in mixed blocks with noninformative and informative priming trials are compared with those in pure blocks. Finally, a contrast variable is used to manipulate the relative timing of preparatory processes based on the prime and the processes that are based on the target.

In the response-inhibition part of the experiment, suppression of the primed response on no-go trials is compared to suppression of the primed hand on change trials, where the target called for the nonprepared response. The former situation is expected to make it easier for the inhibitory system, because a nonselective form of suppression can be employed. The latter situation calls for selective inhibition of the primed response, because a complete arrest of responses would also hamper the correct response, which needs to be executed as fast as possible.

METHOD

Subjects.

Seven adult psychology students (mean age 21.1 years, three males and four females, one left-handed and six right-handed) participated and received course credits and a small financial bonus for fulfilment of the experiment. All subjects reported normal or corrected-to-normal vision.

Stimuli

Each trial began with the presentation of a prime in the center of the screen, consisting of an arrowhead (size 21×7 mm ($h \times w$), visual angle 1.0°) pointing to the left or right. The color of these arrows was red, brown, or white on a black background. This color indicated whether the prime was informative, noninformative prime or signalled a disjunctive trial. The arrows were presented for a duration of 150 ms and were followed by a colored square at a fixed interval of 1500 ms after the onset of the arrow. This target square, sized 21×21 mm; visual angle 1.0° , could be colored in one of five ways.

Responses with one hand were required to a mint-colored (a mixture of RGB values 0-67-67 and 34-100-34) or purple square (100-34-100), while the other hand was to be used in response to a turquoise (a mixture of 0-67-67 and 34-100-100) or yellow square (100-100-34). Dark grey squares (67-67-67) always instructed subjects to withhold responses (no-go trials), while the remaining mapping of target colors to hands and of prime colors to probability of validity was balanced between subjects. Purple and yellow targets constituted a high-contrast condition, while mint and turquoise were used for a low-contrast condition. The squares remained on the screen during 500 ms, or until a response was given. Responses were recorded until 1500 ms after the target onset. The total duration of a trial varied randomly between 3000 and 3500 ms. Within each block of 160 trials, stimulus order was randomized, with the restriction that the same target was never repeated for more than four times in a row. All combinations of response hand and contrast level occurred with the same frequency.

In all experimental blocks, the direction of the arrowhead prime was semi-informative with respect to the upcoming response requirement, while the color of the prime indicated the probability of correspondence with the response (e.g., a left-pointing arrowhead followed by a target that signalled a left-handed response), with values of 50, 80, and 100%. In the 100% condition, however, there was a 20% chance of a no-go stimulus. With reference to the predictive value of the prime for the response, the 50/50 condition will be called noninformative, whereas the informative condition consists of the 80% valid vs. 20% invalid trials. The prime on go/no-go trials changes the choice-RT task effectively into a disjunctive task (Donders C; Donders, 1868).

Recording.

Stimuli were presented and responses were recorded with a 486SX25 PC with SVGA card and monitor, with timing control from a master computer. The master computer, a 486DX33 PC, continuously recorded the electrophysiological signals. These signals included bipolar EMG of the left and right flexor muscle of the index finger, vertical EOG as measured bipolarly from above and

below the left eye, bipolar horizontal EOG between the two outer canthuses, heart rate (ECG), respiration, and EEG. EEG was recorded from the leads Pz, Cz, Fz, Fp1, Fp2, F7, F8, F1, and F2 of the international 10/20 system (Jasper, 1958), plus C3' and C4', located 1 cm medial and anterior to C3 and C4 respectively, and Oz, located between O1 and O2 at the midline. Software from InstEP systems was used for the physiological data acquisition at a sampling rate of 100 Hz.

The combined respiration and ECG signal was recorded with Ag/AgCl electrodes on two lower lateral sites on the chest that were grounded to the left ankle. An on-line circuit separated the two signals and produced a discrete pulse triggered by each R-top of the ECG. EOG and EEG were recorded with a low-pass filter of 35Hz and EMG passed through a 3000 Hz low-pass filter. The time constant was 1 s for EOG and EEG and 0.003 s for EMG. All EEG leads were referenced to the left ear lobe and grounded to the forehead. EMG and EOG were measured with tin electrodes and EEG was recorded with an ECI electrocap containing tin electrodes. The impedance was below 5 k Ω for EEG and EOG, and below 15 k Ω for other electrodes.

ECG and respiration were recorded to examine heart-rate changes in relation to probabilistic priming and response inhibition (cf., Jennings, Van der Molen, Brock, & Somsen, 1992). Also, the frontal EEG leads were recorded to support the topographic analysis of amplitude changes, especially those related to inhibition (cf., De Jong et al., 1990). There is reason to believe that inhibitory processes are, at least in part, instigated by frontal or prefrontal cortical areas (e.g., Brunia, 1997). These data will not be reported in the present chapter.

Procedure

The subjects were tested in a dimly lit, sound-attenuated room. The subjects were seated in a comfortable chair at a distance of 1.25 m from a 15 inch monitor and were instructed to keep their hands resting on a table, with their left and right index fingers resting on the keyboard response keys 'z' and 'm'. Small elevated dots were mounted on these keys to avoid any confusion for the response. Subjects were tested on three separate sessions within one week. Each session contained two hours of real testing time, and one hour overhead. Short rests were possible and encouraged between blocks. Subjects typically had ten-minute breaks after one hour of testing.

At the start of each of these sessions, the color-to-hand mapping was trained in at least one block of 160 nonprimed trials. After that, there were four types of experimental blocks that were grouped within a session. The order of the four conditions was balanced across sessions and subjects. The first five trials of each block were treated as practice trials. Each session consisted of one block of noninformative trials, two blocks of informative trials, two blocks of disjunctive trials and four blocks with an equal mixture of informative and noninformative trials. In consequence, there were at least 92 trials in the smallest cell. The mapping of the colors of imperative stimuli to hands, and of the colors of primes to the probability of valid priming, were balanced between subjects.

The instructions to the subjects emphasized a high accuracy combined with a maximum speed and specific directions about how to handle the prime and target. Subjects were instructed to make use of the direction and the validity of the prime for an optimal preparation of a fast but accurate response. Subjects were instructed on and trained in eye-blink control during the practice blocks.

Data reduction.

Off-line analysis of electrophysiological data was performed on epochs of 512 samples for every trial; starting 1500 ms before the prime, and continuing until 2120 ms after the target. The values of 400 ms before the prime were used as baseline for the analysis of ERPs during the foreperiod. A baseline interval of 400 ms preceding the target onset was used for analyses of ERPs after target onset. This duration of the baseline was chosen to have sufficient samples for a reliable estimate, while a longer interval preceding the prime or target could contain ERPs to previous stimuli. Movement and recording artifacts and flat-line recordings longer than 90 ms were reason

for rejection of EEG, as were trials with EOG exceeding 2.5 standard deviations above mean baseline activity. The remaining EOG was removed from EEG by regression in the frequency domain.

The lateralized readiness potential (LRP) was calculated via a double-subtraction procedure from the potentials recorded at C3' and C4' (see Coles, 1988). By default, the average LRP was calculated with the single-trial RT as the point of synchronization (response-locked). For correct no-go trials, there is no such anchor point, so that some averages were also calculated relative to stimulus onset. The analysis of response-locked LRP has the advantage that the waveform is relatively well preserved, because the duration of the LRP-RT interval is less sensitive to confounds than stimulus-locked LRP (Osman, Moore, & Ulrich, 1995). Analysis of LRP was done after low-pass filtering at 3.9 Hz. Such a filter is thought to capture the most interesting lateralization that is associated with differential response preparation, while it ignores high-frequency changes in lateralizations that may obscure scoring of LRP polarity and latency (cf. Gratton et al., 1988).

Usually the LRP-onset precedes RT by 100 – 200 ms RT. Because preparation could start early in this paradigm, the onset was determined in a time window of 300 ms preceding the response. Smulders et al. (1996; see also Miller et al., in press) recommended that onset can best be scored with a criterion that is proportional to the peak; for example with the crossing of 50% of the peak amplitude. Because we were interested in priming effects that might be more evident in the earliest onset than close to the actual response we chose 20% of the peak as a criterion. The onset criterion should not be too high, so that the latency measure is sensitive to the effects that were present before the start of the initial activation of responses, but not to the effects that occurred during the earliest section of the LRP. Smulders et al. warn that a conservative proportion such as 50% may yield a pattern of data similar to RT. The onset was determined on response-locked averages to analyze the validity effects, but on stimulus-locked averages for the comparison of inhibition effects. In a small number of signals, the amplitude was more negative than 20% of the peak in the full interval. In that case the last local maximum before the response was selected as the onset. The onset could be scored for all but one subject, who tended to have positive lateralizations at the time of responding, but who was comparable to other subjects in speed or accuracy. Response activation for the primed hand before target onset was scored with the LRP in the full foreperiod. The mean amplitude was referenced to a baseline interval of 400 ms before the prime.

For the analysis of P3, we used data that were low-pass filtered at 7.8 Hz. The latency of the maximum positivity on Pz between 250 and 750 ms after the prime and the target in the filtered data, was interpreted as the P3 peak latency. The onset of rectified and integrated EMG was determined for both hands with a criterion of 2.5 standard deviation above mean baseline activity. The amplitude on Cz was calculated by the same method. This allowed us to investigate whether amplitude differences were largest on Pz, as would be expected if the difference were related to the conditions that underlie the P3 in general, or on more anterior areas, as would be predicted for an inhibition-related positivity.

RESULTS

Analyses

In this experiment we performed two sets of comparisons, aimed at the analysis of 1) probabilistic priming effects, and 2) nonselective versus selective inhibition. The first analysis primarily compared data from the informative and the noninformative conditions to see whether and how subjects strategically prepared for the primed response. The tasks with noninformative or informative primes in pure blocks were also compared to the go condition of the disjunctive task. The analysis was subdivided into sections with the performance results, the amplitude effects of

priming, and the effect of probabilistic priming on latencies of physiological responses. Data from the probabilistic-priming task ANOVAs could be classified with the within-subject factors Validity (valid vs. invalid prime), Contrast (high vs. low), Information value (informative vs. noninformative) and Block type (pure vs. mixed blocks). In the second set of analyses, nonselective inhibition was compared with selective inhibition of the prepared response. For this purpose we investigated the disjunctive task and the pure blocks of the informative task. In this respect, the invalid conditions will be referred to as inhibition trials of the change type.

Preparation effects

Performance data

Reaction time

Table 1 shows the reaction times, as well as other latencies. An ANOVA with four factors confirmed main effects for Information value (20 ms; $F(1, 6) = 8.57, p < .03$), Contrast (56 ms; $F(1, 6) = 24.13, p < .005$) and Validity (63 ms; $F(1, 6) = 61.45, p < .001$) in the predicted directions, but no effect of Block type ($p > .7$).

An interaction of Information value and Validity ($F(1, 6) = 42.54, p < .001$) showed that the validity effect was larger for the informative conditions (103 ms) than for the noninformative conditions (23 ms). This result supports the idea that subjects made use of probability information for an optimal preparation. The fact that the interactions of Block type \times Information value ($p > .5$); Block type \times Validity ($p > .4$) and Block type \times Information value \times Validity ($p > .3$) were nonsignificant suggests that the difference between the results on the informative and noninformative priming conditions was due to strategic modulation of preparation effects, and not to conditioned adjustments to the stimulus context. Contrast did not interact with Information value ($p > .1$), and there was no Contrast \times Information value \times Validity effect ($p > .3$).

The prediction that a lengthening of perceptual analysis would enhance the validity effect on mean RT was not supported. There was no significant effect of Validity \times Contrast ($p > .7$), Contrast \times Information value ($p > .1$) or Validity \times Contrast \times Information value ($p > .3$).

Valid priming led to a faster response on informative than on noninformative priming conditions. Given that the disjunctive conditions provided the same probability information about the response as informative conditions, no difference was expected.

A separate ANOVA compared RT on all valid and go trials, with the within-subjects factors Task (noninformative, informative, and disjunctive) and Contrast (high and low). There was an effect of Task, $F(2, 12) = 18.29, p < .001$. Noninformative trials were slower than informative trials (contrast: $p < .01$), but informative trials did not differ in speed from disjunctive trials (contrast: $p > .1$). There was no significant interaction of Task \times Contrast ($p > .1$). This indicates that the effect of Contrast was not removed by the certainty about the hand that should respond.

Table 1: Summary of effects on the mean latency of P3 peak, response locked central motor preparation (LRP), muscle-activity onset (EMG) and reaction time (RT), in ms.

	Task	Contrast	P3		LRP		EMG		RT	
			Pure	Mix	Pure	Mix	Pure	Mix	Pure	Mix
No-go	DIS	Grey	487		<u>220</u>		<u>241</u>		<u>330</u>	
Go	DIS	High	400		223		297		374	
		Low	456		264		323		409	
Valid	INF	High	433	411	288	257	329	335	412	417
		Low	480	500	305	333	361	380	450	472
	NI	High	447	447	370	312	383	381	470	464
		Low	507	519	410	372	431	443	528	530

Invalid	INF	High	511	520	340	368	426	434	509	517
		Low	543	541	467	463	475	480	568	568
	NI	High	500	481	382	318	409	399	496	486
		Low	506	556	418	397	454	463	546	555

Note: Underlined numbers represent the latencies on error trials, other numbers represent the latencies on trials with correct responses. Information value conditions: DIS = disjunctive priming; INF = informative priming; NI = noninformative priming.

Accuracy

The pattern of response accuracy, shown in Table 2, was in agreement with the RT data. The ANOVA on percentage correct showed an effect of Validity; $F(1, 6) = 18.67, p < .01$. Subjects made 16.2% errors on invalid and only 3.7% errors on valid trials. The contrast effect did not reach significance ($F(1, 6) = 5.19, p < .07$): Percentage correct was 87.3% for low contrast and 92.8% for high-contrast stimuli.

The effect of Information value ($F(1, 6) = 6.94, p < .05$) was opposite to the reaction time results in that the faster informative conditions were accompanied by more errors than noninformative conditions. This was not due to a speed-accuracy trade-off, because the high error rate was found for the slow invalid informative condition. This was confirmed by an interaction of Validity \times Information value ($F(1, 6) = 10.20, p < .02$). This pattern would be predicted if subjects were to modulate preparation based on probability information.

The interaction of Validity and Contrast reflected a larger validity effect for low contrast stimuli; ($F(1, 6) = 5.91, p < 0.06$). Furthermore, the information-value effect was smaller for high-contrast conditions (Information value \times Contrast, $F(1, 6) = 4.73, p < .08$). The relatively high error-rate in the low-contrast informative invalid condition was not reflected in a three-way interaction of Information value \times Contrast \times Validity ($p > .1$)

A separate ANOVA compared accuracy on all valid and go trials, with the factors Task (3) and Contrast (2). The effect of Task, $F(2, 12) = 18.29, p < .001$, reflected more errors on valid informative than on disjunctive go trials (contrast: $p < .04$) and more errors on noninformative than on informative trials (contrast: $p < .02$). There was no significant interaction of Task \times Contrast ($p > .3$).

Table 2: Summary of effects on accuracy from the priming conditions and contrast of targets.

	Task	Contrast	Accuracy	
			Pure	Mix
No-go	DIS	Grey	92.0	
		High	99.7	
		Low	98.9	
Valid	INF	High	99.2	98.8
		Low	97.4	97.8
	NI	High	96.7	95.3
		Low	93.4	92.1
Invalid	INF	High	78.1	85.9
		Low	66.2	71.4
	NI	High	94.4	94.2
		Low	90.5	89.5

Note: DIS = disjunctive priming; INF = informative priming; NI = noninformative priming.

Subjects were slower and less accurate in the invalid informative conditions than in other conditions. These findings are consistent with our interpretation that subjects initially prepared for the primed hand, but started to prepare the response on the basis of the target after some time. It would thus be predicted that errors on invalid informative conditions are overrepresented in the fastest bins of the RT distribution, because fast responses would leave little time for the analysis of the target. On the other hand, if the prime is valid, accuracy in the fastest trials should be relatively high. Furthermore, noninformative priming conditions are likely to show a relatively small validity effect on the accuracy of fast responses. Slower responses are likely to be associated with smaller differences in accuracy between invalid informative conditions and noninformative or valid conditions, because the target could be fully analyzed on these trials.

The high error rate on low-contrast trials can also be explained by arguing that the primed response has precedence as long as the analysis of the target has not led to support for a different response. This would imply that the errors of low-contrast trials were in large part fast responses. However, there is an alternative explanation for the high error rate in low-contrast conditions. On a proportion of trials the analysis of low-contrast stimuli may have been too difficult. In that case subjects could have based their responses on the prime instead of the target. This explanation would be consistent with the relatively high error rate in the invalid low-contrast conditions. The conclusion that the analysis of a stimulus is too difficult can not be drawn before some time is spent on its analysis. Therefore, this interpretation of low-contrast errors would imply that the errors were relatively slow responses. The relationship between the speed and errors will now be analyzed by calculating the accuracy as a function of RT per latency bin, also referred to as the conditional-accuracy function (e.g., Gratton et al., 1988).

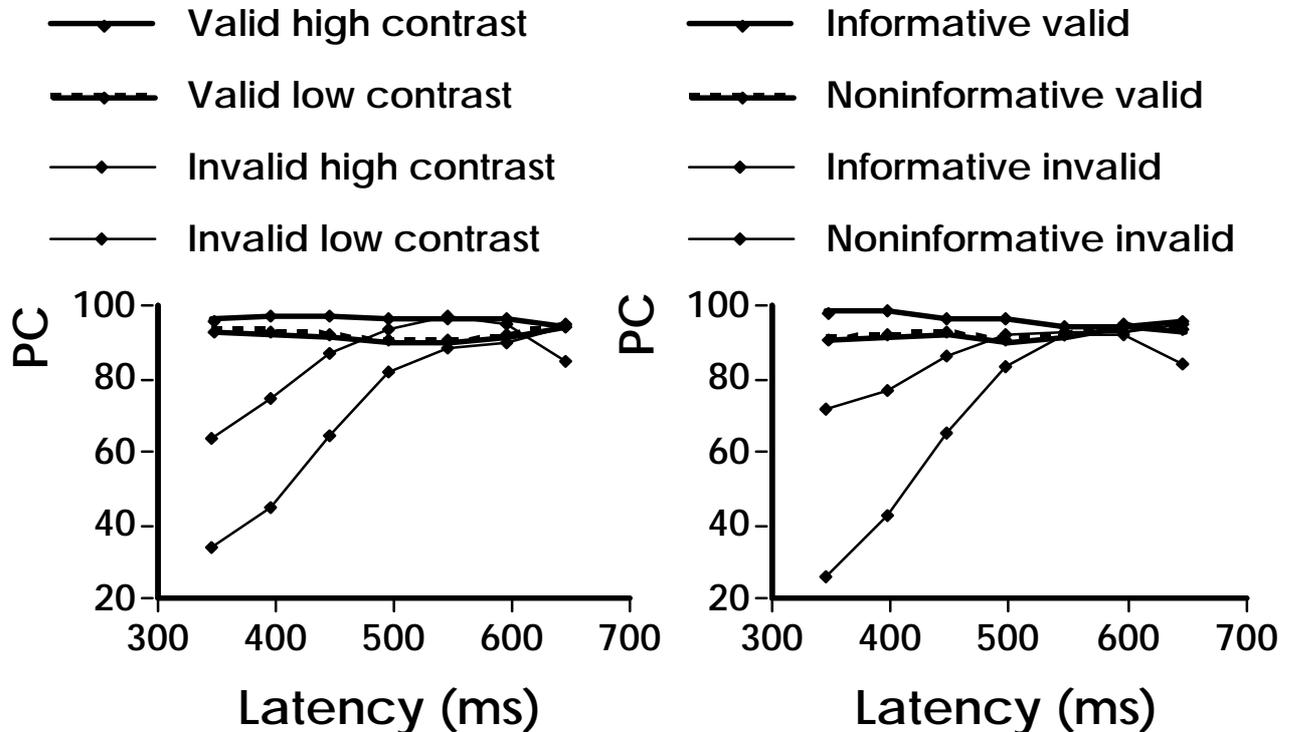


Figure 1: The vincentized conditional-accuracy functions for the interactions of Validity \times Contrast (left panel) and Validity \times Information value (right panel). From the seven latency bins that are displayed, the first, sixth and seventh were not analyzed.

Conditional accuracy

The accuracy was calculated for seven separate bins of 50 ms width, with latency borders based on single-subject reaction time averages. The first bin ranged from RT-175 until RT-125 ms, and the seventh bin ranged from RT+125 till RT+175 ms. In order to be included, smallest cells needed to consist of more than five observations. Because of an excess of missing values, accuracy for the first, sixth, and seventh bin were not included in the analysis.⁴ An ANOVA with five factors was performed on the accuracy, including the new within-subject factor Bin (intervals 2, 3, 4 and 5). Only effects involving the factor Bin are discussed in the following.

Figure 1 displays vincentized results for all seven bins, from which several patterns can be distinguished. There was a constant high accuracy across bins (minimum = 90.9%; average = 94.2%) for valid conditions. Planned comparisons showed that the overall decrease of accuracy with RT for valid trials did not reach significance ($p < .1$), but that the decrease for the informative valid conditions was significant ($F(3, 18) = 8.16, p < .002$). However, due to the dominant effect of invalid conditions there was an increase in accuracy for later bins when invalid and valid conditions were taken together ($F(3, 18) = 30.66, p < .001$). Furthermore there was a decrease of effects for later bins. This became evident from the effect of Bin \times Validity ($F(3, 18) = 31.08, p < .001$), Bin \times Information value ($F(3, 18) = 14.97, p < .001$), and Bin \times Contrast ($F(3, 18) = 8.59, p < .001$). Separate analyses for different bins showed that the effect of Information value remained significant until the third bin (out of seven); in line with the prediction that the strategic effect would be restricted to fast responses. The main effects of Validity and Contrast remained significant until the fourth bin.

Although the noninformative invalid condition exceeded the accuracy of the noninformative valid conditions after the third bin, the informative invalid condition did not catch up with the informative valid condition. This effect suggests that the strategic preparation on informative conditions was not restricted to the fastest responses. On the other hand, it could be argued that the speed of only a subset of processes was responsible for the errors, so that responses with a modal RT were occasionally affected by fast error-prone processes, and contributed to the decrease of accuracy in later latency bins. Note that fast informative invalid trials scored below chance level. It is most likely that this score was reached by a large number of informed guesses, where the prime, rather than the target was the basis for responding. There was a three-way interaction of Bin \times Validity \times Information value ($F(3, 18) = 14.26, p < .001$) indicating that with informative valid trials accuracy was even high at short response latencies; that with informative invalid trials the prime induced below-chance accuracy at short latencies; and that with noninformative primes these effects were of intermediate size. Accuracy below chance level was also found for low RTs to invalid low-contrast trials (44.6% vs. 74.7% for invalid high contrast conditions), as can be seen in Figure 1. A Bin \times Validity \times Contrast effect; $F(3, 18) = 8.59, p < .001$; shows that the difference between high and low contrast invalid conditions faded out with increasing response latency. The Validity \times Contrast interaction was significant until the third bin. This result confirms the interpretation that subjects more often executed the invalidly primed response during the delay of perceptual analysis.

There is considerable similarity between these conditional-accuracy functions and those of Gehring et al. (1992), who used the possible target stimuli as primes with 80% or 50% validity. They also found a slightly decreasing accuracy with increasing latency after valid informative primes, whereas the invalid informative primes led to accuracy below the chance level for fast

⁴In the remaining bins, an occasional missing value (3 missing values vs. 445 observations), was replaced by the group mean. This procedure was suggested by Hand and Taylor (1987; p. 46).

responses, and a higher accuracy for longer RTs. Interestingly though, both the valid and invalid noninformative conditions resulted in an accuracy close to 50% for fast responses in their data.

In summary, the behavioral data suggest that the primed response is prepared as long as the perceptual analysis has not affected this preference. It is also clear that the preparation has larger effects when the prime is informative than when the prime is noninformative, and that the information value of the prime is interpreted, because the same difference between informative and noninformative priming was found in these conditions for pure and for mixed blocks.

It is hard to tell whether the preparation had a perceptual or response-related locus, or both. Psychophysiological data that can shed a light on this issue will now be discussed. The order of discussion is chronological, although it is known that the different processes that are reflected in the psychophysiological measures can overlap in time (e.g., Coles, Smid, Scheffers, & Otten, 1995). The first signs of preparation can be looked for during the foreperiod, when the LRP can lateralize for the primed hand (see e.g. Gehring et al., 1992). After target onset, the primed response can be prepared, while the target is analyzed. The direction of lateralization in the early phase after target onset is predicted to reflect activation for the primed hand. Early processes related to the interpretation of the target are reflected in the P3 latency and amplitude. Finally the magnitude of validity and information-value effects can be followed at different processing levels with the latency of the P3 peak, LRP onset and EMG onset. This will help to clarify which subprocesses are involved in strategic preparation. If effects on reaction time cannot be found on the latency of P3 and LRP, it is concluded that there is more preparation going on downstream from the processes that are reflected in these latency indices.

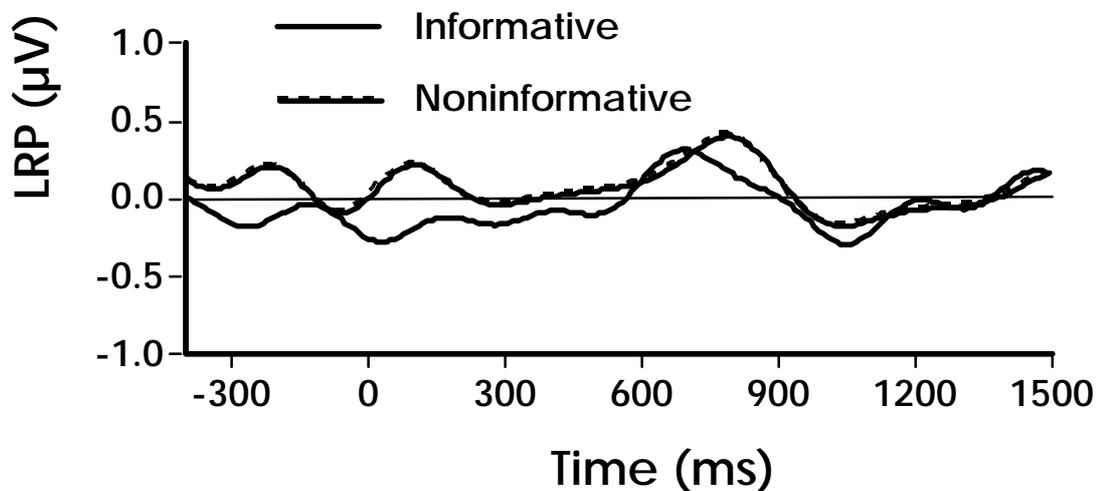


Figure 2: The average stimulus-locked LRPs during the foreperiod. Preparation for the primed hand is negative. Prime onset is at zero ms, target onset is 1500 ms. The overall lateralization is not significantly below zero.

LRP during the foreperiod

The first preparation could take place right after the presentation of the prime. Figure 2 shows the LRP during the foreperiod. The mean LRP amplitudes in the foreperiods were compared in an ANOVA with the factors Block type (pure vs. mixed) and Information value (informative vs. noninformative). The average area across conditions was slightly positive but did not differ significantly from zero (z -test $p > .2$), contrary to what would be expected when preparation for the primed hand would take place during the foreperiod. There was no effect of Information value ($p >$

.1) or Block type ($p > .4$) on the degree of lateralization. Thus, there was no support for the hypothesis that responses were already activated to a subthreshold level in anticipation of the target. Because this effect is in contrast with that of other priming studies (e.g., Gehring et al., 1992), the absence of lateralization calls for an explanation. A technical issue that may have affected the LRP to some extent is the time constant of the EEG recordings. While Gehring et al. used an 8 s time constant, our recordings were made with a time constant of 1 s. This difference would certainly lead to an attenuation of slow potential changes such as the foreperiod LRP. However, it can be seen from the LRPs of Gehring et al. and Gratton et al. (1988) that the lateralization only becomes negative during approximately the last 300 ms before target onset. Although it is hard to estimate how exactly the LRP would have been using a longer time constant, it is not plausible that the overall activity would be positive and that the change of lateralization in the last 300 ms would be positive due to a difference in time constant. We would rather compare the task complexity and the instruction as an explanation of the difference in foreperiod LRP between Gehring et al. and the present experiment. In Gehring et al.'s experiment, the prime and target stimuli were physically identical. This made it easier for the subjects to start the preparation of a response to the prime in the same way as they would to the target. Furthermore Gehring et al. instructed subjects to respond as quickly as possible while remaining reasonably accurate. This small instructional nuance may have stimulated subjects to base the response on the prime more often than in the present experiment.

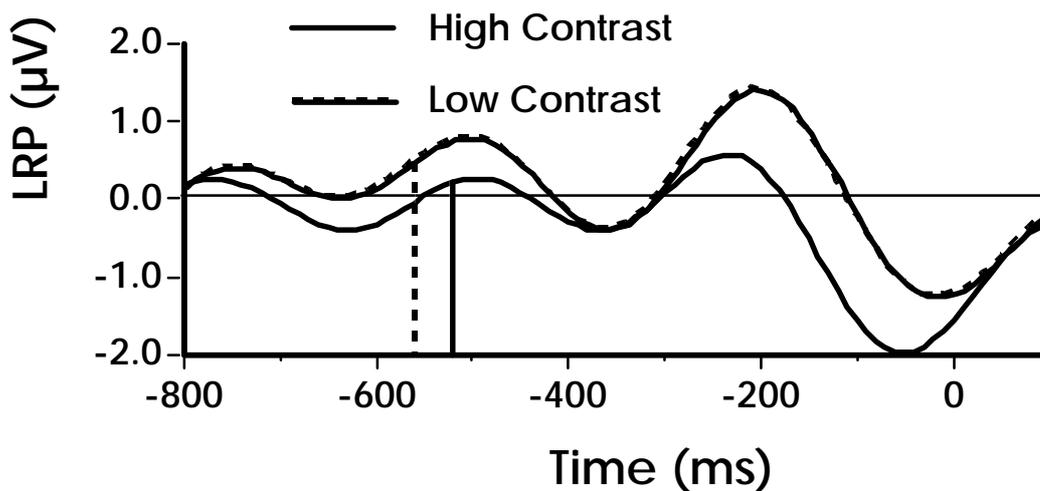


Figure 3: The average response-locked LRPs of invalid informative trials. Preparation for the primed hand is positive. Along the time axis, vertical lines indicate the average stimulus onsets, zero indicates the reaction time.

Incorrect LRP

The absence of LRP in the foreperiod could imply that subjects postpone activation of the primed response until the target onset. On the LRP, this possibility should become evident in invalid conditions from activation of the primed hand, prior to the activation of the correct hand (cf., Coles, 1988). It was expected that this effect would be largest for the informative low-contrast condition, because that was the condition with the strongest preparation effects and the longest delay of the target analysis that could counteract this preparation. To the extent that incorrect activation could be observed, visual inspection indicated that this activation was maximum between 300 and 150 ms before the response. Therefore, the mean amplitude in this interval was submitted to an ANOVA.

In the omnibus ANOVA there was not a significant effect of Validity in these data ($p > .3$). There was a tendency for a difference between informative and noninformative priming conditions ($F(1, 6) = 5.95, p < .06$) that indicates that the noninformative conditions may have attenuated the overall validity effect: The noninformative conditions had limited validity effects ($+0.3 \mu\text{V}$ vs. $+0.1 \mu\text{V}$), whereas the validity effects were larger in the informative conditions ($-0.1 \mu\text{V}$ vs. $-0.7 \mu\text{V}$). In a planned comparison of informative priming conditions (see figure 3), the effect of Validity reached significance; $F(1, 6) = 10.25, p < .02$. The interaction of Validity \times Contrast was not significant ($p > .1$). These results suggest that there was initial activation of the primed response in invalid informative conditions, even though the overt response was correct. They confirm the previous conclusion that if the prime is informative, subjects prepared the primed response as long as the analysis of the target has not counteracted this preparation.

P3 amplitude

The P3 peak amplitudes over Pz (see Figure 4) were submitted to an ANOVA. The validity of prime information had a $1.3 \mu\text{V}$ effect on P3; $F(1, 6) = 11.30, p < .02$. Invalid trials resulted in larger P3's, suggesting that the stimulus did not match the expectations. This interpretation is supported by an Information value \times Validity interaction ($F(1, 6) = 11.32, p < .02$), which showed that the Validity effect was larger in the informative than in the noninformative condition ($2.5 \mu\text{V}$ vs. $0.2 \mu\text{V}$).

The amplitude data make clear that there was substantial strategic preparation for the primed hand before completion of the processes underlying the P3. In the noninformative condition there was no strong expectation to be matched or mismatched. In the informative condition, however, there was a large P3 when the target did not match the expectations.

The finding that the effects such as Information value and Validity were visible on P3 amplitude suggests that the prime resulted in an expectation that was matched or mismatched by the target. However, the nature of the expectation about the target is not clear from the literature about P3. Several authors have argued that the P3 amplitude is sensitive to the amount of information that is extracted from the stimulus (Kok, 1990; Tueting, Sutton, & Zubin, 1970), and this amount is higher when there is a mismatch with expectations. Likewise, if the P3 is interpreted as a reflection of context updating (Donchin, 1981) or resetting the short-term memory (Grossberg, 1984), then a larger P3 suggests that there is more to be updated. One possible form of an expectation would be a template of the coming stimulus. In this experiment, the overall frequency of each target color was 25%, but if an informative prime is interpreted, the chance of each target that corresponds with the prime rises to 40%, while the chance of each disconfirming target drops to 10%. This means that it was not sufficient to prepare for one possible target. Therefore, the representation may have been at a more abstract level, so that a single expectation could cover the probability of two possible targets. Another possibility is that the P3 was larger because stimuli were analyzed in more detail if the earliest output from the perceptual process conflicted with the expectations in a later process.

There was a marginal effect of Contrast ($p < .1$) reflecting a smaller P3 for low-contrast stimuli than for high-contrast stimuli. This difference could reflect a difference in processing requirements for the perceptual analysis (see Kok, 1990), or be the result of more latency jitter in the low-contrast condition. Finally, there was a four-way interaction of Validity \times Block type \times Information value \times Contrast ($F(1, 6) = 11.84, p < .02$) that seemed to be the result of a relatively large validity effect ($4.0 \mu\text{V}$) in the low-contrast condition on the pure informative blocks. This effect was larger than on mixed blocks ($1.7 \mu\text{V}$). This could be the result of the overall frequency of a mismatch of the target with the target that corresponded with the prime, which was lower in pure informative than in mixed blocks. The frequency of a mismatch of expectations is a well-known contribution to the P3 amplitude (Duncan-Johnson & Donchin, 1977).

After the initial response preparation on the basis of the prime, the target analysis started to replace any incorrect preparation for an intentional response. It could be seen from the P3 amplitude that the target analysis was affected by previous preparation effects. The next step is to see how the speed of different target-based processes was affected. There were large RT effects of Validity, Information value and Contrast, and the pattern of interactions suggests that the prime was used for preparation, which was modulated on the basis of the information value of the prime. It was not clear from the RT results whether the modulated preparation was confined to motor processes or was broadened to perceptual processes. This issue is investigated with latency measures that reflect the preparation effects on different processing levels. The latencies are summarized in Table 1.

P3 latency

There was a 50 ms effect of Contrast on P3 latency ($F(1, 6) = 13.06, p < .02$) in line with the common finding that P3 is sensitive to perceptual manipulations (Donchin, 1981). The Validity effect on P3 amounted to 52 ms ($F(1, 6) = 37.47, p < .001$), and there was no main effect of Information value ($p > .6$). More specifically, the Information value effect was modulated by Validity, in that the P3 latencies of the noninformative conditions fell between those of the informative conditions. Furthermore, the effect of Validity on P3 was larger for informative (73 ms) than for noninformative conditions (31 ms). This was confirmed by the Validity \times Information value interaction ($F(1, 6) = 8.19, p < .03$). Planned comparisons showed that the Validity effects were significant in both the informative ($p < .005$) and the noninformative conditions ($p < .02$). A nonsignificant interaction of Validity \times Contrast ($F(1, 6) = 4.62, p < .08$) reflected a larger Validity effect for high-contrast (68 ms) than for low-contrast stimuli (35 ms). Finally, there was an interaction of Block type \times Contrast ($F(1, 6) = 10.94, p < .02$), reflecting a larger effect of Contrast in mixed blocks.

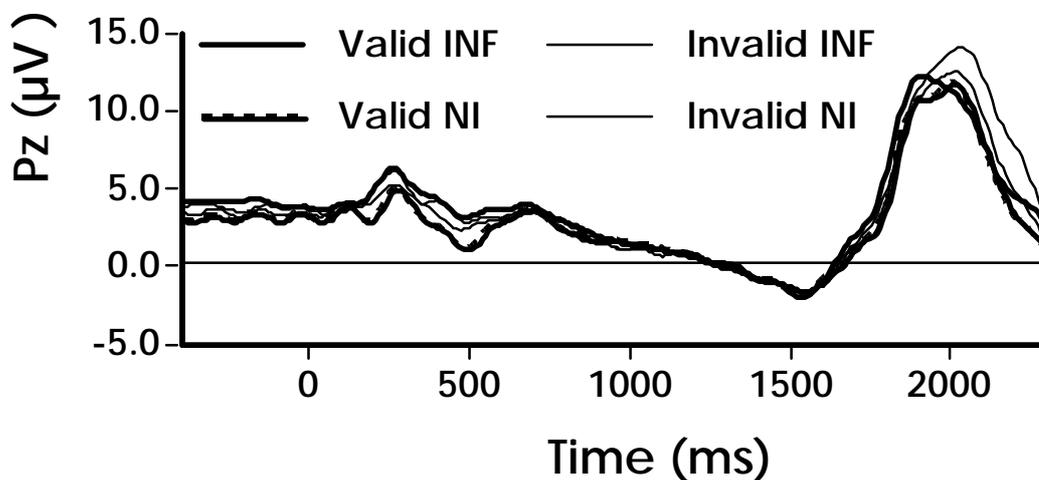


Figure 4: The average stimulus-locked waveform on Pz. Prime onset is at 0 ms, target onset is 1500 ms. NI = noninformative; INF = informative

The P3 results (see Figure 4) suggest that the validity effect was in large part present before the production of the P3. The view that P3 latency is sensitive to manipulations until or during stimulus evaluation (Donchin, 1981), would imply that there was perceptual priming. More specifically, this implies that subjects translated the response prime to expectations for two of the four possible stimuli. Given that even effects of stimulus probability, rather than response probability, are commonly assigned to response selection and motor processes (Sanders, 1980,

1990), an effect of response probability with a perceptual locus is surprising. However, the stimulus evaluation interpretation of P3 latency has been challenged, because some manipulations that are known to affect the speed of response selection also affect the P3 latency (see Verleger, in press, for a meta-analysis of P3-latency effects). In Verleger's framework, the P3-latency effects of Validity and Validity \times Information value could be interpreted as effects on the duration of the response-selection process, because a slow response-selection process is thought to cause a second overlapping P3 that delays the observed peak latency. For the moment, both interpretations of the validity effects can explain the data.

LRP onset

The duration of processes up till the first selective activation of the response hand is reflected in the LRP-onset. An ANOVA was performed on the estimated onsets relative to the stimulus in response-locked averages. The onset latency was 64 ms later for invalid than for valid conditions ($F(1, 5) = 88.39, p < .001$). There was also a 66 ms effect of Contrast on LRP onset ($F(1, 5) = 26.30, p < .005$). The effects of Block type and Information value were not significant ($p > .2$).

The validity effect was larger for the informative (114 ms) than for the noninformative conditions (13 ms). This was confirmed by the Validity \times Information value interaction ($F(1, 5) = 11.46, p < .03$). Planned comparisons revealed that validity effect was significant in the informative ($p < .001$), but not in the noninformative conditions ($p > .4$). This difference suggests that subjects did not start response activation on the basis of noninformative primes, whereas they did so on the basis of informative primes. In addition, there was an effect of Block type \times Information value ($F(1, 5) = 8.07, p < .04$); indicating a relatively late onset in pure noninformative blocks relative to other conditions. The prediction that incorrect preparation would last longer if perceptual analysis was delayed and when primes were informative received weak support from the nonsignificant effect of Validity \times Information value \times Contrast ($F(1, 5) = 4.32, p < .1$). It indicated that the informative low-contrast condition induced a larger validity effect (146 ms) than the informative high contrast (82 ms) or noninformative conditions (13 ms).

In summary, the effects on LRP were comparable to those that were found on RT. This suggests that the effects took place before the start of correct lateralization. To test this implication, the time interval between LRP onset and RT was compared across conditions (cf., Osman et al., 1995). The duration of this interval can be explained as an index of the speed of processes between the start of activation and the execution of the response.

Figure 5 shows response-locked average LRPs that illustrate how fast the preparation of the response rose from activation onset until execution. An ANOVA on the LRP-onset to RT interval duration (average 129 ms) showed no significant effects of Information value ($p > .7$), Block type ($p > .1$) or Validity ($p > .9$). Thus, there was no support for a difference in the speed of activation as a result of preparation of the primed response. This finding makes it more plausible that the preparation effects are confined to early processes. Surprisingly, there was a significant 13 ms difference between high-contrast and low-contrast conditions ($F(1, 5) = 16.69; p < .01$); and an interaction of Block type \times Information value ($F(1, 5) = 19.92, p < .01$) that indicated that the activation was 36 ms faster on pure noninformative blocks than on mixed noninformative blocks.

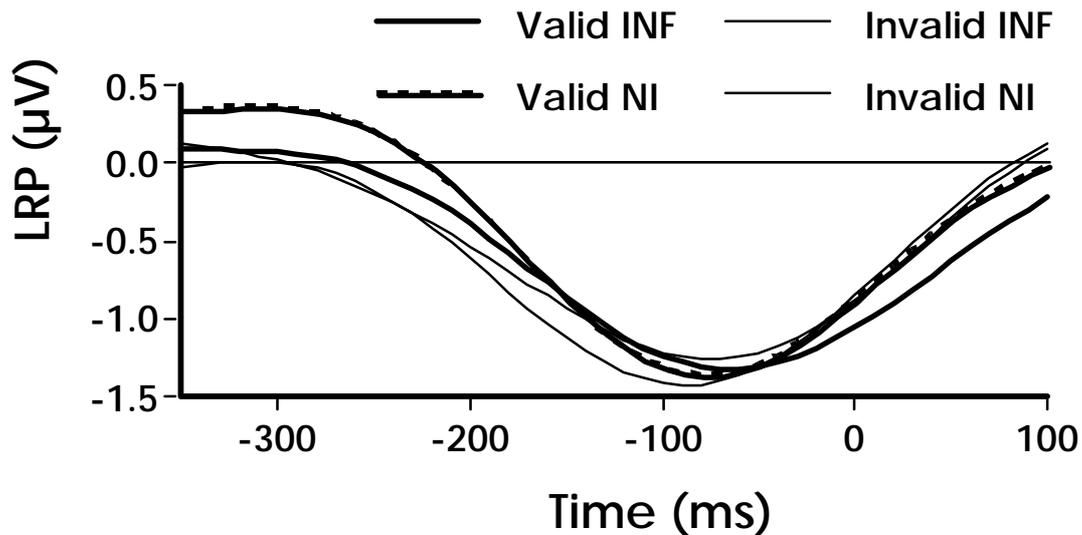


Figure 5: The average response-locked LRPs. Preparation for the correct hand is negative. Zero ms represents the response time. NI = noninformative; INF = informative

EMG latencies

EMG-onset data showed almost the same pattern of effects as reaction time (see Table 1 and Figure 6). The time difference between EMG onset and RT varied between 81 and 97 ms for all conditions. There was an effect of Validity ($F(1, 6) = 52.12, p < .001$); an effect of Information value ($F(1, 6) = 6.52, p < .05$); and an effect of Contrast ($F(1, 6) = 23.91, p < .005$). The effect of Validity \times Information value ($F(1, 6) = 45.55, p < .001$) resembled the strategic preparation effect on RT. Planned comparisons confirmed the existence of validity effects in both the informative ($p < .001$) and the noninformative conditions ($p < .02$). Unlike the RT, the EMG data displayed a trendwise interaction of Information value \times Contrast ($F(1, 6) = 4.31, p < .09$), suggesting that the speed gain from the prime information was smaller for high than for low-contrast targets.

In summary, a validity effect and a difference between informative and noninformative conditions were found on latency measures starting with P3 latency. A reasonable assumption is that the advantage of priming at an early level of information processing delays the onset of later levels, so that the same effect that is found on the latency of perceptual processes should also be found on the latency of motor processes. Unfortunately, it is not possible to compare the absolute effect sizes here, because the indices of the different processing levels have different metrics. That is, while RT and EMG effects are differences in the mean, P3 peak latency is more sensitive to modal differences (see Verleger, in press), and response-locked LRP onsets are affected most by the trials with the slowest activation (see Smulders et al., 1996). Nonetheless, Figure 6 shows a high similarity between LRP onsets, EMG onsets and RT, while P3 results point in the same direction. It can be said that large strategic preparation effects were present in or before the processes reflected by P3, and there is an indication that the difference between informative and noninformative conditions increased slightly afterwards.

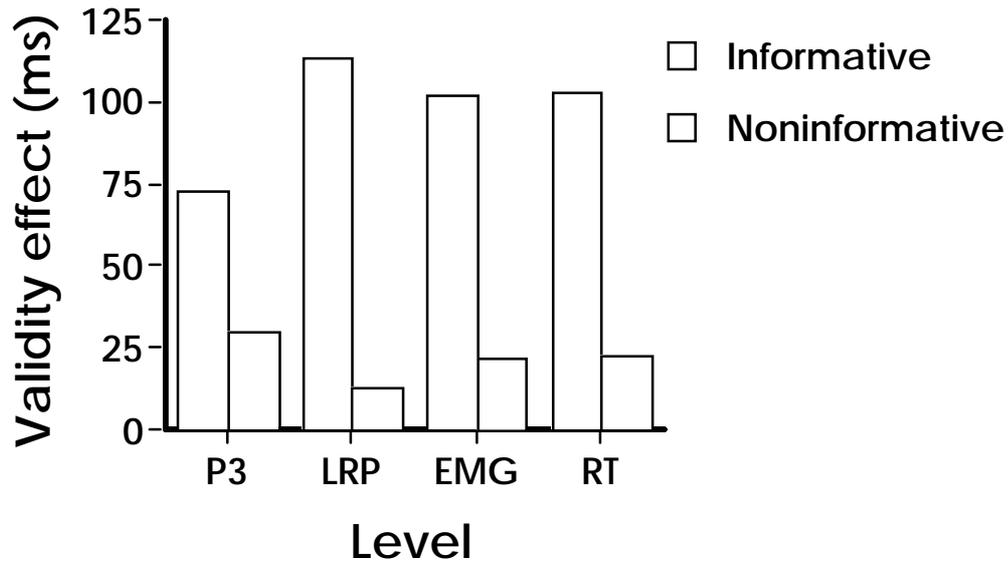


Figure 6: Validity effect sizes for the latency of the peak latency of P3, LRP onset, EMG onset and mean RT. Effect size is the delay of invalid versus valid conditions.

Incorrect EMG

Validity effects could be observed on the direction of LRP, whereas the interval between LRP onset correct and RT was not affected. The interpretation that the preparation effects were confined to early processes was tested by comparing the number of trials on which the incorrect preparation propagated to the level of EMG. The number of correct responses with incorrect initial EMG activity was compared. The criterion for EMG activity was lowered to two standard deviations of baseline activity above the mean. As a result, the absolute number of EMG-trials may have overestimated the amount of incorrect execution, but there is more resolution for the comparison of probabilistic priming conditions.

An ANOVA on the percentage EMG-trials revealed an effect of Validity ($F(1, 6) = 21.15, p < .005$), which indicated 3.8% more EMG trials on invalid than valid priming conditions. The effect of Contrast ($F(1, 6) = 11.97, p < .02$) confirmed that there were 1.7% more EMG trials on low than on high contrast conditions. There was also a marginal difference ($p < .1$) between pure and mixed block types. There was an Information value \times Validity interaction ($F(1, 6) = 17.90, p < .01$), that consisted of a 6.1% validity effect for informative, versus a 1.4% effect for noninformative conditions. Finally there was a Validity \times Block type effect; $F(1, 6) = 6.81, p < .05$; and an Information value \times Contrast effect; $F(1, 6) = 6.02, p < .06$. This pattern of EMG trials can be conceived of as support for the existence of late effects of preparation, and it undermines the conclusion from the absence of such effects on the LRP - RT interval that preparation effects were confined to early processes.

In summary, a consistent pattern of validity effects was found across dependent variables. By far the most striking effect is the interaction of Validity and Information value, that points to a large validity effect when primes are informative, in contrast to a small validity effect when primes are noninformative. The absence of meaningful differences between pure and blocked trials indicates that the subjects were only preparing the primed response if the chance of a match was high, rather than that priming effects result from bottom-up contingency reinforcements.

Inhibition Effects

It was concluded in the previous section, that the correct response is hampered to a larger extent if the invalid prime is used for strategic preparation of a response. Nonetheless, subjects were

able to resolve the conflict at a level that is reflected by P3 latency, and even inhibit the incorrect response when it was already prepared until the levels that are reflected by LRP and EMG. In the next section, the inhibition in change conditions (invalid informative) is further investigated through a comparison with the inhibition in a no-go condition. These two types of inhibition are presumed to be different because the former selectively affects the primed response while leaving the correct response intact, whereas the latter can affect all response-related processes in a nonselective way.

The most meaningful results in the investigation of inhibitory efficiency and inhibitory success are results about responses that escape inhibition at different levels. On the extreme end, inhibition fails if the overt response is not correct. Following the signs of incorrect preparation during the response process, the possible locus or loci of inhibition are uncovered.

Accuracy

Table 2 shows the accuracy of the informative and disjunctive priming tasks. An ANOVA on the accuracy data of no-go trials and high- and low-contrast change trials showed a difference between conditions; $F(2, 12) = 8.41, p < .01$. The accuracy on no-go trials was marginally higher than on high-contrast change trials ($p < .07$), and the high-contrast trials were performed with higher accuracy than the low-contrast change trials ($p < .02$). Errors on no-go trials as well as errors on invalid priming trials consisted of responses with the primed hand for more than 95% of all errors.

The hypothesis that two different mechanisms are involved in nonselective and in selective inhibition implies that they both have their own source of variance in inhibitory efficiency. This leads to the prediction that the accuracy of conditions that call for the same mechanism should be correlated.

Table 3: The correlations between the error rates on no-go and invalid informative priming conditions (above the diagonal), and the Student's $t(6 \text{ df})$ of the correlation values (below the diagonal).

	No-go	High INF	Low INF
No-go	--	$r = 0.08$	$r = 0.55$
High INF	$t = 0.18$	--	$r = 0.75$
Low INF	$t = 1.49$	$t = 2.57$	--

Note: High and low denote the contrast conditions. INF = informative priming.

Table 3 shows to what extent the no-go and change conditions were correlated. It can be seen that there is a substantial interrelationship ($p < .03$) between the two change conditions, and a moderate relationship ($p < .1$) between the accuracy on low-contrast change trials and no-go trials, but a much weaker relationship ($p > .4$) between no-go and high-contrast change trials. These data are consistent with the tentative explanation that inhibition on no-go trials and inhibition on high-contrast change trials were exerted with different mechanisms, and that the inhibition on low-contrast change trials includes two modes of inhibition.

Table 4: The P3 peak amplitude in μV on central and parietal midline leads in the valid priming conditions and three inhibition conditions

Condition	Cz	Pz
All valid	9.5	13.4
No-go	14.9	22.0
Change high contrast	11.2	15.4
Change low contrast	9.9	16.1

P3 amplitude

The P3 amplitude on Pz was larger for no-go stimuli than for go stimuli in the disjunctive task; $F(2, 12) = 59.60, p < .001$. No-go P3s differed from change P3s; $F(2, 12) = 10.29, p < .003$. Pairwise comparisons showed that the no-go P3 was larger than that of low-contrast change trials ($p < .02$), whereas the two change conditions did not differ significantly ($p > .6$). The differences between inhibition trials and noninhibition trials could reflect the overlap of P3 with a different inhibition-related ERP component. For example, De Jong et al. (1990) found a positivity overlapping with P3 that was unique to stop conditions. This positivity reached a maximum over fronto-central electrode positions. However, it can be seen from Table 4 that the P3 enhancement at central electrodes was smaller than at parietal electrodes for all inhibition conditions. Thus, there is no reason to associate the P3 enhancement with an anterior contribution. Instead, it is more parsimonious to attribute the enhancement to the same processes that underlie the normal P3.

An unsolved issue is why the no-go condition caused a larger P3 enhancement than change conditions. It could be argued that the no-go stimulus was an infrequent stimulus, and that this caused an oddball-effect. However, the frequency of the no-go stimulus was not very different from that of other colors. In the disjunctive task every stimulus was presented on 20% of all trials. Therefore, the color of the stimulus alone cannot have caused a larger oddball effect on no-go trials than on go trials. An alternative would be that the P3 enhancement was related to the amount of information contained in the stimulus or a combination of the color and the amount of information. However, the amount of information in the no-go condition was equal to that in change trials. Furthermore, given that the subject has detected a mismatch of the prime and target information, the expectation of the no-go color would still be twice as high as the expectation of each color of the change condition. Therefore, there was no support for an explanation of the no-go effect on P3 in terms of stimulus frequency.

LRP amplitudes

A strong case for the distinction between central and noncentral inhibition could be made if the LRP results for change trials would show attenuation of lateralization for the primed hand, whereas the LRP would be full-grown on no-go trials. De Jong et al. (1995) based such a comparison on the hypothesis that the LRP amplitude at the moment of triggering a response is approximately constant (Gratton et al., 1988). In order to lend credibility to this hypothesis for the present purpose, we first compared the LRP-level associated with responses on the go-trials between all the informative and noninformative conditions.

The amplitude differences between conditions were small. The average LRPs of the two Validity levels, as well as the two Information-value levels and the two Contrast levels were all between 1.64 and 1.69 μV .⁵ In short, the data showed high consistency in the levels of LRP preceding the response.

Because the amount of activation for the primed hand was rather variable between subjects, a regular comparison between activation for the primed hand and the mean amplitude during activation would not have enough power to detect attenuation. The standard error of the LRP amplitude in the change conditions was 0.36 and 0.44 μV . In the larger set of go-conditions that served as a criterion, the standard error was reduced to 0.14 μV . Therefore, the group average of peak amplitudes was compared to a criterion, set at mean + 2.5 standard error of the averages from go-conditions.

⁵There was a marginal difference between LRP amplitudes on pure vs. mixed conditions; $F(1, 6) = 5.00, p < .07$, with mixed conditions containing larger amplitudes. Furthermore, there was a Validity \times Information value \times Contrast interaction; $F(1, 6) = 6.29, p < .05$; which we believe to be a type I error, because there was no clear pattern in the data.

For the change conditions, response-locked averages were used for both the criterion and the tested inhibition signals. Of course, correct no-go LRPs can not be averaged response-locked. Therefore, the criterion for no-go LRPs was calculated from stimulus-locked averages of go LRPs. The lower thresholds amounted to $-1.31 \mu\text{V}$ in the response-locked averages and $-0.60 \mu\text{V}$ in the stimulus-locked averages.

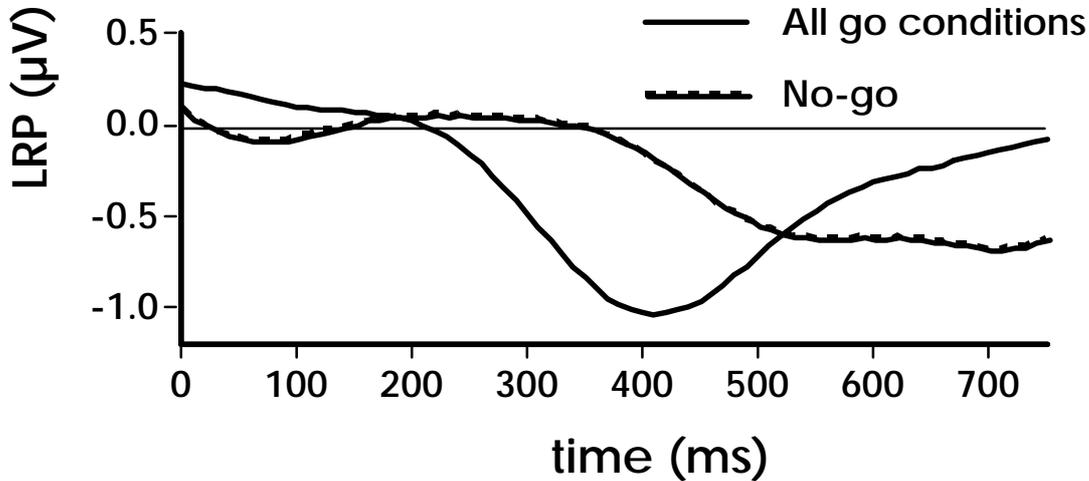


Figure 7: The average stimulus-locked LRPs of all response trials, synchronized across conditions to individual subjects' mean RT, and the comparable section of the no-go LRP. Preparation for the primed hand is negative.

In the no-go task, where LRPs were predicted to exceed the threshold for responding, the amplitude did not pass the $-0.60 \mu\text{V}$ level until 560 ms post target, and reached a peak of -0.669 after 730 ms (see Figure 7). In comparison with the mean RT on go-trials (392 ms), and the speed of commission errors on no-go trials (330 ms), this activation was too late to argue that the LRP passed the threshold. This indicates that nonselective inhibition on no-go trials was manifest at a central level, as expressed in the attenuated LRP amplitude, thus contradicting De Jong et al.'s prediction of full-grown LRPs.⁶ Based on these LRP results, it is hard to maintain that the inhibition on no-go trials was exclusively exerted after the central processes that lead to an LRP.

In the change condition, the LRP for the primed hand was predicted to remain under the criterion of $-1.31 \mu\text{V}$ for response-locked averages. Subjects would need to suppress the incorrect activation before the LRP would exceed the threshold, because it is not possible to use the alleged nonselective inhibitory mechanism downstream from the motor command. On average, the maximum LRP-amplitude for the primed response in an interval of 300 ms prior to RT crossed the criterion. Post hoc comparisons indicate that the incorrect activation exceeded the threshold for low-contrast ($-1.73 \mu\text{V}$), but not for high contrast change trials ($-0.85 \mu\text{V}$) (see Figure 3); although the difference between these two conditions was not significant ($p > .1$). Apparently, there was more opportunity for building up incorrect activation during the extended perceptual analysis in

⁶It could be argued that the no-go LRP did not reach a peak so early due to a selection of responses that are correctly inhibited. Inhibition is more likely to fail if the response process is relatively fast in the race against the inhibitory process, whereas correct trials more often result from slower response processes (cf. Logan & Cowan, 1984). To test this possibility, we calculated the mean of the longest RTs in the go-RT distribution, assuming that the go-RT distribution is a valid approximation of the duration of response processes on no-go trials. For example, if a subject responded on 26% of all no-go trials, the no-go trials were compared to the upper 74% of go-RT distribution. The mean of that subset was 409 ms for high contrast trials and 449 ms for low contrast trials. This indicates that the LRP on no-go trials remained significantly smaller than the LRP-amplitude that is associated with overt responses until at least 100 ms after the highest estimation of the duration of response processes.

low-contrast conditions. Thus, it could not be demonstrated that selective inhibition is associated with an attenuation of activation before the LRP is generated.

EMG as partial responses

It has been argued by De Jong et al. (1990), that the occurrence of EMG on correct inhibition trials supports a possible locus of nonselective response inhibition at a peripheral level. In contrast, a central mechanism is thought to be required for selective inhibition (De Jong et al., 1995). Unfortunately, De Jong et al. (1995) do not report the amount of EMG on the selective stopping task.

The distinction between a central selective and a peripheral nonselective inhibitory mechanism has implications for the pattern of EMG results that should be expected. In the present experiment, the change condition should show few, if any trials with EMG for the primed hand in the absence of an overt erroneous response. By contrast, the no-go condition should show a substantial amount of trials with EMG in addition to response trials.

The proportion of trials with EMG for the primed response in the absence of an overt error was compared between conditions in an ANOVA with the factors condition (no-go, high-contrast change and low-contrast change). When a lenient criterion of two standard deviations above the mean of baseline EMG was used, there was a significant difference in this type of partial errors; $F(2, 12) = 14.54, p < .001$. There were more partial errors for no-go trials (34.3%) than for change trials (16.0%), but there was no significant difference between low and high-contrast change trials (0.4%; contrast: $p > .8$).

Because the overall error rate on no-go trials was even lower than that on change trials, the differences in partial responses cannot be explained as a consequence of overall differences in accuracy. Rather, the difference in partial responses suggests that the inhibition on no-go trials was in large part exerted at the last moment, whereas the inhibition on change trials was more often exerted at an earlier point. However, the ANOVA in the Section "Preparation effects" showed that there was indeed a difference between conditions that did and conditions that did not evoke incorrect preparation. This implies that some inhibition took place during EMG production on change trials, and that the EMG trials were not all due to random EMG activity. Although the difference between no-go and change trials is consistent with De Jong et al.'s (1995) distinction between nonselective and selective inhibition, the mere presence of late inhibition on change trials can not be explained with their model.

Thus, an interesting pattern of results occurs. The go conditions allowed for a faster response than the informative valid conditions, so the activation is likely to start earlier. However, the inhibited no-go commission error responses are associated with a weaker LRP than the primed but inhibited change responses. These results are opposite to those of De Jong et al. (1995).

GENERAL DISCUSSION

The present experiment investigated the possibilities for two types of top-down effects on the response process; strategic modulation of preparation and suppression of response preparation. The first type of top-down effect, strategic preparation for a probabilistic response requirement, was investigated with a response-priming task in which the response prime pointed to the correct response for the upcoming target on 50 or 80 percent of all trials. The results converge on the explanation of strategic preparation in terms of early processes.

Showing strategic modulation of preparation starts with showing that there was preparation at all. There were clear effects of the validity of primes on speed, accuracy and the latency of all the tested psychophysiological indices, comparable to what is generally found in the response-priming literature (e.g., Gehring et al., 1992; Hackley et al. 1990). It suggests that, conscious or not, the

subject is biased toward the primed response. The effect of priming in general could be found at the levels indicated by P3 latency, LRP onset, EMG onset and RT. However, there was no sign of activation of the primed hand during the foreperiod.

The absence of LRP during the foreperiod deviated from similar studies of Gehring et al. (1992) and Gratton et al. (1992). A simple explanation of this difference is that subjects emphasized speed over accuracy to a larger extent in previous literature than in the present experiment. As a result, the activation of the primed response would need to start before the onset of the target. A deeper investigation would take other signs of preparation during a foreperiod into account. Several other experiments show that preparation for motor action remains restricted to the moment where it is useful. In a simple RT task, Gottsdanker (1975) found that subjects could improve their performance with the help of a warning stimulus. However, subjects did not make full use of the warning stimulus in the case of a low stimulus probability. In addition, performance decreased if the interval between the warning and imperative stimuli was longer or shorter than intervals with a higher probability (cf. Alegria, 1975). Gottsdanker concluded that preparation is an aversive state that is avoided unless the momentary probability of a stimulus onset is high.

Van der Molen, Boomsma, Jennings, and Nieuwboer (1989) observed an anticipatory heart rate deceleration in the foreperiod of a warned go/no-go task. This deceleration is considered to be associated with the allocation of resources for stimulus processing. The deceleration and other preparatory physiological changes were in large part delayed until just before the response. However, the preparatory changes were more gradual if the duration of the foreperiod was variable. Hence, the expectancy of the imperative stimulus seems to be the dominant factor to determine whether subjects start activating a response (Niemi & Näätänen, 1981). In the light of these other results of preparation during the foreperiod, the absence of LRP is less surprising.

For the investigation of top-down influences it is more relevant that there were clear signs of strategic modulation of the magnitude of preparation. It could be observed on performance measures that subjects prepared the primed response during the early phase of target analysis if primes were informative, but much less so if they were noninformative. The alternative would be to explain this difference in terms of bottom-up processes. For example, as a result of an increased usefulness of the prime in the informative conditions subjects might tend to base their responses more on the prime, without making a strategic choice to do so. Such a bottom-up explanation is not very plausible because the difference between the validity effects in pure blocks of informative and noninformative conditions was replicated in blocks with a mixture of both conditions. The presence of a solid interaction of Information value \times Validity in mixed blocks indicates that subjects adjusted the degree of preparation for the primed hand from trial to trial, on the basis of the probability information that was contained in the color of the prime. The conditional-accuracy functions showed that subjects made much more use of the prime on fast than on slower responses, and that a delay of target identification by way of a contrast manipulation could prolong and enhance the influence of strategic priming effects.

Psychophysiological measures were used to investigate in more detail what kind of strategic preparation was responsible for the performance results. Although the primes contained information about the response, activation of the primed hand was confined to the situations where this was useful. Response activation of the invalidly primed hand following the target onset was restricted to informative conditions, as can be judged from the absence of incorrect lateralization and validity effects on noninformative priming conditions. Therefore, the probability information must have been applied to earlier processes, such as the speed of perceptual evaluation or central processes between target identification and response activation. The probability-dependent validity effects on P3 latency indicate that there was probably some perceptual priming. This finding is quite surprising, because it is complicated to translate an arrowhead prime to an expectation for two very

dissimilar color patches out of a set of four. Still, the presence of substantial validity effects on P3 latency supports a perceptual locus (Donchin, 1981).

Recently, however, it has been argued (Falkenstein, Hohnsbein, & Hoormann, 1994; Ragot & Fiori, 1994; see Verleger, in press for a review of this position) that the P3 sometimes consists of two overlapping components, the first of which can be interpreted as a time index of stimulus evaluation. The second component is thought to be absent on tasks with a direct stimulus-response (SR) route, but to be present when tasks require an active SR translation (cf. Frith & Done, 1986; Kornblum et al., 1990). In this view, the latency of the P3 peak may be sensitive to the duration of processes after stimulus evaluation. In that case, the validity effects are consistent with effects on central processes preceding the activation of the response, which were biased towards the primed response. A plausible model consistent with this interpretation of the P3 effects is that subjects recruit the motor program for the primed hand (cf. Rosenbaum, 1980) more often if the chance of validity is higher. This type of preparation was constrained for noninformative conditions, however, because suppressing the incorrect and subsequent recruitment of the correct motor program would lead to a slower response than recruiting of the motor program on the basis of the target.

In summary, the strategic modulation of validity effects started before response activation. However, LRP lateralization for the invalidly primed hand on informative, but not for noninformative trials, suggests that some degree of activation took place that was modulated strategically. At the same time, the absence of lateralization during the foreperiod questions the magnitude of strategic activation, and suggests that the activation is not triggered before target onset.

An integrative view holds that there was preparation on informative priming conditions that led to the selection of a response (cf. Sanders, 1990) or presetting of response parameters (Rosenbaum, 1980). Some time after target onset, any partially prepared response was activated. If the invalid informative prime had preset the incorrect response, there was initial activation of the incorrect response. In contrast, the response program was typically not prespecified in the noninformative conditions, because such a program would have a high chance of being incorrect. Thus, a difference in the amount of incorrect activation between informative and noninformative conditions can be explained as a consequence of strategic preparation in central processes that precedes the activation of responses. This interpretation is not entirely consistent with that of Gratton et al. (1988), who suggested that primes can activate an entire response channel, which they defined as “the complex of structures whose activities are more or less directly related to the mechanical event that is defined as the overt response”. This definition includes the process that translates the stimulus to the response, as well as later motor processes. Gratton et al.’s interpretation can not explain why the activation of primed responses as measured on the LRP in informative conditions was delayed until some time after the target onset. Furthermore it is not able to explain why the activation of the primed response in noninformative conditions was absent while there was a validity effect on EMG latency and RT.

A dissociation of response priming effects and presence of LRP was also discussed by Miller, Coles, and Chakraborty (1996). They found that in a given state of preparation for a response on a primary task, there was no LRP lateralization. However, if a probe was presented during the same state of response preparation, the RT to the probe was faster for responses that were consistent with the primary response, than for responses with the opposite hand. While the LRP finding suggested that there was no preparation for a response on the primary task, the probe RT finding showed that there was a preference for one response over the other. Miller et al. interpreted this dissociation on the basis of Rosenbaum’s (1985) hypothesis that there are at least two steps in the preparation of a response. In the first step, a motor program is selected, and in the second step the parameter values are specified. The priming effects that could be measured on probe RT are attributed to the selection

of the motor program, whereas priming effects on LRP lateralization can only be observed if the parameters are specified.

The hypothesis that the strategically modulated preparation effects were present on early processes is also consistent with the finding of validity effects on the amplitude of P3. In several interpretations the P3 amplitude reflects the subjective probability of the stimulus (cf., Duncan-Johnson & Donchin, 1982; Grossberg, 1984). The results suggest that based on informative primes subjects developed an expectation or schema. The schema of the probable event is then mismatched by the target, leading to a larger P3 amplitude than in the case of a match (cf., Donchin, 1981)

The second type of top-down effect on speeded task performance that we tested, was inhibitory control of primed responses. A comparison was made between the inhibition on no-go trials, and the allegedly selective inhibition on invalid informative (change) trials. We included the latter condition because the literature has shown that there is substantial preparation for the primed response on invalid informative trials, but not on noninformative trials (e.g., Gehring et al., 1992), and this difference was confirmed in the first part of the experiment. In this respect, the condition required active suppression of the response that was primed, in favor of the correct response. The no-go trials were chosen to constitute a nonselective-inhibition requirement. The two conditions correspond to the inhibition conditions of the stop paradigm and the stop-change version of this paradigm, where one response should be replaced by another (De Jong et al., 1995; Logan & Burkell, 1986).

It is important for the comparison of our results with those in the stop-signal paradigm that there was a strong tendency to respond on inhibition trials. On no-go trials, the proportion of errors was limited, but there was a large number of trials with EMG for the primed response, which suggests that there was ample preparation and activation of the response to investigate the inhibition process. On change trials, there was a high error rate, supplemented with partial errors and strong lateralization for the primed hand, so that there was a clear need to suppress incorrect preparation for the execution of a correct response.

Knowing that subjects had a strong tendency to respond with the primed hand, we looked at the locus of response inhibition. The error rates suggested that primed responses were somewhat easier to withhold in the no-go than in the change condition. In addition, the number of correct trials with incorrect EMG suggests that late inhibition during the execution of the response was quite successful on no-go trials, but much less so on change trials. These findings are consistent with the view that selective inhibition, as used on change trials, is exerted at an earlier locus than nonselective inhibition.

More specifically, De Jong et al. (1995) argued that selective inhibition takes place at a level before or during LRP production, whereas nonselective inhibition can take place after LRP production, for example by interception of the command that energizes the programmed response at a peripheral level (Bullock & Grossberg, 1991). Their interpretation was based on the finding that the LRP for the primed hand on correct stop-change trials did not exceed the amplitude that would otherwise be associated with a response, whereas the stop-all LRP was large enough to conclude against attenuation at a central locus.

In the present experiment, however, the LRP for the primed hand on change trials was not attenuated. Although it is hard to argue against the central-attenuation point of De Jong et al. (1995) on the basis of a failure to reject the null-hypothesis that change LRPs were equal to response LRPs, it is a telling result that the mean LRP amplitude for the primed hand on change trials actually exceeded the mean amplitudes before correct responses. On the other hand, the LRP on no-go trials remained well under the amplitude of response trials. These results are not consistent with the distinction between central selective and peripheral nonselective inhibition. The data show that the

LRP for the primed response exceeded the response amplitude, but that the actual response was correct. This implies that responses are inhibited after the LRP is produced on change trials.

The finding of a larger incorrect LRP on change trials in the present experiment than in De Jong et al.'s (1995) experiment is hard to explain as a difference in the size of the response tendency. In the stop-signal paradigm subjects are allowed to respond on approximately half of all the stop trials, which is even higher than in the present experiment. Subjects have to start the reaction process for the initial response on De Jong et al.'s stop-change condition, whereas they can delay or weaken the reaction process on the present change conditions. This would suggest that the LRP would be higher in the stop-change condition than in the change condition. A tentative explanation of the difference is that in De Jong et al.'s task, responses needed to be suppressed at a central level, because there had previously been a primary task stimulus that cleared the way for execution. In contrast, the change condition in the present experiment allowed for any amount of activation of responses, until a signal for execution is released (cf. Kornblum et al., 1990). The large change LRP may indicate that the execution of the activated response requires an additional triggering or gating process, that was postponed during response conflict.

A reduced amplitude of LRP on no-go trials is not uncommon. For example Miller and Hackley (1992; see also Band & Miller, Chapter 4; Osman et al., 1992) used two-dimensional stimuli with different processing complexity in a go/no-go task. A fast dimension allowed for preparation of the correct hand, although the slow dimension could carry a no-go instruction. In this task, there was LRP on no-go trials. However, the no-go LRP remained substantially lower than go LRPs.

It is also possible to explain the weak no-go LRP in the same framework as the preparation effects. It was suggested in the discussion of priming effects that response activation goes through two steps. In the first step the response program is selected, and in the second step, a setting of parameters results in lateralization of the LRP. On change trials, subjects start activating the response some time after the onset of the target, regardless of whether there is a preference for the correct or incorrect response. The activation is required in order to execute the correct response in time. Depending on the interaction between the primed response and the response that is indicated by the target, the response that is executed may or may not be correct. It is not efficient to wait until the correct response has been selected before activation is allowed. After the activation on LRP exceeded the criterion for execution, there was still another possibility to interrupt the execution. Until the signal for execution of the prepared response (cf., Kornblum et al., 1990) is given, the execution is prevented. The stop-change condition also first required the activation of the motor program. However, if the activation as reflected in the LRP exceeds the criterion, the response is executed, because the signal for execution is already given before the stop-signal becomes effective. In this condition, only trials where LRP remained below the criterion for execution led to successful inhibition. In the no-go condition the activation of the motor program could be suppressed; resulting in a small LRP. On trials where activation took place and led to a command for execution, there was still a possibility to inhibit the response on the level indicated by EMG. On stop-all trials, the activation of the motor program could not be prevented, because the stop signal was present much later than the command to execute a response. However, the activation of the motor program was allowed to exceed the criterion for execution, because there was still a possibility to inhibit the response after the LRP level.

In summary, the explanation of inhibition performance on the stop-signal paradigm and the present experiment incorporates multiple response-prevention or inhibition processes. Provided that the reaction process is not already put in motion, there are three options. First, the activation of a selected response does not always follow the selection of a motor program – this continuation is under subjective control. This option is supported by the attenuated LRP on stop-change and no-go

conditions. It is comparable to what De Jong et al. (1995) referred to as central inhibition. Second, the execution of a response can be interrupted between exceeding the activation criterion, indicated by LRP; and the onset of muscle activity, indicated by EMG. This option is supported by a conflict between the finding that the LRP on change trials exceeded the criterion for response execution, whereas there were few trials with EMG for the primed and an overt response of the correct hand. De Jong et al. did not find data that support this type of response inhibition, possibly because the response path was already opened for the primary response. Third, the response execution can be frozen after the onset of EMG activity. This late option is more effective for nonselective inhibition than for selective inhibition. It corresponds to what De Jong et al. referred to as a noncentral inhibition mechanism downstream from the production of LRP. However, De Jong et al. concluded that this late mechanism could only be used for nonselective inhibition, whereas the present experiment found change trials with a correct overt response, but with EMG for the primed response.

The P3 enhancement on inhibition trials was partially consistent with De Jong et al.'s (1990) studies. De Jong et al. found a positivity on inhibition trials overlapping the P3, with a maximum over Cz (cf., Eimer, 1993; Jodo & Kayama, 1992; Kok, 1986; Naito & Matsumura, 1994; Simson, Vaughan & Ritter, 1977). They speculated that this waveform might be related to a call for inhibition from the frontal cortex. In our data, there was a large enhancement of the P3 on no-go trials, and a smaller P3 effect on change trials. Furthermore, the enhancement was stronger on Pz than on other leads, which suggests that the enhancement originated from a different source than the one that De Jong et al. found. An enhancement of the P3 does not necessarily reflect an inhibitory process, although it could do so. After all, interpretations of the P3 such as the one of Grossberg (1984) hold that the P3 amplitude is related to the amount of new information contained in the stimulus and to the effort of resetting working memory. According to Donchin and Coles (1988) the P3 amplitude reflects the updating of subjects' strategies and expectancies.

In conclusion, there were consistent results in support of strategic preparation of early processes such as perceptual analysis and motor programming. Subjects were able to affect the efficiency of responding by developing an expectation about the upcoming target, or by presetting a motor program. This preparation was modulated on the basis of the probability of a valid prime.

After stimulus onset, the response program that was preset was activated on response trials, and was not always activated on no-go trials. If the preparation was incorrect, there were two late options for inhibition. The transfer of activation to execution could be interrupted, or the execution could be stopped.

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4 Mental Rotation Interferes With Response Preparation⁷

Abstract

Reaction times (RTs) and lateralized readiness potentials (LRPs) were studied to find out whether response preparation begins after mental rotation finishes, as assumed by discrete-stage models. Stimuli were disoriented normal or mirror-image characters, with character name determining which hand would respond. In Experiment 1, the normal/mirror-image information determined whether the response was to be executed ("go") or withheld ("no-go"), and LRPs indicated that responses were weakly prepared before the end of mental rotation. Mental rotation was not required in Experiment 2, and significantly more response preparation was observed. In Experiment 3, probe RT trials embedded in the mental rotation task indicated that hand information is available to the response preparation process during rotation. Apparently, some response preparation occurs before mental rotation finishes, but rotation interferes with response preparation.

Many models in cognitive psychology describe the mind's activities using the abstraction of processing stages or levels. In these models, each stage is thought to perform a separate function within the overall information processing sequence. In speeded choice tasks of the sort most commonly studied in the laboratory, for example, it is generally agreed that separate processes are responsible for perception of the stimulus, decision about which response is appropriate, and preparation and execution of the selected motor response (e.g., McClelland, 1979; Sanders, 1980; Smith, 1968).

A controversy has developed since the late 1970's between theorists proposing discrete transmission of information between serial stages (Donders, 1868/1969; Sanders, 1990; Sternberg, 1969) and theorists who support alternative continuous models allowing parallel processing (e.g., Eriksen & Schultz, 1979; McClelland, 1979). These opposing views have resulted in three main classes of models, with different descriptions of the information processing system and, in consequence, conflicting conceptions of the meaning of reaction time (RT).

Discrete models (e.g., Sternberg, 1969) are based on the assumption that only one stage can be active at a time, although separable aspects of a stimulus may be processed in parallel within a stage. The output of each stage is transmitted in a single portion, as soon as the stage has finished, but there is no transmission of preliminary stage output. According to this view, stages follow each other in strict sequence, without temporal overlap, and RT is the sum of the durations of the individual stages involved in the task.

Continuous models (e.g., Eriksen & Schultz, 1979; McClelland, 1979) reject the assumptions of discrete transmission and serial processing, instead allowing stages to receive partial inputs and begin processing on the basis of such inputs. Such models have strong appeal, because they seem

⁷ Authors Note: This chapter is in press with *Journal of Experimental Psychology: Human Perception and Performance*, with Guido P.H. Band as the first, and Jeff Miller as the second author.

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consistent both with certain aspects of neural physiology and with certain continuous aspects of performance data (e.g., the speed-accuracy trade-off), although some have argued that these facts can easily be reconciled with discrete models (e.g., Meyer, Irwin, Osman, & Kounios, 1988; Miller, 1988). According to McClelland's Cascade model, for example, a level or stage of stimulus processing (e.g., stimulus identification) produces preliminary partial output that immediately becomes available to the next processing level. While the first level continues to process, the second level can start working with the preliminary information; thus, the two levels operate in parallel. In such models, total RT depends on many factors, because activation driving the response effector accumulates gradually as a function of activation several levels back in the system; RT is not simply the sum of the times needed for each of the individual levels.

The third class of models contains architectures that are neither fully discrete nor fully continuous, but rather are hybrids that share some features of each of these two extremes. For example, Miller (1982, 1988) suggested that intermediate models could be regarded as differing in the size of information packets transmitted from one stage to the next – "grain size" – with discrete and continuous models representing extreme positions. Grain size is infinitesimal in continuous models like that of McClelland (1979), because any arbitrarily small bit of partial output immediately begins to have an effect on the next processing level. In contrast, discrete models require maximal grain sizes, because they assume that all stimulus information is transmitted in a single packet. Based on an analysis of packets transmitted from the perceptual system to the response system, Miller (1982) proposed the intermediate Asynchronous Discrete Coding (ADC) model, in which the grain size of information is the stimulus attribute (e.g., size, shape, color). In this model single-attribute stimuli are transmitted discretely, but the various attributes of multi-attribute stimuli may be transmitted separately, allowing different stages to operate with some temporal overlap. Unlike continuous models, partial information in ADC models is not constantly available for the subsequent stage, because only complete attributes may be transmitted. Other, more quantitative, hybrid models have been developed from models based on scheduling and queuing theories (e.g., Fisher & Goldstein, 1983; Miller, 1993; Schweickert, 1978).

Many studies have been designed to compare discrete and continuous models by attempting to find out whether response preparation processes can begin before perceptual analysis has finished (e.g., Coles, Gratton, & Donchin, 1988; De Jong, Wierda, Mulder, Mulder, 1988; Miller & Hackley, 1992; Miller, Riehle, & Requin, 1992; Osman, Bashore, Coles, Donchin, & Meyer, 1992; Smid, Lamain, Hogeboom, Mulder, & Mulder, 1991). Such preliminary response preparation would never be allowed by discrete models, but would often be allowed by continuous models, and would be allowed under certain circumstances with each of the hybrid models. Contrary to discrete models, a number of results indicate that perception can overlap with response preparation, at least under some circumstances (see Miller, 1988, for a review), although there is still some disagreement about whether the overlap is most consistent with continuous models (e.g., Smid et al., 1991) or with a hybrid like the ADC model (e.g., Miller, 1982, 1983; Miller & Hackley, 1992).

Although the evidence just reviewed strongly suggests that response preparation can sometimes overlap with simple perceptual discriminations, it does not justify any general conclusions about the discreteness versus continuity of human information processing. One theoretically crucial limitation is that temporal overlap has only been demonstrated for one particular pair of processes (i.e., perceptual discrimination and response preparation). It is not safe to assume that the transmission of information from one process to the next is the same for all pairs of processes (Miller, 1988), so it remains an open question whether response preparation can also overlap with other mental processes. Another limitation stems from the restricted set of choice-RT tasks examined in this work, each of which required subjects to select a response according to a relatively simple stimulus-response (S-R) mapping that was held constant throughout the

experiment. It is easy to imagine that partial information might cause preliminary preparation of easily-selected responses, even if such preparation cannot occur in tasks for which response selection is more difficult (cf., Coles, De Jong, Gehring, & Gratton, 1991; Meyer, Irwin, Osman, & Kounios, 1988).

The purpose of the present study was to contribute to a more general analysis of sequential versus overlapping information processing stages by examining the question of whether response preparation can overlap with a process other than perceptual discrimination. In particular, we investigated the mental rotation process, to see whether response preparation begins before mental rotation finishes. Mental rotation was selected for several reasons: First, starting with the seminal studies of Shepard and his colleagues (Cooper & Shepard, 1973; Shepard & Metzler, 1971), mental rotation has been studied extensively for 20 years, and the results of these studies identify mental rotation as a relatively clearcut example of a distinct mental operation.⁸ One way to extend this work is to determine whether mental rotation operates sequentially or concurrently with other mental processes. Second, mental rotation is a slow process whose duration can be manipulated over a large range by varying stimulus orientation. This provides a long interval over which preliminary response preparation could in principle occur, thereby enhancing the power of experiments to detect such early preparation. Third, judging from the relatively long times required for mental rotation and our introspective judgements that it is completely under deliberate control (cf., Cooper & Shepard, 1973; Corballis, 1986), we believe that rotation imposes a heavy processing load. Although previous studies indicate that response preparation can overlap with simple perceptual discriminations, we suspected that such overlap might not be possible with a more effortful, controlled process such as mental rotation.

Like a number of recent studies examining the overlap of perceptual discrimination and response preparation (e.g., De Jong, et al., 1988; Miller & Hackley, 1992; Osman, et al., 1992; Smid, et al., 1991), the present study used lateralized readiness potentials (LRPs) to monitor response preparation. In brief, the LRP is an electroencephalographic (EEG) reflection of preparation to move a hand. It is measured by comparing average EEG activity, across trials, over the left and right sensorimotor cortical areas, which control movements of the right and left hands, respectively. Average EEG is more negative at the electrode contralateral to the responding hand than at the ipsilateral one (e.g., Kutas & Donchin, 1974), and the difference wave showing this contralateral negativity is known as the LRP. The LRP often begins several hundred milliseconds prior to a hand movement, lasts at least until the movement is actually initiated, and can be observed in the absence of any overt movement as long as the intention to move is clearly present. Much is known about the neural generators of the LRP, the experimental variables that influence it, and its behavioral correlates (see Coles, 1989, for a review), and this evidence strongly suggests that the LRP is a sensitive on-line measure of motor preparation suitable for examining preparation of a hand movement (cf., De Jong et al., 1988; Miller & Hackley, 1992; Osman et al., 1992; Smid et al., 1991).

Experiment 1 of Osman et al. (1992) provides a good illustration of how the LRP may be used to detect preliminary partial response preparation. In that experiment, subjects responded according to the location and identity of alphanumeric stimuli that appeared one at a time to the left or right of fixation. Subjects had to respond with the left hand when a digit appeared on the left, with the right

⁸There is some remaining dispute about the nature of the rotation process. For example, it may either bring the frame of reference into alignment with the representation of the presented character (e.g. Corballis, 1988; Koriat & Norman, 1984) or vice versa (e.g., Robertson, Palmer, & Gomez, 1987). In addition, it may be characterized as a single process or as a collection of related subprocesses. Such disputes are not critical to the present study, which focuses on the question of whether mental rotation – whatever it does, and whether it is unitary or not – must finish before response preparation can begin.

hand when a digit appeared on the right, and had to withhold the response (no-go) when a letter appeared in either location. As expected, LRPs were observed on go trials, when subjects responded with one hand or the other. More importantly, however, LRPs were also observed on no-go trials, when letters were presented, suggesting that subjects briefly prepared the hand that would have responded had the stimulus been a digit. Osman et al. noted that the location of a stimulus could be perceived before its alphanumeric category, so information about which hand might respond was available before information indicating whether the response was to be made or withheld. They concluded that the early information about location was used to prepare the hand which might respond – thereby generating a short-lived LRP – before information about alphanumeric category arrived to indicate that no response was required. In short, the fact that an LRP was found in the absence of a response suggests that some response preparation was carried out before perceptual analysis (of alphanumeric category) was complete, contrary to discrete models. Analogous results have been reported by Miller and Hackley (1992) and Smid et al. (1991).

The present study used similar analyses of LRP on no-go trials and – as will be seen later – an additional converging analysis of LRP on go trials, to see whether response preparation begins before mental rotation is complete.

Experiment 1

The subjects' task was an elaboration of the usual mental rotation task, with the identity of the stimulus character determining which hand should respond, and its form (normal vs. mirror-image) determining whether the response had to be made ("go trial") or withheld ("no-go trial"). For example, a subject might be instructed to respond with the left hand if the digit "2" appeared in its normal form, to respond with the right hand if the digit "7" appeared in its normal form, and to withhold responses to both digits if they appeared in their mirror-image forms.

In this task, our major question was whether information about the identity of the stimulus is used to prepare the appropriate response hand before mental rotation is complete. Previous studies show that the time needed to identify a disoriented letter is much less – and much less affected by orientation – than the time needed to make the mirror/normal discrimination, indicating that identity is recognized well before mental rotation finishes, especially for stimuli rotated far from upright (e.g., Cooper & Shepard, 1973; Corballis, 1988). Corballis, Zbrodoff, Shetzer, and Butler (1978), for example, conclude that "subjects first establish the identity of a character, then determine its angular orientation, and, finally, mentally rotate an internal representation to the upright in order to determine whether the character is normal or backward" (p. 105). If letter identity is recognized before rotation is complete, then information about stimulus identity could in principle be used for response preparation at the same time as mental rotation is being carried out. Although mental rotation must finish before the mirror/normal discrimination can be made, determining the go-no-go decision, considerable hand preparation might already have taken place by that time, especially for stimuli rotated many degrees from upright. Continuous models predict that such preliminary response preparation should take place, because "information about stimuli accumulates gradually in the visual system, and as it accumulates, responses are concurrently primed or partially activated" (Eriksen & Schultz, 1979, p. 252). The hybrid ADC model makes the same prediction, because stimulus identity would fully activate a distinct stimulus code (i.e., letter or digit name), and this code could be transmitted out of the perceptual system to enable response preparation prior to the end of mental rotation. Only fully discrete models predict that response preparation could not begin before mental rotation had finished. Such models require stages to operate in strict sequence, with no bypassing of stages, in which case the stimulus would have to be fully evaluated (i.e., identity and mirror/normal form) before response processes could begin.

If response preparation does begin before mental rotation is complete, two predictions can be made about LRPs. First, LRPs should be observed on no-go trials. This prediction is based on the same logic described above in connection with the study of Osman et al. (1992): If response preparation takes place before the go-no-go decision is made, then the LRP should reflect this preparation whether the go or no-go decision is ultimately reached. Second, the LRP should begin at approximately the same time regardless of stimulus orientation on go trials. Even though the effect of orientation on RT is several hundred milliseconds, LRP can begin as soon as identity is recognized, and the effect of orientation on identification time is small or nonexistent (e.g., Cooper & Shepard, 1973; Corballis et al., 1978). Thus, the LRP should begin at approximately the same time regardless of orientation.

If response preparation does not begin until after mental rotation is complete, on the other hand, quite different predictions about LRP can be made. First, no LRP should be observed on no-go trials, because responses would not be prepared if they were known not to be necessary. Second, LRP should begin later for upside-down stimuli than for right-side-up stimuli, with intermediate times of LRP onset for intermediate orientations. If response preparation does not begin until mental rotation is finished, then whatever delays the completion of mental rotation (i.e., orientation) should delay response preparation similarly. Thus, the interval from the onset of preparation to the response should be independent of orientation.

An additional factor included in this experiment was memory set size. In different blocks, there were either 2 or 6 possible stimulus characters, with half assigned to each response hand, so the memory set size for each hand was either $M = 1$ or $M = 3$. An example of the smaller memory set size has already been given (2 vs. 7), and an example of the larger memory set would be: "respond with the left hand to a normal G, P, or R, and respond with the right hand to a normal F, J, or Q". This factor was included to check for an influence of memory search difficulty on the use of preliminary information. We reasoned that increasing the memory load might make it more difficult to determine which hand was indicated by stimulus identity, and thereby reduce or delay the onset of any preliminary response preparation that might occur.

Method

Subjects.

Twenty-eight undergraduate students at the University of California, San Diego, were paid \$5 per hour for participating in a single session that lasted 2-3 hours. All subjects were right-handed, as assessed with the Edinburgh handedness inventory, and all reported normal or corrected-to-normal vision. Data were discarded for four subjects who did not show LRPs on go trials, because results from these subjects cannot help test different models' predictions concerning either the presence of LRP on no-go trials or the relative timing of LRPs on different kinds of go trials. Other studies in our lab have also found that go-trial LRPs are absent for approximately 10-20% of subjects, so this is not an unexpected number of discards. Of the remaining 24 subjects, 12 were male and the mean and standard deviation of age were 20.88 years and 2.53 years, respectively.

Apparatus.

Subjects were tested individually in a moderately lit, sound-attenuated room. They sat in a comfortable chair at a distance of approximately 90 cm from an NEC Multisync 2a monitor on which the stimuli were displayed. An IBM-PC compatible computer controlled the stimulus display and recorded both behavioral and psychophysiological responses. Each behavioral response was made by pressing three keys on the standard computer keyboard with the index finger, ring finger, and middle finger, in that order (the keys were c, z, and x for the left hand and comma, slash, and period for the right hand). Three-keypress responses were used in an attempt to maximize

experimental power, based on the finding that they produce larger LRPs than single keypress responses (Hackley & Miller, 1995).

Stimuli.

The stimuli were the alphanumeric characters 2, 7, F, G, J, P, Q, and R, chosen because they did not give rise to orientation or name confusions when presented at various orientations and in both normal and mirror-image versions. They were presented one at a time as white characters – approximately .5° in height – on a black background. Each character appeared in either its normal or its mirror-image form, rotated 0, 60, 120, or 180 degrees clockwise from the upright position. The two numeric characters were used in blocks with a memory set size of 1 per hand. One digit was assigned to each response hand, and the digit's form (i.e., normal vs. mirror-image) determined whether the response was to be made (go) or withheld (no-go), as described below. The six alphabetic characters were used in blocks with the memory set size of 3 per hand. Three letters were assigned to each response hand, and the letters' forms distinguished between go and no-go trials. For both memory set sizes, the assignment of characters to response hands was randomly determined for each subject and remained constant throughout the session.

Design.

Each subject was tested in 10 blocks of 96 trials, alternating blocks of set sizes 1 and 3. Within each block, half of the trials used stimulus characters assigned to the left hand and half used characters assigned to the right. Within these two halves, each combination of orientation (0, 60, 120, or 180 degrees) and response (go vs. no-go) also occurred equally often, with a different random order of trials for each subject and block. In blocks with the larger memory set size, one of the three possible characters assigned to the desired hand was selected randomly on each trial.

S-R mapping was counterbalanced across four equal groups of subjects. In one group, subjects made go responses to normal characters and no-go responses to mirror-image characters, and in a second group this S-R mapping was reversed. In a third group, subjects made the go response when characters assigned to the left hand appeared in normal form and when characters assigned to the right hand appeared as mirror-images, but made the no-go response in the other two cases. In a fourth group, subjects made the go response to mirror-images with the left hand and to normal images with the right hand. Although there is no prior evidence that the results would depend on the S-R mapping, it seemed reasonable to try all the possibilities in case this factor had an effect.

Procedure.

The stimuli and task were explained verbally to the subjects at the start of the experiment, and specific written instructions reminding the subject of the S-R mapping were presented before each block. Subjects were requested to respond as quickly as possible with the correct hand on go trials, but to be careful not to respond on no-go trials. Before the experiment started, subjects practiced their performance in the presence of the experimenter. In order to minimize artifacts in EEG recordings, subjects were cautioned to blink only between trials.

To familiarize the subject with the S-R mappings, each of the first two blocks began with 32 unrecorded practice trials. Subsequent blocks began with 4 unrecorded warm-up trials. Subjects initiated blocks at their own pace, and they were encouraged to take breaks of a few minutes duration between blocks.

At the beginning of each trial, a plus sign was presented in the middle of the screen for 200 ms to serve as a fixation point and warning signal. The test stimulus character appeared at the fixated location sometime between 300 and 600 ms following the offset of the plus sign, with this interval varying across trials according to a uniform probability distribution in order to minimize the influence of the plus sign on the stimulus-locked EEG to the test stimulus. To avoid a learning effect due to prolonged exposure of no-go stimuli, all test stimuli stayed on the screen for 2.5 sec,

regardless of RT. After the test stimulus disappeared, the word 'right' or 'wrong' appeared on the screen as feedback regarding response accuracy. The next trial began with the presentation of another plus sign approximately 2.5 sec after the offset of the test stimulus.

Electrophysiological Recording.

Electrophysiological measures were recorded with Ag/AgCl electrodes attached with Grass EC-2 paste, amplified with a Beckman model RM polygraph, and digitized at 250 Hz per channel. Electrode impedances were kept below 5K Ohm for facial and scalp electrodes, and below 15K Ohm for arm electrodes measuring EMG. On each trial, digitization began 200 ms preceding stimulus onset and continued for 2.7 sec.

EEG was recorded at Pz, C3', and C4', the latter two sites being located 1 cm anterior and superior to the C3 and C4 of the International 10-20 System, all of which were referenced to the left ear lobe (A1). Horizontal EOG was measured at sites approximately 2 cm from the left and right canthuses, and these were also referenced to A1. For the above scalp and facial electrodes, a time constant of 8 sec was used. Vertical EOG was measured with a bipolar recording, comparing sites just above and below the left eye, using a time-constant of .45 sec. EMG was measured on the left and right arms, with bipolar recordings comparing activity at ventral forearm electrodes placed approximately one- and two-thirds of the way from the wrist to the elbow. EMG was rectified off-line, so that all activity was positive, and the time constant was .015 sec. For EMG only, a hardware notch filter excluded activity at frequencies near 60 Hz.

Data Reduction and Analysis.

Individual trials were excluded if they were contaminated by electrophysiological artifacts, such as blinks, horizontal eye movements, scalp EMG, or amplifier saturation. Following previous studies (e.g., Miller & Hackley, 1992), we also excluded no-go trials if noticeable EMG activity occurred, so that any LRP activity would reflect preliminary response preparation rather than an erroneous decision to respond, whether derived from discrete or continuous processing, that was countermanded before a keypress actually occurred.⁹ For each subject, we only eliminated trials with artifacts occurring between the onset of recording (i.e., 200 ms prior to stimulus onset) and the moment corresponding to the 90th percentile of RT for that subject and condition. Trials with artifacts occurring later than this were not excluded, because we wanted to retain as many trials as possible and because such late-onset artifacts would not have any effects on the comparisons of interest. For most subjects, a total of approximately 25-30% of the trials were eliminated due to artifacts.

LRP waveforms were computed for each subject and orientation memory set size condition using the artifact-free trials. Within each condition, trials were first split into two groups, depending on whether the stimulus character belonged to the memory set assigned to the left or right hand. At

⁹Preliminary analyses of the distributions of individual-trial EMG activity, scored by finding the maximum mean EMG amplitude within any 100 msec window, tended to suggest that subjects did occasionally generate substantial EMG activity on no-go trials, consistent with the idea that they sometimes activated and then countermanded motor responses. Specifically, we examined the distributions of EMG activity obtained on (a) the responding arm on all go trials, (b) the arm associated with the stimulus name on all no-go trials, and (c) the arm not associated with the stimulus name on all no-go trials. For the most part, the distributions of EMG activity on no-go trials were well below the minimum EMGs observed on go trials, with a median EMG on no-go trials that was approximately 3% of the median EMG on go trials. The distributions of EMG activity on no-go trials were highly skewed, however, with the largest 2-3% of no-go EMGs being approximately the same size as the smallest 2-3% of EMGs observed on go trials. The two different no-go distributions, from associated and non-associated arms, were virtually identical to one another up to approximately their 95th percentiles, but diverged slightly at the very highest percentiles. This divergence may indicate that subjects were more likely to prepare, then countermand, the response hand associated with the stimulus name than the opposite hand. To check for an influence of a small proportion of such near-responses on no-go LRPs, we analyzed the no-go LRPs once including all no-go trials regardless of EMG and once with strict criteria for maximum EMG, excluding approximately 10% of no-go trials on which the most EMG was observed. Very similar results were obtained in the two sets of analyses; the reported figures come from the analyses with exclusion.

each time point (see next paragraph), the voltages from the electrode located over the left sensorimotor cortex, $C3'$, were averaged across trials within a group to obtain $C3'_L$ and $C3'_R$, where the subscript indicates the hand to which the stimulus character was assigned. Analogous computations yielded the average voltages from the electrode over the right sensorimotor cortex, $C4'_L$ and $C4'_R$. The LRP was then computed as the average difference between contralateral and ipsilateral voltages, which may be expressed as

$$\text{LRP} = (C3'_R - C4'_R + C4'_L - C3'_L) / 2$$

The LRP waveform was obtained by performing the above calculations separately for each 4-ms time point for which voltages were digitized. To obtain stimulus-locked LRPs, time points were defined relative to stimulus onset; specifically, a value of LRP was computed for each 4-ms time point in the interval starting 200 ms before stimulus onset and ending 2.5 sec after stimulus onset. To obtain response-locked LRPs, time points were defined relative to the instant of the first keypress response; specifically, these were computed over the interval starting 2 sec prior to the response and ending at the response. Statistical analyses were performed on the LRP waveforms as just described, but both types of LRPs were digitally smoothed before they were plotted in the figures, using a low-pass filter with a half-power cut-off of 3 Hz.

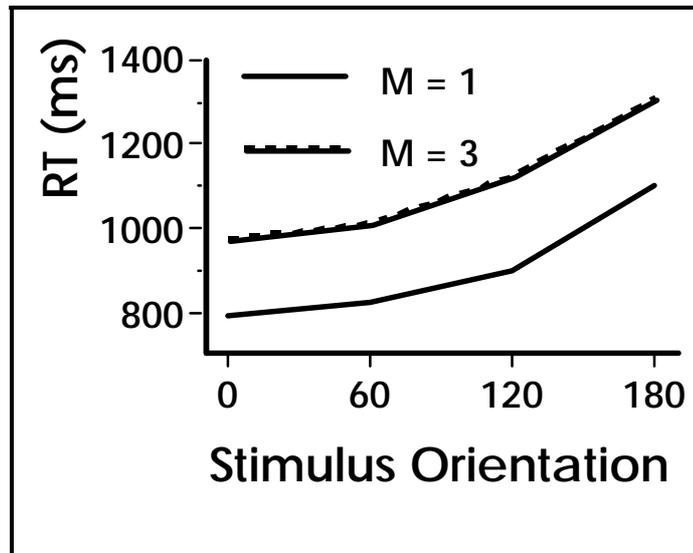


Figure 1: Experiment 1: Mean correct reaction time as a function of memory set size ($M = 1$ or $M = 3$) and stimulus orientation (0° , 60° , 120° , or 180°).

Table 1: Percentage of Correct Responses as a Function of Memory Set Size, Orientation, and Response.

Memory Set Size:	Resp:	Orientation				Mean
		0°	60°	120°	180°	
1	Go	98.3	98.2	98.1	94.9	97.4
1	No-go	98.7	98.6	97.5	91.5	96.6
3	Go	96.6	97.3	95.0	91.6	95.1
3	No-go	98.6	97.4	96.7	91.2	96.0
	Mean	98.1	97.9	96.8	92.3	96.3

Results and Discussion

Performance data.

Mean correct RTs and percentages of correct response (PCs) were calculated on the basis of the first of the three keypresses, and averages of these measures across subjects are shown in Figure 1 and Table 1. RTs were evaluated with an analysis of variance including the two within-subjects factors of memory set size (1 vs. 3) and angle from upright (0°, 60°, 120°, or 180°), and the between-subjects factor of S-R mapping (four levels: normal assigned to go response for both hands; mirror assigned to go response for both hands; normal assigned to go response for right hand and mirror for left; normal assigned to go response for left hand and mirror for right).

There was a 199 ms effect of memory set size on RT, $F(1; 20) = 92.78$; $p < .001$, replicating the usual finding that subjects respond faster when comparing the stimulus against smaller rather than larger memory sets (e.g., Sternberg, 1969). This effect is somewhat larger than is usually obtained, possibly because stimulus materials (i.e., letters vs. digits) were confounded with memory set size, or because subjects had to remember one memory set for each hand rather than simply one positive set (cf., Sternberg, 1969).

As is typical in mental rotation studies, orientation had a strong effect on RT, with RTs to upside-down stimuli averaging 318 ms more than those to right-side-up stimuli, $F(3; 60) = 248.93$; $p < .001$. The linear trends accounted for 86% and 92% of the variance, respectively, in the conditions with memory set sizes of one and three.

Interestingly, the effects of set size and orientation on RT were approximately additive, $F(3; 60) = 209$; $p > .1$. Such additivity is predicted by models with sequential stages (e.g., Sternberg, 1969), in which subjects cannot begin memory search until mental rotation is finished (or vice versa). In contrast, an underadditive interaction is predicted by many models in which mental rotation occurs at the same time as memory search (cf., Egeth & Dagenbach, 1991; Miller, 1993; Schweickert & Townsend, 1989; Stanovich & Pachella, 1977; Townsend & Ashby, 1983; but see McClelland, 1979, for a parallel model that can predict additivity). Intuitively, if memory search is carried out in parallel with a very time-consuming mental rotation process (i.e., with upside-down stimuli), then the duration of memory search should have little or no effect on total RT.¹⁰

Finally, the between-subjects effect of S-R mapping was not significant, $F(3; 20) = 1.38$; $p > .2$. There was a significant interaction between orientation and S-R mapping, $F(9; 60) = 3.14$; $p < .004$, but we suspect this interaction is a Type I error because it was not systematic.

Similar analyses of PC confirmed the effects found in RTs. Accuracy decreased as memory set size increased, $F(1; 20) = 9.92$; $p < .005$, and as orientation increased, $F(3; 60) = 30.39$; $p < .001$, whereas S-R mapping had no effect on PC. This analysis included the additional factor of go vs. no-go response, which had a small and nonsignificant effect on overall PC, $F(1; 20) < 0.01$; $p > .9$. There was an interaction of orientation and go-no-go, $F(3; 60) = 5.73$; $p < .002$, caused by an increase in the number of responses on no-go trials at 180°. Set size and go-no-go also interacted, $F(1; 20) = 6.87$; $p < .02$: The number of errors on no-go trials did not depend on set size, but there were 2.3% more errors on go trials for set size 3 than for set size 1. The former, no-go errors were virtually all commission errors (responses with the correct hand which should not have been emitted), whereas the latter, go errors were mostly responses with the incorrect hand. There was also an interaction of orientation, set size, and S-R mapping, $F(9; 60) = 2.41$; $p < .02$, which showed no clear pattern.

Stimulus-locked LRPs.

Figure 2 shows average stimulus-locked LRPs for both go and no-go trials, as a function of orientation and memory set size. As usual, strong movement-preceding lateralization was found on

¹⁰Wijers et al. (1989) found an interaction between set size and orientation for normal stimuli, which contrasts with the present findings. However, the interaction was overadditive, so it does not support models allowing overlap of rotation and memory search.

Figure 2: Experiment 1: Grand average ($N = 24$) stimulus-locked lateralized readiness potentials as a function of memory set size ($M = 1$ or $M = 3$), stimulus orientation (0° , 60° , 120° , or 180°), go vs. no-go response (solid vs. broken lines), and time (in milliseconds) since stimulus onset.

LRPs were clearly much weaker on no-go trials, suggesting that little if any response preparation occurred until mental rotation was complete. In parallel tests of mean no-go LRPs, there were significant no-go LRPs ($p < .05$) in only six of the 56 100-ms intervals defined by the factorial combination of four orientations, two memory set sizes, and seven time periods (starting from 300-900 ms after stimulus onset). Five of these were in the condition with memory set size of one and an orientation of 60° , and the sixth was in the condition with memory set size three and orientation 120° . Unfortunately, it is difficult to determine the exact probability of this outcome under the null hypothesis of no no-go LRP, because observations in different time intervals within the same memory set size and orientation condition are not independent.

Testing no-go LRP separately for each condition is clearly a conservative procedure, because it is not sensitive to the consistent trend toward no-go LRPs apparent across conditions. Because there are clear hints of no-go LRPs in Figure 2, especially for $M = 1$, we also tested for the presence of LRP on no-go trials by including all eight conditions in factorial analyses with two memory set sizes and four orientations. In these analyses, overall mean LRP was statistically reliable ($p < .05$) in the 100-ms intervals starting at 400, 500, 800, 900, and 1000 ms. Moreover, LRP was significantly larger for memory set size one than three ($p < .01$) for the intervals starting at 300 and 400 ms. Additional analyses considering the two memory set sizes separately revealed significant LRPs for memory set size one in the 100-ms intervals starting at 300 and 400 ms ($p < .01$), and at 800, 900, and 1000 ms ($p < .05$), but no significant LRPs for set size three. Thus, the no-go LRP data indicate that some response preparation does occur on no-go trials, at least with memory set size one. In none of these factorial analyses did LRP vary across orientations (p 's $> .20$).

Response-locked LRPs.

As described in the introduction, another prediction of the hypothesis that response preparation does not begin until mental rotation is finished is that the onset of LRP should depend on orientation approximately as much as RT does. Judging from mean RTs, for example, the LRP would be expected to start approximately 300 ms later for upside-down than right-side-up stimuli. If response preparation can begin before mental rotation is finished, however, the LRP should start at approximately the same time regardless of orientation. These predictions are most easily tested with go trials, of course, since these produced the clearest LRPs.

Because LRP has a gradual onset and noise is present, it is difficult to judge from Figure 2 whether LRP onset depends on orientation or not. A deeper problem with testing this prediction from the data depicted in this figure, however, is that onset latencies of stimulus-locked LRPs are not commensurable with mean RTs. Mean RTs, by definition, are equally influenced by all trials within a condition. The onset of the stimulus-locked average LRP, however, is determined by the fastest trials within the condition (cf., Meyer, Osman, Irwin, & Yantis, 1988). For example, if an LRP starts 400 ms after stimulus onset on the faster half of the trials, then an identifiable LRP onset will occur by 400 ms in the overall average, regardless of how much later the LRP is produced on the slower half of the trials. This is clearly a problem: if mean RT and stimulus-locked LRP onset are incommensurable, then orientation need not have comparable effects on the two measures. In fact, if the fastest responses are approximately independent of orientation – e.g., because orientation has most of its effect on relatively slow responses, because there are special fast responses on trials that are exact repetitions, or because subjects make occasional fast guesses – then stimulus-locked LRP onset might not vary with orientation at all.

A comparison of minimum RTs indicates that the incommensurability problem is quite real. The minimum RTs in each of the four orientations were 409, 410, 411, and 460 ms for memory set size 1 and 439, 430, 470, and 576 ms for memory size 3. As compared with mean RTs, these minima reveal very little effect of orientation, and therefore suggest that it is impossible to compare the effect of orientation on stimulus-locked LRP onset with that on mean RT.

Fortunately, these problems can be avoided by using response-locked LRPs to examine the effect of orientation on LRP onset. These waveforms are computed in the same way as the stimulus-locked LRPs, except that individual trials are temporally aligned at the moment of the response (i.e., the first keypress) rather than at the moment of stimulus onset. For example, a response-locked LRP measure at time -200 ms indicates the size of the LRP, on average across trials, at the moment 200 ms before the first keypress. Response-locked LRPs are generally regarded as better measures of response preparation than stimulus-locked LRPs (cf., Osman & Moore, 1993), because they are less affected by variance in the duration of sensory and decision processes that precede LRP onset. Their main disadvantage, of course, is that they are not defined for no-go trials.

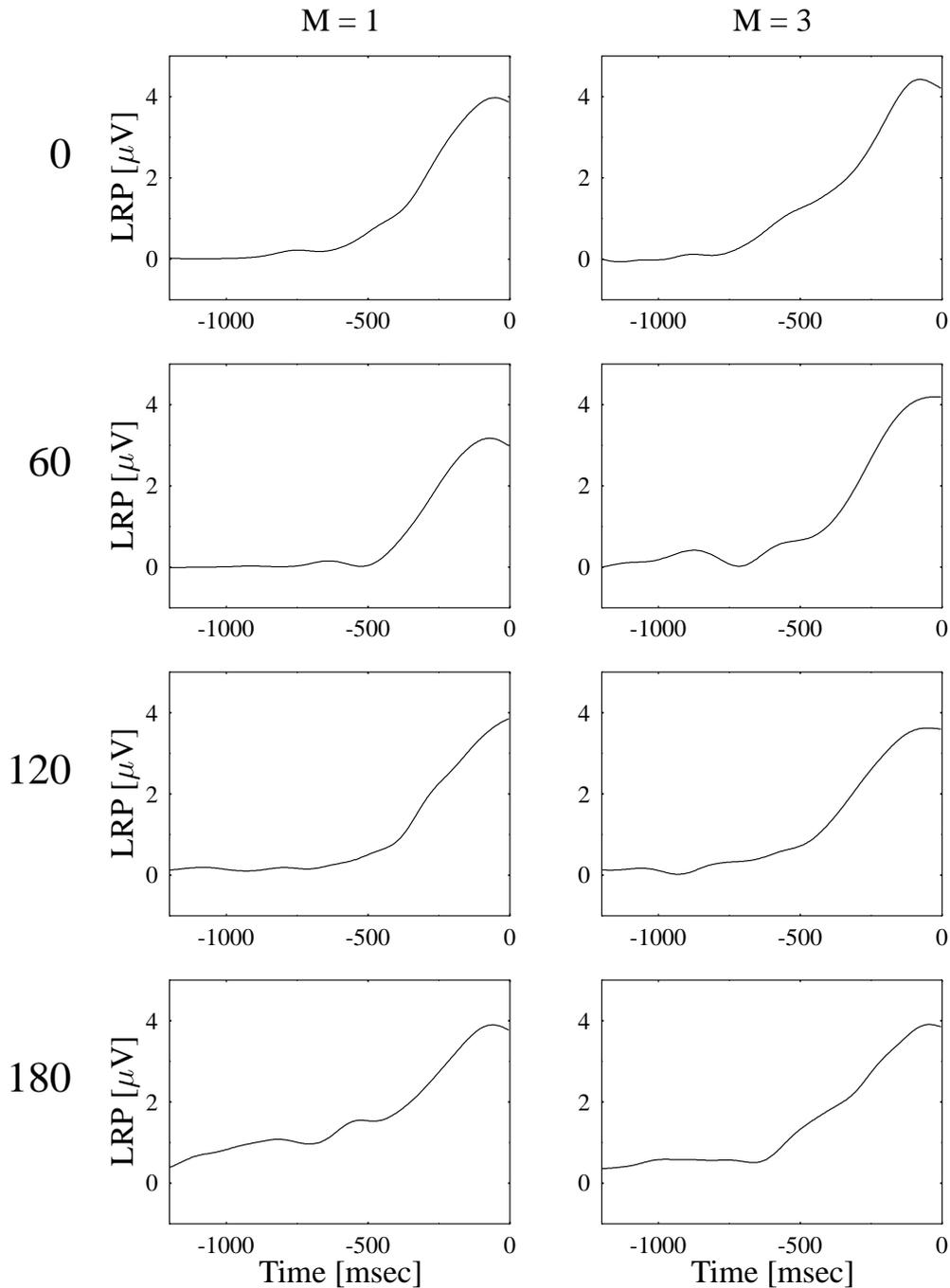


Figure 3: Experiment 1: Grand average ($N = 24$) response-locked lateralized readiness potentials on go trials as a function of stimulus orientation (0° , 60° , 120° , or 180°), memory set size ($M = 1$ or $M = 3$), and time (in milliseconds) prior to the first keypress response.

Discrete and continuous models make distinct predictions concerning response-locked LRPs that are similar to those made concerning stimulus-locked LRPs. According to discrete models, in which response preparation cannot begin until mental rotation is complete, all of the information needed to activate and execute the response is available at the moment of LRP onset. This implies that the interval from LRP onset to the keypress should be independent of the orientation of the stimulus, because the time needed to activate and initiate the response should not depend on how much time was previously needed to decide which response to make. If response preparation begins before mental rotation is complete, on the other hand, then LRPs should start building up before

rotation is finished. Since orientation influences the duration of rotation, such models predict that LRP should precede the response by longer times for greater orientations.

In short, to see whether orientation has all of its effect prior to the onset of response preparation or has some of its effect after the onset of response preparation, we can look to see whether response-locked LRPs begin further in advance of the response with upside-down than with right-side-up stimuli. Such a comparison has the obvious advantage that it compares commensurable measures (i.e., LRPs) rather than incommensurable ones (i.e., LRP onset with mean RT). A more subtle advantage is that this comparison can in principle reveal a difference if response preparation ever begins before rotation is finished. Even if early response preparation only took place on 10% of all trials, for example, average LRPs would be slightly different for different orientations, and an experiment with sufficient statistical power could reveal that difference.

Figure 3 shows response-locked LRPs for each combination of orientation and memory set size.¹¹ For memory set-size three, it appears that LRPs begin at approximately the same time, relative to the response, regardless of orientation. For memory set-size one, on the other hand, the timecourse of LRP preceding the response does seem to vary with orientation. Specifically, the LRP seems to start further ahead of the response when the stimulus is upside-down than when it is at one of the other orientations.

To test statistically for differences in response-locked LRPs, mean amplitude was computed for each 100-ms interval preceding the response, for each subject and condition. These mean amplitudes were first examined in overall analyses of variance, conducted separately for each 100-ms interval, with factors of group, memory set size, and orientation. These analyses provided somewhat equivocal evidence that response-locked LRPs depend on orientation, with marginally significant effects of orientation ($p < .10$ or better) in the five intervals starting from 800-1200 ms prior to the response. In all intervals, the means indicated that 180° stimuli yielded the most lateralization. No significant effects of memory set size were found, and the interaction of orientation and set size was only significant ($p < .02$) in the interval from 500-600 ms prior to the response. As can be seen in the figure, this interaction reflects the fact that orientation has a greater effect for set size one than for set size three.

Because the overall analyses were somewhat equivocal about whether response-locked LRP starts earlier with stimuli at larger orientations and because there are good a priori reasons to expect larger effects with set size one than with set size three, we conducted further analyses looking separately at the data from the two memory set size conditions. In the set size one conditions, there were significant effects of orientation on mean LRP amplitude for each of the 100-ms intervals starting at 1100-1200 ms prior to the response and ending at 400-500 ms prior (every $p < .05$). Additional ANOVAs excluding the upside-down stimuli revealed no significant differences, so the orientation effect may be completely attributable to the upside-down stimuli. In the set size three conditions, orientation had a significant effect in only one interval (1600-1700 ms prior to the response, $p < .05$), which we took to be a Type I error because it was quite nonsystematic across orientations (in fact, the mean LRP in this interval was smallest for upside-down stimuli).

¹¹Because EEG measurement began 200 ms prior to stimulus onset, the LRP could not be computed 1600 ms prior to the keypress response (i.e., at or near the left edge of Figure 3) on trials with $RT < 1600 - 200 = 1400$. For these trials, zero was used as the LRP value for all time points prior to those at which EEG measurement began. This procedure seems appropriate, because the LRP is defined relative to the correct response, which is unknown before stimulus onset.

Summary of LRP Results.

Together, the stimulus- and response- locked LRPs suggest that response preparation can begin before mental rotation is complete. The effects supporting this conclusion are small, however, and are mainly present in blocks with memory sets of size one, not in blocks with memory sets of size three. In the former blocks, there are stimulus-locked LRPs on no-go trials, albeit weak ones, and response-locked LRPs begin earlier with upside-down stimuli than with stimuli in other orientations. The small amount of LRP on no-go trials could either indicate that a small amount of preparation took place on many trials or that a larger amount of preparation took place on only a few trials. Similarly, the effect of orientation on the onset of response- locked LRP could be the result of early onsets of substantial preparation on a subset of trials, or of an attenuated preparation on a larger proportion of trials. Although LRPs cannot differentiate between these two possibilities, either possibility is consistent with the conclusion that only a small amount of response preparation took place during mental rotation. In the set size three blocks, neither a no-go LRP nor an effect of orientation on the onset of response-locked LRP was present. Thus, it appears that response preparation can sometimes begin before mental rotation is complete, as long as it is sufficiently easy to determine which response should be activated given stimulus identity.

Experiment 2

Although the results of Experiment 1 indicate that some response preparation occurs before mental rotation is complete, they do not indicate whether response preparation occurs at its normal rate during mental rotation or instead occurs at a reduced rate due to some sort of interference of mental rotation with response preparation (cf., Ilan & Miller, 1994; Ruthruff, Miller, & Lachmann, 95). In fact, two aspects of the results suggest that there may have been some interference. First, the no-go LRPs were quite small in comparison with those found in previous studies (e.g., Miller & Hackley, 1992; Osman, et al., 1992), as if response preparation were taking place more slowly than usual. Second, no-go LRP did not depend on stimulus orientation. If response preparation were carried out in parallel with mental rotation and there were no interference, no-go LRPs should have been much larger with upside-down than right-side-up characters, because the former provide more time to prepare responses during rotation.

To test for interference between mental rotation and response preparation, the second experiment was a "control" experiment designed to measure response preparation in a task that did not require mental rotation. In this experiment, the crucial change was that the go-no-go decision was determined by the color of the stimulus rather than its mirror/normal form. Once again, the name of the stimulus character should be available fairly rapidly, regardless of the character's orientation, and this information could be used to begin preparing the response hand to which that character was assigned. Rather than having to mentally rotate the character to carry out the mirror/normal discrimination, however, in this task the subject had to make a color discrimination, since the go-no-go decision was determined by color rather than mirror/normal form. If color discrimination interferes less with response preparation than does mental rotation, then LRPs should indicate that more preliminary response preparation takes place with this task.

A crucial technical problem in this experiment was to make sure that the color discrimination took approximately as long as the mental rotation plus mirror/normal discrimination required in Experiment 1. In both experiments, the LRP signs of preliminary response preparation should be generated after the character has been identified but before the go-no-go decision has been made, so it is essential to equate these two intervals across experiments in order to have a fair comparison. If the color discrimination of Experiment 2 took much longer than the mirror/normal discrimination of Experiment 1, for example, we would naturally expect larger LRP signs of preliminary response preparation – not because of the change from mental rotation to color discrimination, but merely

because of the increased time available to generate such effects. On the other hand, if color discrimination took much less time than the mirror/normal discrimination, then Experiment 2 would seriously underestimate the amount of LRP to be expected without interference from mental rotation.

To this end, we asked pilot subjects to perform the task of Experiment 1 in half the blocks and the task of Experiment 2 in the other half. As might be expected, responses were much faster when the go-no-go decision was determined by color than when it was determined by mirror/normal form, especially for non-upright characters. Then, we modified the procedure for the color discrimination blocks so that stimulus characters were initially presented in a neutral color, and they changed to either the go color or the no-go color after a delay. After some trial and error, we determined that upright characters should change color 300 ms after their onsets in order to equate (approximately) the RTs to upright characters in the blocks corresponding to the two types of experiments. To simulate the additional delay introduced by mental rotation, we further delayed the color change by 34, 117, and 317 ms for characters present at 60°, 120°, and 180°, respectively.¹² These additional delays match the increments in RT, relative to upright stimuli, produced by each of these orientations in Experiment 1, with matching as close as possible given the timing limitations of a 60-cycle video display.

In summary, this experiment was designed to be as similar as possible to Experiment 1 except that the go-no-go decision was determined by stimulus color rather than mirror/normal form. The onset of color information was delayed to mimic the time consumed by mental rotation and mirror/normal discrimination in Experiment 1. If mental rotation actually interferes with response preparation, the present experiment should yield larger effects of response preparation on the LRP than were observed in Experiment 1, because in this case the LRP can occur while subjects are waiting for color information to be presented and then making the color discrimination, neither of which should cause much interference with response preparation (Hackley & Miller, 1995). If mental rotation does not interfere with response preparation, however, this experiment should yield approximately the same effects of response preparation as observed in the first experiment.

Method

This experiment was conducted using the same equipment, block structure, procedure, and types and numbers of trials as the previous experiment, except as noted otherwise. Subjects were 12 right-handed volunteers, none of whom had served in Experiment 1.

Each stimulus was initially presented in a reddish-purple color, generated by setting the red, green, and blue palettes of an IBM-compatible VGA card to values of 46, 28, and 28, respectively. The stimulus was either normal or mirror-image, which was determined randomly, independently of color and orientation. After the appropriate delay, the stimulus color changed to red (palette settings of 50, 0, and 0) or purple (palette settings of 38, 32, and 42). The delay between character onset and the color change was 300, 333, 417, or 617 ms for stimuli at orientations of 0°, 60°, 120°, and 180°, respectively. The order of $M = 1$ and $M = 3$ blocks and the assignment of letters and digits to response hands were varied as in Experiment 1. The assignment of red vs. purple to go vs. no-go was counterbalanced across four groups following the same pattern used to assign mirror vs. normal to go vs. no-go in the earlier experiment.

For comparability with Experiment 1, RT was measured from the onset of the character rather than the color information, even though it was not logically possible to respond correctly until color information was provided.

¹²Throughout the description of Experiment 2, the term orientation, rather than delay is used because it facilitates the comparison with Experiment 1. The delay was actually more relevant as a manipulation of response times, but the delays were consistently associated with one of the four orientations.

Results and Discussion

Performance data.

An initial analysis of correct mean RT and PCs was conducted to check for practice effects. Across the five $M = 1$ blocks, there was virtually no practice effect, so all of these blocks were included in the analyses. There was a large improvement from the first to the second $M = 3$ block, however, so the first of these blocks was excluded from all further analyses.

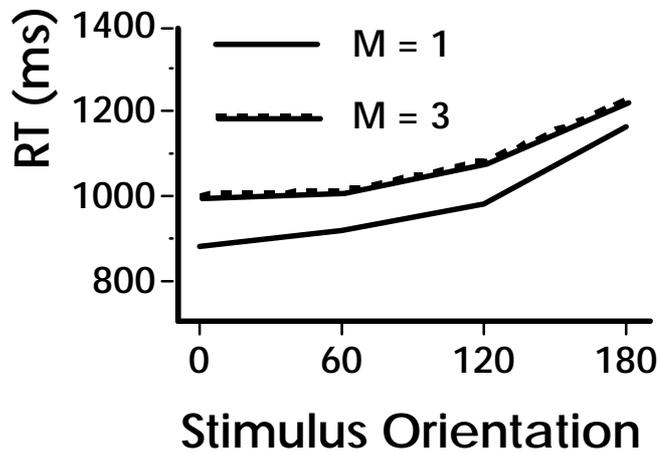


Figure 4: Experiment 2: Mean correct reaction time as a function of memory set size ($M = 1$ or $M = 3$) and stimulus orientation (0° , 60° , 120° , or 180°).

For each subject, PC and mean correct RT were computed for each combination of memory set size, response hand, and orientation, and these summary measures were submitted to a four-factor ANOVA with the additional factor of group. As shown in Figure 4, RT was significantly affected by orientation, $F(3; 24) = 170$; $p < .001$, and memory set size, $F(1; 8) = 34$; $p < .001$. The interaction of these two factors was the only other source of variance to approach significance, $F(3; 24) = 2.38$; $p < .12$. In the analysis of PC, the only significant sources of variance were the main effects of hand, $F(1; 8) = 9.27$; $p < .02$, with slightly higher accuracy on trials requiring right- than left- hand responses (97.1% vs. 95.9%), and group, $F(3; 8) = 8.82$; $p < .01$, with lower accuracy in the group for which red was the go color for both hands (93.7%) than in any of the other three groups (96.1%, 98.5%, and 97.8%).

Stimulus-locked LRPs.

Figure 5 shows average stimulus-locked LRPs as a function of memory set size, stimulus orientation, and go vs. no-go response. As in Experiment 1, mean LRP amplitudes were scored for go and for no-go trials, separately for each subject and for each 100-ms interval following stimulus onset.

Initial statistical analyses tested the significance, across subjects, of the LRP in each 100-ms interval, separately for each of the 16 waveforms displayed in Figure 5. For go trials, reliable LRPs ($p < .01$) were observed in most of the 100-ms intervals for all eight conditions. LRPs were also clearly present on no-go trials, indicating that response preparation began before the go-no-go decision had been made. The effect was most clearcut in the condition with $M = 1$ and upright stimuli, which yielded significant LRPs ($p < .01$) at 12 of the 100-ms intervals beginning between 300 and 1600 ms after stimulus onset. Significant no-go LRPs were also obtained in at least two of the 100-ms intervals in this range for five of the other seven combinations of memory set size and

orientation, the exceptions being the condition with $M = 1$ and an orientation of 120° , and the condition with $M = 3$ and upright stimuli. In total across these seven combinations, 13 of the 98 tests for LRP were significant at $p < .01$, a result that would be expected to occur by chance quite rarely, even allowing for some dependence of different time intervals within a given condition. In further analyses combining data from all eight conditions, the overall mean LRP was significant ($p < .01$) for most windows from 400- 1600 ms. Other sources of variance were generally not significant in these analyses, except that the interaction of orientation and memory set size was significant ($p < .05$) in the windows from 500-900 ms. In these windows, upright stimuli produced larger no-go LRP, relative to the other orientations, in blocks with the smaller memory set size.

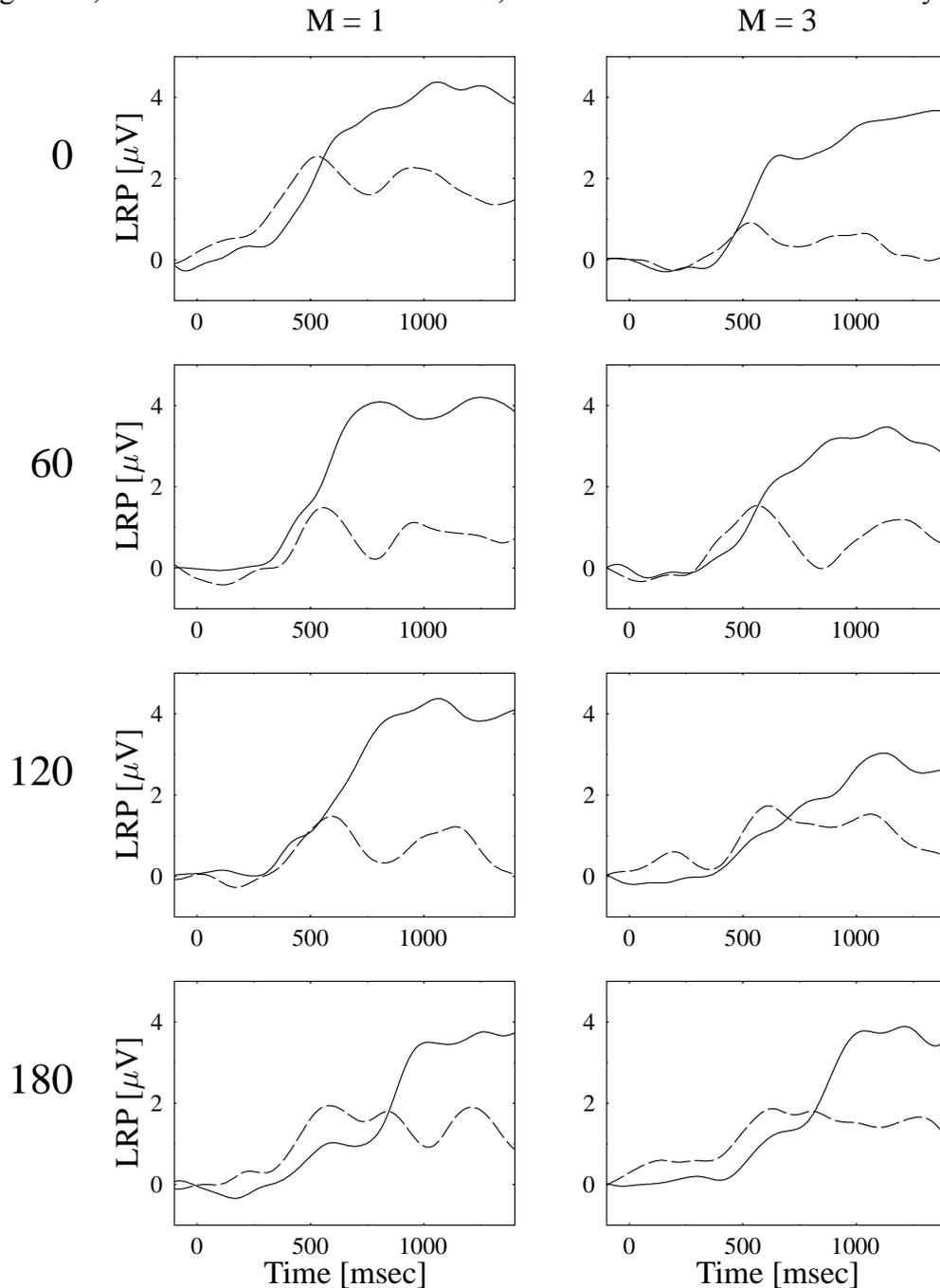


Figure 5: Experiment 2: Grand average ($N = 12$) stimulus-locked lateralized readiness potentials as a function of memory set size ($M = 1$ or $M = 3$), stimulus orientation (0° , 60° , 120° , or 180°), go vs. no-go response (solid vs. broken lines), and time (in milliseconds) since stimulus onset.

Comparable analyses of EOG indicate that neither the go nor the no-go LRP were artifacts of eye movements. Lateralization was consistently smaller at the ocular electrodes than at the central ones; if central lateralization were an artifact of eye movements, lateralization should have been four to five times larger at the ocular electrodes (Hillyard & Galambos, 1970). Moreover, lateralization at the ocular electrodes was statistically reliable in only a small fraction of the 100- ms windows yielding statistically reliable lateralization at the central electrodes (0% of the windows for go trials, and 8% for no-go trials).

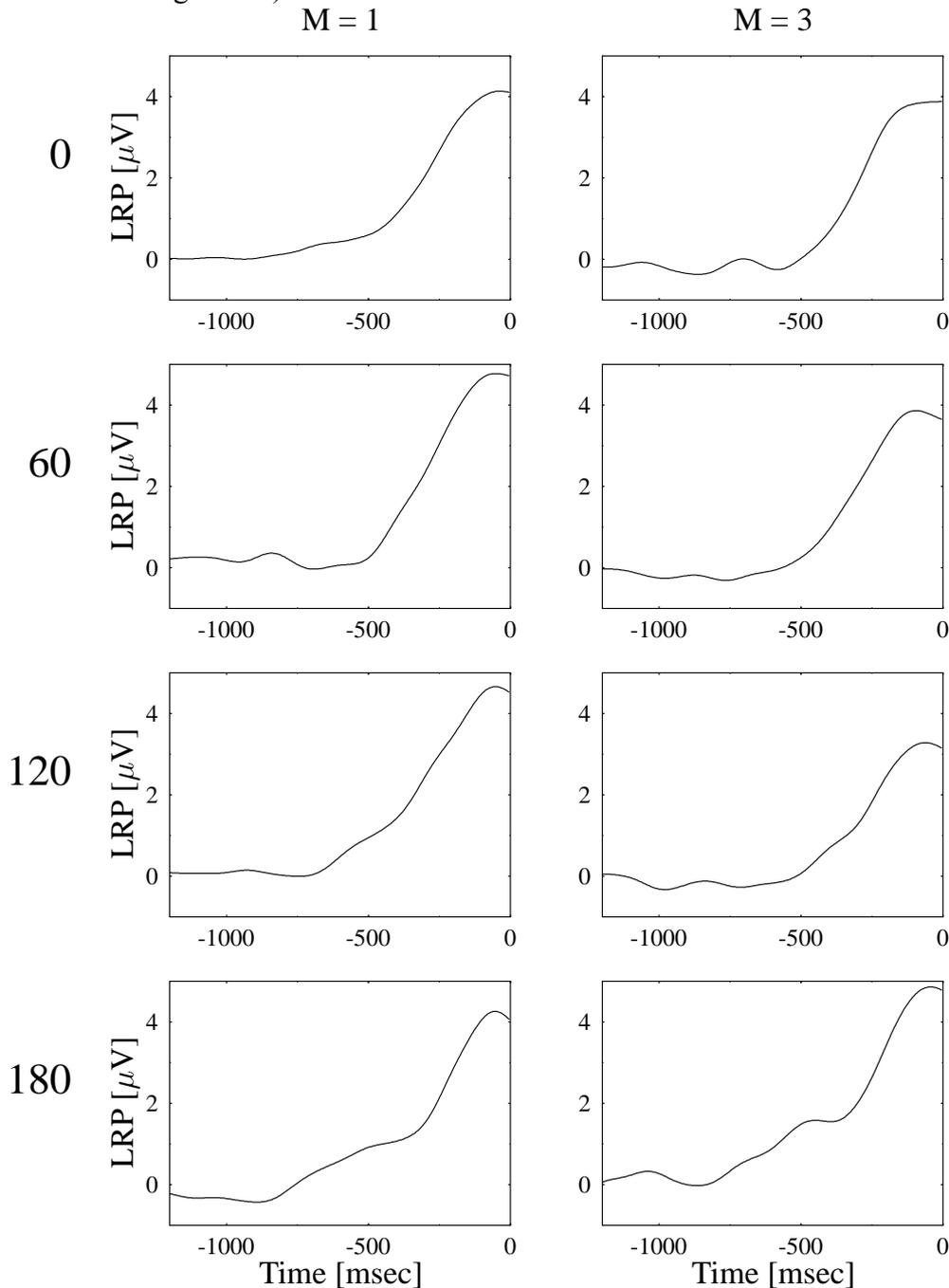


Figure 6: Experiment 2: Grand average ($N = 12$) response-locked lateralized readiness potentials on go trials as a function of stimulus orientation (0° , 60° , 120° , or 180°), memory set size ($M = 1$ or $M = 3$), and time (in milliseconds) prior to the first keypress response.

It is clear that the early portions of the LRPs on both go and no-go trials were generated prior to the go-no-go decision. Stimulus-locked LRPs began approximately 400 ms after stimulus onset, regardless of memory set size and orientation (cf., Figure 5), but the color information on which the go-no-go decision was based was not even presented until 617 ms after stimulus onset with upside-down stimuli. This is consistent with previous findings that LRP can develop in response to hand-informative precues presented slightly before a go- no-go decision is to be made (Hackley & Miller, 1995).

Response-locked LRPs.

Figure 6 shows average response-locked LRPs as a function of memory set size and stimulus orientation. The significance of the LRP was tested separately for each 100-ms interval of each condition, and significant LRPs were obtained for the last 3-4 windows preceding the response in all conditions except the one with $M = 3$, 120° , for which LRP was only significant at the $p < .02$ level in the last two windows.

Consistent with the hypothesis that LRP onset occurs further ahead of the response at greater orientations, there were significant LRPs from 400-600 ms prior to the response with stimuli at 120° ($p < .07$) and 180° ($p < .03$) but not with stimuli at 0° or 60° ($p > .20$). In addition, the mean size of the response-locked LRP measured during the 400-600 ms time window showed a significantly increasing linear trend with orientation ($p < .03$). The only apparent exception to this trend in Figure 6 is the lateralization more than 500 ms prior to the response in the memory set size one condition with stimuli at 0° , but this lateralization does not approach statistical reliability.

Comparisons Between Experiments.

In order to conclude that mental rotation interferes with response preparation, it is necessary to show that the signs of such preparation are present to a significantly greater extent in Experiment 2 than in Experiment 1. In order to do this, a number of ANOVAs were carried out to compare the results of Experiments 1 and 2, each with a factorial structure of 2 experiments 2 memory set sizes 4 stimulus orientations. Several statistically reliable differences between experiments were uncovered.

With respect to RT, Experiment 1 produced reliably larger effects than Experiment 2 of both memory set size, $F(1; 34) = 10.3$; $p < .01$, and orientation, $F(3; 102) = 4.326$; $p < .01$. These experiment-by-factor interactions are quite consistent with the view that subjects can more readily carry out memory search and response preparation while waiting for color information (Experiment 2) than while mentally rotating (Experiment 1), although other explanations of both interactions are also possible. There were no significant differences between experiments in PC.

Figure 7 shows cross-experiment comparisons of the psychophysiological measures of response preparation, averaged across memory set size and orientation. To test for statistically reliable differences between experiments, ANOVAs were used to compare mean LRP amplitudes in 100-ms windows. These ANOVAs had factors of experiment, memory set size, and orientation, and were conducted separately for LRPs measured on stimulus-locked go trials, stimulus-locked no-go trials, and response-locked go trials. The factor of experiment did not significantly interact with memory set size or orientation in any of the ANOVAs, but there were several significant main effects of experiment as discussed below.

As shown in the top panel of Figure 7, stimulus-locked go LRPs were fairly similar in the two experiments. There was some tendency for LRPs to begin slightly earlier in Experiment 1 than in Experiment 2, resulting in significantly larger LRPs in the first experiment for the time windows of 400-500 and 500-600 ms ($p < .01$). This effect probably reflects earlier onset of response execution rather than differential response preparation, however, because the cumulative probability of RT

likewise starts rising slightly earlier in Experiment 1 than in Experiment 2. Go-LRP did not differ reliably in the later time windows ($p > .05$).

As shown in the middle panel, stimulus-locked no-go LRP was clearly larger in Experiment 2 than in Experiment 1, producing a significant main effect of experiment in the six 100-ms windows starting at 400, 500, 600, 900, 1100, and 1200 ms ($p < .01$). In addition, this effect was significant ($p < .05$) in all but two of the remaining windows from 200-1600 ms. The presence of larger no-go LRPs in Experiment 2 than in Experiment 1 is strong support for the conclusion that mental rotation interferes with response preparation on no-go trials.

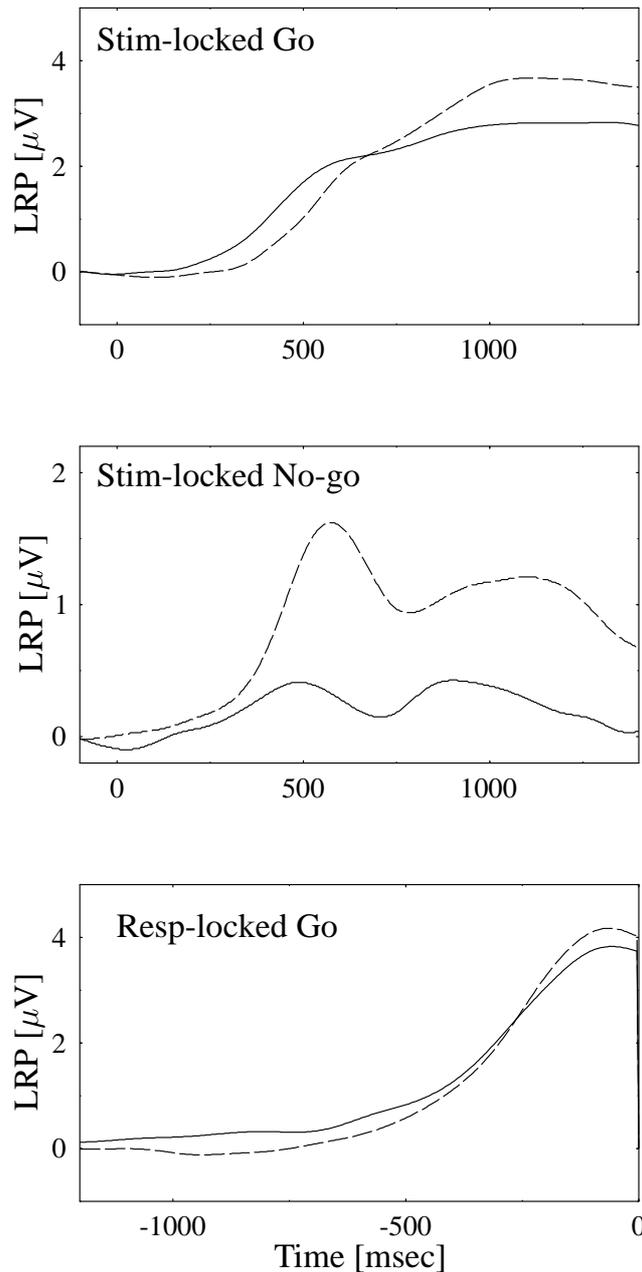


Figure 7: Comparisons of grand average LRPs from Experiments 1 (solid lines) and 2 (broken lines), averaging across memory set sizes and stimulus orientations as well as subjects. Top panel: Stimulus-locked go trial LRPs. Middle panel: Stimulus-locked no-go trial LRPs. Bottom panel: Response-locked go trial LRPs.

As shown in the bottom panel, the latter parts of the response-locked LRPs on go trials were quite similar in the two experiments. These LRPs did not differ significantly in the last seven 100-ms windows leading up to the response ($p > .10$). LRPs did appear to precede the response more in Experiment 1 than in Experiment 2, however. There was a small but statistically reliable difference between experiments in the window preceding the response by 900-1000 ms, with marginally significant differences ($p < .10$) in nearby windows (700-900 and 1000-1100); in these windows, response-locked LRPs were larger in Experiment 1 than in Experiment 2. The finding of slightly longer response-locked LRPs in Experiment 1 than in Experiment 2 is quite consistent with the conclusion that mental rotation interferes with preliminary response preparation. Subjects should have recognized the name of the stimulus character and begun preliminary activation of the associated hand at approximately the same time in both experiments, because the same characters were presented at the same orientations. Similarly, subjects should have recognized the go signal and begun final response activation and execution at approximately the same times (which depended on orientation) in both experiments, because of our attempts to match the RTs in the two tasks. The main difference between the two experiments, we believe, is that preliminary response preparation was less efficient in Experiment 1 than in Experiment 2, because of interference from mental rotation. As a consequence of this reduced efficiency, less response preparation would have been accomplished at the time of go signal recognition in Experiment 1 than in Experiment 2, leaving more response preparation to carry out after go signal recognition in the former experiment. Thus, response preparation would start at the same time in both experiments but take longer to evoke a response in Experiment 1, causing longer response-locked LRPs in that experiment. This explanation is also quite consistent with the finding that responses were faster in Experiment 2 than in Experiment 1, especially at greater orientations.

Finally, we compared the two experiments with respect to the EMG activity generated on no-go trials. The purpose of this comparison was to evaluate the hypothesis that the extra signs of response preparation on no-go trials in Experiment 2 were produced by a larger percentage of trials on which the response was fully prepared (i.e., to the point of producing substantial EMG activity) but countermanded before a keypress actually occurred, so no trials were excluded for EMG activity before making this comparison. Contrary to the hypothesis, we found no difference in no-go EMG activity between the two experiments ($p > .10$). Thus, it seems clear that the extra response preparation seen in Experiment 2 is truly covert.

Experiment 3

The differences in results between Experiments 1 and 2 suggest that relatively little response preparation takes place during mental rotation. The most obvious interpretation of this result is that mental rotation interferes with response preparation in one fashion or another. But in some ways this interpretation is little more than a restatement of the result that there is less response preparation when mental rotation is required than when it is not. Clearly, this interference could arise in any of a number of different ways, including competition for limited resources (Kahneman, 1973), outcome conflicts (Navon, 1985), changes in processing rates or asymptotes (McClelland, 1979), and so on.

The third experiment was designed as an initial step toward elucidating further the nature of the interfering effect of rotation on response preparation. The specific possibility examined here was that mental rotation blocks the information on which response preparation is based rather than influencing the speed of preparation itself.¹³ In particular, mental rotation might prevent the subject from deciding which hand is consistent with the stimulus letter; clearly, response preparation could

¹³This idea was suggested by Ritske De Jong.

not begin until this decision had been made. On this view, it would actually be possible for response preparation to occur at the usual speed during mental rotation, if only the information needed for response preparation were available.

The present experiment was designed to test this explanation of the interference using a variant of the response priming paradigm (Rosenbaum & Kornblum, 1982). On each trial, subjects saw either a normal or mirror-image character at one of four different orientations, just as in the first two experiments. As in Experiment 1, half of the characters were assigned to each hand, and subjects were to respond or not depending on whether the character was normal or mirror-image. The new feature of this experiment was that a no-go character was sometimes accompanied, after a slight delay, by a tone presented to the left or right ear via headphones. Subjects were instructed to respond to a left or right tone with the corresponding hand, as quickly as possible.

In this paradigm, the crucial question is whether RT to a tone will be influenced by the identity of the no-go character with which it is presented. If subjects really do not determine which response hand is associated with a rotated no-go character, then RT to the tone should not depend on whether the tone requires the response with that hand ("consistent" trials) or with the opposite hand ("inconsistent" trials). Alternatively, if subjects do determine the response hand associated with the rotated character, then doing so could well prime the response, and thereby produce a priming effect on RT (i.e., faster responses on consistent than inconsistent trials). Clearly, if such a priming effect is observed, we can conclude that subjects do indeed determine the response hand associated with the rotated character. To maximize the chances of finding such priming, we varied the stimulus onset asynchrony (SOA) between letter onset and tone onset, in case the response priming lasts only a short time.

This experiment is clearly based on the assumption that response priming and LRPs are not equivalent measures of response preparation. After all, Experiment 1 has already demonstrated that the LRP shows little evidence of response preparation in this task; if response priming measured the same sort of preparation as does the LRP, then RTs should not show much response priming either. Recent evidence, however, indicates that response priming and LRP are not equivalent measures of response preparation. Measuring response priming and LRPs within a single experiment, Miller, Coles, and Chakraborty (1996) found clear evidence of response priming in the RTs, but no evidence of response preparation in the LRPs. They interpreted this dissociation as evidence that response priming indexes response preparation at a relatively central level, which can be present even without preparation at a more peripheral level indexed by the LRP. Similarly, Ilan (1995) has also reported response priming effects on RT in tasks yielding no evidence of response preparation in the LRP.

Method

This experiment was conducted using the same stimuli, responses, equipment, block structure, procedure, and types and numbers of trials as Experiment 1, except as noted otherwise. Twenty-four undergraduate students at the University of Otago, New Zealand, participated in a single 45 min session, either to fulfil an introductory psychology course requirement or for \$5 payment. All subjects reported being right-handed and having normal or corrected-to-normal vision.

Each subject participated in four blocks of 144 experimental trials, with the first block preceded by 40 practice trials and subsequent blocks preceded by four warm-up trials each. As in the previous experiments, two digits were used as stimuli in the odd-numbered blocks, and six letters were used in the even-numbered blocks.

On each trial a single character was presented (letter or digit, depending on the block), either upright or rotated 60°, 120°, or 180°, and in either its normal or mirror-image form. Assignments of characters to responses were identical to those used in Experiment 1, with the character's name

determining which hand might respond and its mirror versus normal form determining whether the character was assigned to the go or no-go response. The precise assignments of characters to left and right hands and of mirror and normal forms to go versus no-go responses were counterbalanced across subjects, as in Experiment 1.

The single stimulus character was presented by itself in 96 trials per block (characters assigned to the left versus right hands 4 orientations mirror versus normal 6 replications each), exactly as in Experiment 1, and it required a go response on half of these trials and a no-go response on the other half. In the remaining 48 trials, the subject was presented with a character whose mirror/normal form indicated that it was assigned to the no-go response, but a 900 Hz tone was also presented via headphones to either the left or right ear. In these trials, subjects were to respond as quickly as possible with the hand on the same side as the tone (e.g., left hand response to tone in left ear). The tone was 56 ms in duration, its loudness was approximately 70 dB, and the SOA from the onset of the character to the onset of the tone was either 350, 500, or 650 ms. These 48 tone-present trials included one trial at each combination of tone in left versus right ear character associated with left versus right hand SOA of 350, 500, or 650 ms character orientation of 0°, 60°, 120°, or 180°. In summary, then, on any given trial the subject was equally likely to respond to a character, respond to a tone, or not respond.

Subjects were instructed that they would see a single character in each trial, and that they should respond to this character with the left hand, right hand, or not at all, depending on the character's identity and orientation. They were also told that they would sometimes hear a tone in one ear or the other, and that they should respond immediately to any tone with the hand on the same side of the body. Subjects were informed that they would have to respond sometimes to the character and sometimes to the tone, but never to both, and that sometimes they were not supposed to respond at all. The only remaining difference from Experiment 1 was that stimulus displays were response-terminated on go trials, rather than remaining on the screen for 2.5 sec, so that it would be possible to test more trials within the 45 min session.

Results and Discussion

Results from trials without tones basically replicated the patterns obtained in Experiment 1. On average, mean RTs for correct responses to characters were 86 ms faster in blocks with memory set size 1 than with memory set size 3, $F(1; 22) = 13.5$; $p < .01$, and RT increased dramatically with deviation from upright, $F(3; 66) = 74.3$; $p < .001$ with a difference of 338 ms between upright and upside-down stimuli. Once again, there was no hint of a memory set size by orientation interaction, $F < 1$ in the RT data. Across both go and no-go trials, PCs decreased with increasing deviation from upright, $p < .001$.

Figure 8 shows mean correct RTs for trials in which subjects responded to tones. These RTs were evaluated statistically with an ANOVA having a between-subjects factor of S-R mapping and within-subjects factors of memory set size, tone/character consistency, SOA, and orientation. Overall, consistency had a 31 ms effect, $F(1; 22) = 28$; $p < .001$: Responses were faster when the tone required a response by the hand associated with the name of the simultaneously presented no-go character (i.e., consistent trials) than when it required a response by the opposite hand (inconsistent). In addition, responses to tones were approximately 50 ms slower when the orientation of the character was 180° than when it was not, $F(3; 66) = 15.2$; $p < .001$, and responses were 20 ms slower at the SOA of 300 ms than at either of the other two SOAs, $F(2; 44) = 4.23$; $p < .05$. Interestingly, the effect of consistency remained relatively constant across orientations, as indexed by the non-significance of the consistency by orientation interaction, $F(3; 66) = 0.57$; $p > .2$. This is further support (cf., Corballis et al., 1978) for the assumption, underlying the rationale of Experiment 1, that orientation hardly, if at all, affects identification speed. The three-way

interaction of orientation, consistency, and SOA shown in Figure 8 approached significance, $F(6; 132) = 2.32$; $.05 < p < .06$, but does not appear systematic.

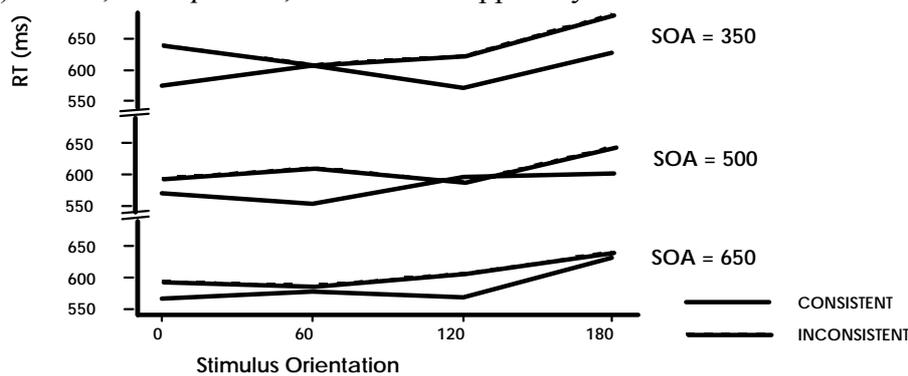


Figure 8: Experiment 3: Mean reaction time (RT) to tones as a function of character orientation, stimulus onset asynchrony (SOA), and consistency of the responses associated with tone and character.

The primary result of this experiment is the consistency effect: The response to a tone was faster when the tone indicated the hand to which the rotated character was assigned than when the tone indicated the opposite hand. This indicates that the response hand associated with the character had been determined, and that this determination caused some priming of this response hand. Clearly, this seriously undermines the hypothesis that mental rotation prevented determination of the appropriate response hand, and thus strengthens the conclusion that mental rotation interferes directly with the sort of response preparation which the LRP reflects. In addition, the fact that the consistency effect did not increase with SOA suggests that little or no active response preparation, of the sort that might increase the consistency effect or cause an LRP, could be accomplished during rotation.

The present data, in combination with those of Experiment 1, provide further indication of a dissociation between response priming and LRP as measures of response preparation. Like the data of Miller et al. (1996) and Ilan (1995), the present results suggest that evidence of preparation can be observed in the response priming measure even when it is absent from the LRP. These data are compatible with the interpretation offered by Miller et al. (1996); namely, that the priming measure indexes central preparation, which is sometimes but not always followed by more peripheral preparation of the sort causing measurable LRPs. It could be argued, for example (cf., Rosenbaum, 1985), that effects on probe RT reflect the recruitment of a motor program – in this case on the basis of the name of a character – but that subsequent activation of that program is needed to produce measurable LRP. The interpretation that a qualitative choice could overlap with mental rotation, but that activation of the response associated with that choice was obstructed, is consistent with the possibility that the interference stems from a limited capacity pool of resources needed by both the mental rotation and activation processes (e.g., Hockey, 1993; Kahneman, 1973), because resource theories tend to identify capacity limitations with intensive or activational aspects of information processing.

General Discussion

The results of Experiment 1 indicate that response preparation can begin before mental rotation is finished, at least in a task with one character assigned to each of two responses. This conclusion is supported both by the presence of stimulus-locked LRPs on no-go trials and by the dependence of response-locked LRPs on orientation on go trials. In addition, the consistency effect

obtained in Experiment 3 confirms that some response-level processing can occur before the end of mental rotation.

The results also clearly indicate, however, that mental rotation interferes with response preparation. Compared with preparation that occurs while waiting for a go or no-go color (Experiment 2), preparation that occurs before the end of mental rotation is less effective in producing LRP. This difference is evident both in smaller stimulus-locked LRPs on no-go trials and in longer lasting response-locked LRPs on go trials. Judging from the fact that RTs were longer in Experiment 1 than in Experiment 2, the reduced LRPs in the former case are indicative of less effective preparation for the upcoming behavioral response.

The present results have clear implications for extant models of information transmission. At least for this task, purely discrete models are refuted by the finding that some response-level processing begins before mental rotation is finished, because such models assume strictly serial processing (e.g., Donders, 1868/1969; Sternberg, 1969). Thus, the present results extend previous evidence against discrete models (e.g., Miller & Hackley, 1992; Osman et al., 1992) by showing that response selection and preparation can sometimes begin before the termination of even a fairly demanding process like mental rotation.

Continuous models are compatible with the present evidence of processing overlap, but nonetheless need some modification to handle the present results. At least as described previously, these models do not explain the interference between mental rotation and response preparation, because they do not contain provisions for interference between different stages operating simultaneously (e.g., Eriksen & Schultz, 1979; McClelland, 1979). The same difficulty arises with hybrid models, like the ADC model, that allow stages to operate simultaneously in certain cases (e.g., Miller, 1982, 1993; Schweickert, 1978). The present results suggest that such models must be elaborated to include some such sources of interference, such as resource competition (e.g., Kahneman, 1973; Navon & Gopher, 1979), blocking of a single-channel mechanism (e.g., Pashler, 1992; Welford, 1967), or outcome conflict (e.g., Navon, 1985; Navon & Miller, 1987).

How general are the conclusions that response preparation can begin before mental rotation is finished and that mental rotation interferes with response preparation? Experiment 1 departed from the standard mental rotation task in a number of ways (e.g., requirement to respond to stimulus identity as well as mirror/normal form, presence of no-go trials), and one might argue that these paradigmatic differences created a special task that produced atypical processing. Unfortunately, one may never show that this argument is absolutely wrong, but its plausibility is low when the experiment produces effects of the usual sizes. In this experiment, the effects of both stimulus orientation and memory set size were quite similar to those found previously (cf., Cooper & Shepard, 1973; Sternberg, 1969).

Another possible reservation about the generality of the findings is that subjects might be able to accomplish more response preparation before the end of mental rotation if given more incentive to do so. As already emphasized, however, the long duration of mental rotation provided ample opportunity for preliminary response preparation to occur, and the incentive for such preparation was no less in Experiment 1 than in Experiment 2. Comparing across studies, then, one must conclude that it is more difficult to prepare responses while rotating than while making simple perceptual discriminations. This conclusion implicitly concedes the idea that mental rotation interferes with response preparation, and it is incompatible with continuous models in which successive processes operate in parallel and without interference because they involve different neural systems.

The present conclusions also seem to have some generality because they converge well with four previous findings concerning the relationship of mental rotation and response preparation. First, in an experiment of Goldberg, Meyer, Yantis, and Langolf (1984), the mirror/normal form of

a rotated "prime" letter predicted the response to a subsequent test stimulus, and the prime preceded the test by either a short, medium, or long duration. The probability distribution of RTs following the medium-duration primes was not simply a mixture of the distributions of RTs following short- and long-duration primes, indicating that the response preparation induced by the prime was not all-or-none but instead went through at least one intermediate level. This finding is quite consistent with the present, LRP-based, evidence of partial response preparation (e.g., on no-go trials) in mental rotation tasks.

Second, Ilan and Miller (1994) examined performance in tasks requiring mirror/normal discriminations and found faster responses to upright stimuli in blocks containing only upright stimuli than in blocks containing a mixture of upright and rotated stimuli. In addition, the advantage for upright stimuli in all-upright blocks was much larger in a choice-RT version of the task than in a go-no-go version. Because choice and go-no-go tasks differ with respect to response selection and preparation processes, this interaction suggests that subjects had particular difficulty simultaneously maintaining readiness for mental rotation and selection or preparation, which is consistent with the idea that these two operations interfere with one another. Because the results of our third experiment suggest that there is not too much interference between mental rotation and response selection, the interference observed by Ilan and Miller now seems most likely to have resulted from a competition between rotation and response preparation.

Third, Ruthruff et al. (1995) studied mental rotation using the psychological refractory period (PRP) paradigm, in which the subject is presented with two stimuli in rapid succession and must make a separate response to each one. Using a diagnostic developed by Pashler (1984), they found evidence that mental rotation of a second stimulus does not begin until after a single-channel mechanism has finished selecting the response to the first stimulus (but see Van Selst & Jolicoeur, 1994, for studies supporting the opposite conclusion). If mental rotation and response selection do require access to the same single-channel mechanism, as Ruthruff et al. concluded, then they would certainly be expected to interfere with one another in the current paradigm. In fact, on this view it is a bit of a mystery why our subjects were able to do any preliminary response preparation at all in the mental rotation task of Experiment 1, and why they showed evidence of response priming in Experiment 3. One might suggest that our subjects selected and prepared responses while they were making mirror-normal discriminations, not while they were mentally rotating. It is indeed possible that some response processing occurred during the mirror-normal discrimination, but such processing cannot be the whole story. By itself, it would not explain the early appearance of LRP in the stimulus-locked waveforms (300-500 ms after stimulus onset), since there is good reason to believe that mental rotation could not have finished that early. Nor can it explain the effect of orientation on response-locked LRPs: If orientation only prolongs processes prior to the mirror/normal discrimination, then the interval from the onset of mirror/normal discrimination to the response should be independent of orientation. Another possibility is that our subjects carried out some response selection and preparation before they began mental rotation, with no further preparation carried out during rotation. This explanation is consistent with the fact that no-go LRP amplitude failed to increase with stimulus orientation in Experiment 1, despite the increasing time available for preparation during rotation, although this fact could also be explained by arguing that the expected orientation effect was small and the experiment had insufficient power to detect it. Most importantly, however, this explanation is entirely consistent with the conclusion that some aspect of mental rotation interferes with the subject's ability to prepare responses at the level indexed by the LRP.

Fourth, Stoffels (1996) studied performance in a four-choice RT task in which the four stimuli were normal and mirror-image versions of two letters and the four responses were made by the index and middle fingers of the two hands, using a paradigm developed by Miller (1982). When all

stimuli were upright, so that no mental rotation was required, Stoffels found that responses were faster when the normal and mirror-image versions of a given letter were assigned to response fingers on the same hand than when they were assigned to fingers on opposite hands, suggesting that subjects could prepare the response hand indicated by the name of the letter while they were making the mirror/normal discrimination. When stimuli were rotated, however, RT no longer depended on the stimulus-response assignment, as if preparation were no longer possible. From these findings, Stoffels concluded that the requirement to perform mental rotation prevents the subject from preparing the hand indicated by the name of the letter.

If we can generalize beyond mental rotation to the class of effortful processes that it was chosen to represent, the ultimate conclusion of the present study is that one cannot extend to such processes the types of massively parallel models (e.g., McClelland & Elman, 1986; Rumelhart & McClelland, 1987) currently being considered for automatic processes like simple perceptual analysis and consistently-trained response selection (Schneider & Shiffrin, 1977). Although there is evidence that certain types of simple perceptual discriminations may be performed in parallel with response preparation with little interference (e.g., Miller & Hackley, 1992; Osman et al., 1992), it appears that the more deliberate processes are probably carried out mostly one at a time, due to interference, even when the task provides considerable opportunity for parallelism. This conclusion is perfectly consistent with the conclusion emerging from studies of overlapping tasks (e.g., Pashler, 1992) – that people are generally unable to do two tasks at the same time because the tasks must queue up for access to a computational device that can only work on one at a time – and in fact extends it by showing the sequentiality of bottleneck operations involved in selecting a single response. It remains to be determined whether this sequentiality is due to the use of a single general-purpose computational device (e.g., Welford, 1967), to resource limitations (e.g., Kahneman, 1973), or to other system constraints (cf., Navon, 1985).

Finally, because memory set size was varied in both experiments, the present results also shed some light on the relationship between processes of response preparation and short-term memory search. Both experiments yielded larger no-go LRPs with smaller memory set sizes (the effect was statistically reliable in Experiment 1), suggesting that memory search interferes with response preparation. The interpretation of this effect is not entirely clear, however, because subjects had to complete memory search before knowing which hand should be prepared. Naturally, anything that delayed the completion of memory search (e.g., larger set size) would be expected to reduce preparation, if only by delaying its onset. We cannot, therefore, conclude that memory search per se interfered with response preparation; a test of that hypothesis requires a task in which the subject can determine which hand to prepare before starting the memory search, just as the present task allowed the subject to know which hand to prepare before starting to rotate.

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5 Optimizing the Design and Interpretation of the Stop-Signal Paradigm¹⁴

Abstract

Performance on the stop-signal paradigm is usually interpreted on the basis of a horse-race model (Logan & Cowan, 1984). According to this model the relative finishing times of a response-generating process and a response-inhibitory process determine whether a response can be withheld or not. Violations of assumptions that underlie the horse-race model can affect conclusions about inhibitory efficiency. The behavior of the horse-race model was simulated under violations of the independent-process assumption, the constant stop-speed assumption, and other changes in parameters of the race. Different methods for evaluating inhibitory efficiency were compared. The results suggest that the speed of stopping can be estimated reliably, but there are presently no reliable methods to estimate variability in inhibitory control. Efficient measurements of stop-speed are possible with the staircase-tracking algorithm employing 400 trials. The relationship between effect size and the statistical power was investigated under several degrees of population heterogeneity.

Inhibitory or executive control is a concept with increasing importance for psychological theories about development and psychopathology as well as normal adult performance (see Logan & Cowan, 1984, for a review about the control concept). Complete suppression of an action is one of the most straightforward examples of control. For example, while hammering a nail, you may realize that the next hit will be on your thumb. To avoid injuries you need to freeze your hand; which requires a fast and reliable inhibitory system. More subtle forms of inhibition are required for adaptation, rather than cancellation of ongoing behavior. After you hit your thumb, the words that first come into mind may need some adjustment, but you do feel the motivation to speak out loud.

The efficiency of inhibitory control can be investigated with the stop-signal paradigm. It is a laboratory equivalent of every-day tasks that call for a change (e.g. Logan & Burkell, 1986) or abortion of actions (Lappin & Eriksen, 1966; Logan, 1994; Logan & Cowan, 1984). The stop-signal paradigm allows a careful examination of response inhibition, in connection with factors such as the task to be stopped and the timing of the signal to stop. The quantitative interpretation of performance on the stop-signal paradigm is enhanced by the horse-race model, which basically asserts that the inhibitory process and the to-be-suppressed process compete for the first finishing time (see Logan & Cowan, 1984, for an analytic approach). This model allows the calculation of the speed of stopping on the basis of a small set of assumptions. At the same time, predictions from the model are used to test its validity. Usually, empirical data can be described quite well by the horse-race model, and the tests of the model support the validity of the model (e.g. De Jong, Coles, Logan, & Gratton, 1990; Jennings, Van der Molen, Brock, & Somsen, 1992; Logan & Cowan, 1984; Osman, Kornblum, & Meyer, 1986).

It has been shown that the stopping process can be exerted on a variety of primary tasks, whereas the speed of stopping is relatively constant. For example, the speed of stopping of young adults was close to 200 ms when subjects tried to stop continuous actions such as typing (Logan, 1982), responses that appear to be overlearned, such as speaking (Ladefoged, Silverstein, & Papcun,

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1973), or controlled actions, such as responses on a stimulus-response incompatible instruction (Logan, 1981). Thus, parsimony argues for a single stopping mechanism that can be used to stop a variety of tasks. However, if different primary tasks would require different stop-mechanisms, this theoretical dependence would not affect the validity of the race model and its calculations.

Stop-signal inhibition should be distinguished from other forms of inhibition, such as neurological inhibition and reactive inhibition (see Logan, 1994, for a review). Stop-signal inhibition comprises a top-down process, whereas neurological inhibition is a low-level contribution to a mental activity at a larger scale (e.g. Rumelhart & McClelland, 1986). Furthermore, although stop-signal inhibition as a whole works against the activation of a response, the name inhibition does not imply that a neurological model of response inhibition would consist primarily of inhibitory connections. Reactive types of inhibition, such as inhibition of return (Posner & Cohen, 1984), negative priming (Tipper, 1985) and refractory effects in dual-task situations (Welford, 1952), differ from stop-signal inhibition because they are unintended side-effects of a different mental activity, whereas stop-signal inhibition is an entire intentional action.

The race model makes it possible to calculate the speed of stopping and other indices of inhibitory efficiency, and thus allows a comparison of the inhibitory process between task conditions (e.g. Kramer, Humphrey, Larish, & Logan, 1994) and between subject groups (e.g. Schachar & Logan, 1990b). Research in this domain has revealed some interesting changes in the efficiency of stopping over the life span, and differences between clinical groups and normal controls.

As a result of improvements to the horse-race model methodology of the past 12 years, more subtle differences in inhibitory efficiency are investigated, such as the variability of the inhibitory process. At the same time, however, the increased refinement of the methodology sets higher demands for the validity of the race model. It is therefore important to quantify how violations of the horse-race assumptions can affect measures of inhibitory efficiency, and validate dependent variables. In addition, measurement error and heterogeneity in a population can affect the power of statistics, as dependent measures are calculated through more steps. These two issues; i.e. the effect of violation of horse-race assumptions, and the power of statistics that are based on the model, are explored in the following.

In this paper, performance on the stop-signal paradigm is simulated on the basis of the horse-race model in its original form and in forms where single parameters are manipulated. These Monte Carlo simulations serve several goals. First of all, they expose the consequences of violations to the assumptions of the horse-race model and of other factors of the primary process and inhibition process that affect its behavior. The second goal of the simulations is to ease the interpretation of results from the stop-signal paradigm, and to provide illustrations of possible outcomes given a number of differences in task performance and inhibitory control. Finally, the study aims to show the balance between the power of statistics that are derived from the horse-race model and the number of measurements under several levels of heterogeneity in the population.

In the following sections the stop-signal paradigm and the horse-race model will be explained in more detail, and experts in the field can skip this section without missing the point of the article. These readers are referred to Study 1, in which the effects of parameter changes to the horse-race model are discussed. Researchers who are primarily interested in suggestions that can minimize the costs of their stop-signal experiment can jump to Study 2, in which the accuracy of measurements is discussed, or to Study 3, where power and design issues are explored. The following sections provide a deeper motivation of the article, and may be important for readers who are not experienced in the stop-signal paradigm. They also discuss the method of the simulations and the dependent variables that are covered in the results.

The paradigm

In the stop-signal paradigm, subjects perform on a task that is occasionally supplemented with a signal that instructs subjects to withhold the response that was in preparation, if possible. In a typical setting, the primary (go) task is a reaction time (RT) task, in which the stimuli 'X' and 'O' are assigned to left- and right-hand responses. On 25% of all trials, an auditory signal is presented after a variable delay, called the stimulus onset asynchrony (SOA). The remaining 75% are nonsignal trials. The paradigm yields data about the response rate (RR), or conversely, the inhibition rate on signal trials, as a function of SOA. This relationship is known as the inhibition function. Furthermore, it yields reaction times for signal and nonsignal trials.

Since the first experiments with stop-signals (Vince, 1948), there has been an increasing refinement in stop-signal methodology, partially as a result of changes in research goals. While initially, stop-signals were employed primarily as part of psychological refractory period experiments (e.g., Harrison, 1960; Slater-Hammel, 1960; Vince, 1948; Welford, 1952), later studies addressed the stopping process itself, through the description of RT and RR as a function of SOA (e.g. Lappin & Eriksen, 1966; Ollman, 1973). Only more recently the inhibition function, which describes the relation between the SOA and inhibitory success, became a tool for the calculation of the speed of stopping (stop-signal reaction time, SSRT; e.g. Logan & Cowan, 1984; De Jong et al., 1990).

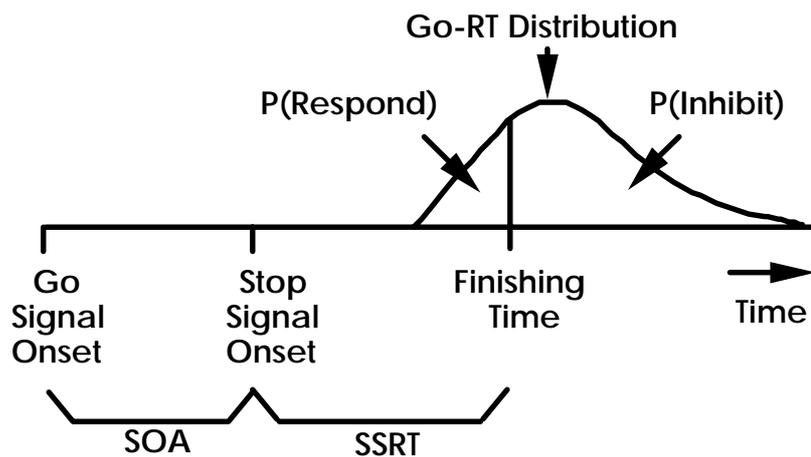


Figure 1. An illustration of the horse-race model of response inhibition. The stop-signal is presented after a stimulus-onset asynchrony (SOA) relative to the go-signal. The stop-process finishes after the stop-signal reaction time (SSRT) relative to the stop-signal onset. Assuming that the SSRT is constant, this finishing time intersects the distribution of go-RT. Responses from the left part of the go-RT distribution are too fast to be inhibited, whereas responses from the right part are correctly withheld. Thus, the finishing time of the stop-process divides the go-RT distribution into the chance of responding; $P(\text{Respond})$ and the chance of inhibiting; $P(\text{Inhibit})$.

For the interpretation of performance on a stopping task, several authors have proposed that the success of the inhibitory process depends on a race against the go-process (Lappin & Eriksen, 1966; Logan, 1981; Osman et al., 1986; Vince, 1948). The formal horse-race model (e.g. Logan & Cowan, 1984; Ollman, 1973) holds that the two processes are independent and take part in a race, and that the winner determines whether a response is withheld or executed (Lappin & Eriksen, 1966; Vince, 1948). If the inhibitory process finishes before the go-process ($RT > \text{finishing time}$), response inhibition is successful, otherwise ($RT < \text{finishing time}$) a commission error or signal response is made.

Figure 1 illustrates how variation of SOA, go-RT or SSRT affects the probability of inhibition. It displays the finish line of the stop-process, determined by the SOA and SSRT on a given trial relative to the distribution of go-RTs across trials. For example, changes in the SOA shift the finish line relative to the RT distribution. The finish line also moves if the SOA remains the same but the SSRT varies. If the finish is delayed and the RT distribution remains the same, a larger proportion of the go-distribution falls to the left of the finishing time, and therefore more trials escape the inhibitory control. If the RT is increased, however, the same finish line is projected onto an earlier point of the RT distribution. Because there are then less responses to the left of that line, there is a higher chance of inhibition. If the variability of go-RT is high, the effect of moving the finish line is smaller than if the variability is low; because the same shift of the finish line passes a smaller part of the RT distribution.

Stop-speed

The speed of stopping can be derived from observed RTs on nonsignal trials, and the inhibition function. Under the assumptions that the speed of the go-process is not affected by the presence of stop-signals, the nonsignal-RT distribution can be treated as the underlying distribution of go-processes on signal trials. As the stop-signal paradigm contains a large number of nonsignal trials, a high-resolution estimate of the cumulative distribution of the go-process duration is obtained. Meanwhile, signal trials yield a response rate for every SOA.

In order to estimate the true speed of stopping $SSRT_{in}$ (input SSRT, or internal SSRT), four methods have been proposed that require different assumptions and different levels of detail of input information. The basic idea behind all calculations is that the $SSRT_{Obs}$ (observed SSRT) is the difference between the starting time and the finishing time of the stop-process (Logan, 1981). The start is known to be the SOA, but the finishing time needs to be estimated on the basis of RTs. If it is assumed that the speed of stopping is constant, then the finishing time of the stop-process is also constant on a given SOA. The response rate represents the proportion of go-processes that finish before the constant finishing time. This logic implies that the finishing time of the stop-process equals the x th percentile score of the go-RT distribution, in which x is the response rate. Thus, with this first method, the $SSRT_{Obs}$ can be calculated for every SOA, by subtracting the SOA from the x th percentile score (Logan, 1981; Logan, Cowan, & Davis, 1984).

An advanced way to determine the speed of stopping needs to take variability of the stop-process duration into account (Colonius, 1990; Logan & Cowan, 1984; Osman et al. 1986, 1990). If the preceding method is used, variability in the speed of stopping leads to the incorrect observation that SSRT decreases with SOA (De Jong, et al. 1990; Logan & Burkell, 1986). This is the case because on early SOAs the race can even be won by relatively slow stop-processes, whereas on late SOAs only very short $SSRT_{in}$'s stand a chance to finish before the go-process. Logan and Cowan proposed two methods for the estimation of $SSRT_{in}$, that do not require the assumption of a constant $SSRT_{in}$. One summary score of the speed of stopping can be obtained by calculating the average of the $SSRT_{Obs}$'s on different SOAs. We will refer to this measure as *average SSRT* ($SSRT_{Av}$).

A different method is based on the entire inhibition function and on mean nonsignal RT. The inhibition function can be interpreted as a distribution, and its mean represents the average SOA. Similarly, mean RT represents the average finishing time of the go-process. Therefore, a single measure of $SSRT_{Obs}$ can be calculated as the distance between the mean of the inhibition function and mean RT. This second measure will be referred to as the *distance SSRT* ($SSRT_{Dist}$). The method can be facilitated somewhat if the central SOA, rather than the mean of the inhibition function is used. Note, however, that this approach is based on the assumption that the inhibition

function is symmetrical. It is also possible to subtract central SOA from the median RT (Logan, 1994; Logan & Cowan, 1984). This third procedure will be referred to as the *median SSRT* ($SSRT_{Med}$).

Although $SSRT_{Dist}$ and $SSRT_{Med}$ methods are considered to be robust against $SSRT_{in}$ variability, it does not give an estimate of how variable the stop-process is. However, there is a method, proposed by De Jong et al. (1990, appendix) and Colonius (1990) that is meant to recover the distribution of $SSRT_{in}$ for every SOA. In this method, the cumulative distribution of $SSRT_{Obs}$'s is calculated based on the ratio between the density functions of signal and of nonsignal RT, and the probability of responding on signal trials. Formally,

$$P(SSRT + SOA > t | SOA) = RR(SOA) \cdot f_s(t | SOA) / f_{ns}(t) \quad (1)$$

where as a function of time t , $P(SSRT + SOA > t | SOA)$ is the cumulative distribution function of SSRTs, given a SOA; $RR(SOA)$ is the response rate at SOA; and f_s and f_{ns} are the density functions of the signal and nonsignal RT-distributions respectively.

Because Equation 1 estimates the entire distribution of $SSRT_{in}$, it is possible to derive the median from that distribution ($SSRT_{Col50}$) as a fourth estimation of stop speed that, but it is also possible to estimate variability of $SSRT_{in}$ with the interquartile distance ($SSRT_{Col75} - SSRT_{Col25}$)

Unfortunately, this method requires smoothed and stable distributions of RT and a monotonic inhibition function (e.g. Osman et al., 1986). As experiments do not usually employ more than 30% signal trials, the signal RT distribution has to be based on approximately 15% of all trials, given that half of all responses are inhibited. Moreover, these signal trials are usually distributed over several SOAs, which leaves even less data to describe a distribution per SOA. Possibly for these reasons, this method does not seem to be in use in empirical studies.

A different method for estimating the variance of $SSRT_{in}$ is suggested by Logan and Cowan (1984). They argued that the inhibition function can be interpreted as the cumulative distribution of a random variable T_d , and its variance can be calculated from the slope of the inhibition function at the *central SOA* (i.e. the SOA where $RR = .5$). For example, if a normal distribution is assumed, the relation between the slope and standard deviation is

$$\text{slope at median} = 1 / [SD_{Td} \cdot (2\pi)^{-1/2}] \quad (2)$$

As the only sources of variance in the inhibition function are the variance in the arrival of the stop- and go-processes, and as these sources are assumed to be independent, the variance of T_d is the sum of the variances of RT and SSRT. Therefore, the SD_{SSRT} can be calculated by

$$SD_{SSRT}^2 = \left(\frac{1}{\sqrt{2\pi} * \text{Slope}} \right)^2 - SD_{RT}^2 \quad (3)$$

Other measures from the horse-race model

Although the speed of stopping has received a lot of attention in the literature, the horse-race model provides more measures that are of interest. The inhibition function shows how the response rate is related to SOA, and this relationship follows a sigmoid function, which holds that the slope of the function is small in the ranges close to 0 and 100% inhibition, whereas it is steep around the central SOA. If the extremes are ignored and the range of SOAs between 15 and 85% inhibition is considered, the function is close to being linear. The slope and position of the linear regression function through the middle range of the inhibition function can be further explored with help of the horse-race model.

The position of the mean of the inhibition function can be explained with mean RT, SOA and $SSRT_{Obs}$ (Logan & Cowan, 1984). In contrast, the slope of the inhibition function in the middle range primarily contains information about the variability of go-RT. But if the slope is corrected for the variability in go-RT, other contributions are revealed, which stem from other trial-to-trial differences in inhibitory control, the go-process or SOA. All of these corrections are intended to express the inhibition function in terms of the relative finishing times, so that the remaining slope can be interpreted in terms of factors that have not been removed. Variation of the SOA can be the result of the algorithm for adjustment to go-RT or success at stopping, and the speed of stopping is also likely to vary. An occasional failure to trigger the stop-process or the go-process also affects the slope of the inhibition function, because in the former case it is impossible to reach 100% inhibition and in the latter it is impossible to reach 100% responses at any SOA. In other words, these failures cause the inhibition function to level off. The failure to trigger the go-process may occur in anticipation of stop-signals, but can be recognized, because it also affects the response rate on nonsignal trials. A failure to trigger the stop-process can be considered as an inhibitory deficiency. In this way, the horse-race model provides an explanation of the inhibition function, and stepwise corrections can reveal what is going on during performance on a stop-task.

The correction of the inhibition function for individual task parameters has also been used to detect the source of differences between groups of subjects (Logan, 1994; Logan & Cowan, 1984; Schachar & Logan, 1990b). For example, Schachar and Logan were able to remove differences between the inhibition functions of different age groups by displaying response rate as a function of $RT - SOA$, and therefore concluded that the difference in the inhibition function was due to developmental differences in the speed of the go-process. In a comparison of ADHD children with normal controls, however, differences remained. Schachar and Logan performed a corrections for the average $SSRT$; $(RT - SOA - SSRT_{Av})$, yielding the relative finishing time of the stop-process, and for the standard deviation (SD) of the go-process (Equation 4), yielding a Z -score of the relative finishing time ($ZRFT$). As neither correction was sufficient to align the inhibition functions of ADHD children and to that of normal controls, it was concluded that differences in go-RT, SD_{RT} and $SSRT_{Av}$ were not responsible for all the observed differences between the groups. Schachar and Logan hypothesized that the differences in inhibitory control of these two populations were localized in other factors, such as $SSRT_{in}$ variability or a deficiency in triggering the stop-process.

$$ZRFT = f(SOA) = (RT - SSRT_{Av} - SOA)/SD_{RT} \quad (4)$$

In addition to measures of inhibition, the horse-race model yields predictions about the speed of signal responses, which are used to test its validity (Logan & Cowan, 1984). Predicted RT of signal responses are calculated with the assumption that the finishing time of the stop-process is constant. Figure 1 illustrates how the constant finish line divides the go-RT distribution into signal responses that are withheld, and responses that are executed. According to the horse-race model, signal RTs correspond to the part of the nonsignal RT distribution that lies to the left of the finish line. However, a deviation from independence in the model is thought to lead to an increase of the difference between the two distributions (e.g. De Jong et al., 1990; Jennings et al. 1992; Logan & Cowan, 1984). Influences from the inhibitory process on the speed of the go-process are expected to be reflected by a difference between mean observed and predicted signal RT. This has been reason to treat the difference between these means as a test of the independence assumption. Of course, it is recognized (Logan, 1994; Logan & Cowan, 1984) that the speed of stopping is not constant. In consequence, the finishing time of the stop-process does not work as a sharp and consistent separator between fast and slow go-processes like in Figure 1 (see also Logan & Cowan, 1984). Relatively fast go-processes are inhibited on some trials, whereas relatively slow go-processes may

occasionally escape inhibition. It is therefore likely under the independence assumption that the observed signal RT is higher than predicted signal RT, and this expectation is confirmed by empirical findings (De Jong, et al., 1990; Logan, 1994; but see Jennings et al. 1992 for an opposite observation). De Jong et al. have shown with simulated data that a difference occurs as a result of $SSRT_{in}$ variability, even if RT and $SSRT_{in}$ are uncorrelated. This suggests that a *t*-test for differences between observed and predicted RTs is more conservative than is necessary to test the validity of the independence assumption.

Issues in stop-signal research and the horse-race model

In the following section, four issues about the stop-signal paradigm and the horse-race model are explained, as an introduction to the simulation studies. The first issue concerns the way to handle the delivery of stop-signals. It is a design consideration with potential consequences for interpretability of the data and the duration of an experiment. The second issue concerns a crucial assumption of the horse-race model; independence of the stop- and go-processes. Although research with normal adults suggests that the race model describes empirical data quite well, research from other paradigms challenges the plausibility of the model. Because a violation of its assumptions needs to be recognized and the consequences of it need to be known, different degrees of violations are simulated. Third, there is a theoretical consideration. Regardless of whether the horse-race model holds for empirical data, researchers want to be able to explain differences between conditions or between groups. If a difference stems from deficiencies in inhibitory control, it is important to know the locus of the deficiency. The simulations show what the possibilities are to demonstrate differences with the present methodology. The robustness of indices of inhibitory deficiencies is tested with variations to the go- or stop-process. Finally, the issue of statistical power is addressed for the stop-signal paradigm. It needs to be acknowledged that empirical data are not as homogeneous as computer-generated data can be. Therefore, the effect sizes are related to the influence of within-group differences, and it is calculated what the sample size should be before deficiencies can be shown with sufficient statistical power.

When to present stop-signals

An important feature of the stop-signal paradigm in comparison with other paradigms for the measurement of inhibitory efficiency (e.g. the continuous performance test; Halperin, McKay, Matier, & Sharma, 1994, and the go/no-go task; Weber, 1993) is the possibility to manipulate the delivery time of the inhibition stimulus. As a result of changing the SOA, the finishing time of the inhibitory process in the race against the go-process can be moved to the latencies that are most interesting for the assessment of the inhibitory function. It is not very informative to extend measurements for the inhibition function outside the response-rate range of .15 - .85. Selection of SOAs should primarily be based on this consideration.

However, SOAs should also be selected with the strategy of subjects in mind. If the stop-signal is always presented after 50 ms, it suffices for the subject to await the first 60 ms in order to reach a perfect score (Lappin & Eriksen, 1966; Logan, 1981). One way to prevent such a strategy, is by mixing early and late SOAs, so that there is no way to escape all stop-signals. A more direct way is to make adjustments to the timing of the stop-signal as subjects increase their scores.

But there is yet another anticipation effect. There is a tendency to delay responses as the percentage of trials with stop-signals increases (Logan, 1981; Logan & Burkell, 1986). In order not to affect the speed of the primary task, a rate of approximately 25% stop-signal trials is recommended (Logan, 1994; Logan & Burkell, 1986). Unfortunately, lowering the percentage of stop-signal trials, as well as mixing early and late SOAs, imposes restrictions on the number of trials that can be presented per condition within an average experimental session. By selecting the

proper number of SOAs and the proper way to avoid strategy effects, stop-signal experiments can be run in shorter sessions and with less investment of time and finance. The present article is meant to help in this selection.

In early studies of stopping, the SOAs were often selected as constants (e.g. Lappin & Eriksen, 1966; Logan & Burkell, 1986), ranging from 0 ms to the RT. One advantage of the *fixed SOA* procedure is that it is easy to implement on a computer. A disadvantage is that many different SOAs are required to be prepared for individual differences in RT and $SSRT_{in}$. Strategic effects can be another problem, because subjects can anticipate the timing of stop-signals. For example, if early and late SOAs are combined, subjects can choose to await the occurrence of the early, but to risk the occurrence of a late stop-signal.

Other studies employ adaptive procedures for the determination of SOA. In one such a procedure, SOAs are adjusted to momentaneous changes in RT (e.g. Logan et al., 1984; Schachar & Logan, 1990b). For example, Schachar and Logan determined the mean go-speed after each block of trials, and subsequently presented stop-signals on SOAs of $RT - 500$ ms, $RT - 400$ ms, $RT - 300$ ms, $RT - 200$ ms, $RT - 100$ ms and $RT - 0$ ms. The advantage of this *RT-tracking* procedure is that individual differences in RT, as well as strategic hesitation in anticipation of a stop-signal are compensated for. Schachar and Logan displayed the probability of inhibition as a function of $RT - SOA$, and found that these functions covered the range from approximately 70% inhibition to 20% inhibition for all age groups between seven years and adults, even though there was a 303 ms effect of age on mean RT. The functions almost overlapped: There was no difference between the slopes, but there was a slight difference in the position of the function, that could be explained by the differences in $SSRT_{in}$.

There are several alternative ways of RT-tracking to compensate for individual differences or changes in RT. The timing of the SOA can be adjusted from trial to trial or after each block. Adjustments to changes in RT should be performed frequently, however, or else the occurrence of the stop-signals may become predictable. Furthermore, SOAs can be based on mean RT alone (Schachar & Logan, 1990b), or on its distribution (Kramer, et al. 1994; Logan et al., 1984). It may not always be sufficient to adjust SOAs to mean RT if there is a large amount of RT-variability. Subjects with a high variability of RT can best be tested with large differences between SOAs, because the range of the inhibition function is primarily determined by the variance of go-RT. Kramer et al. implemented the RT-tracking procedure by subtracting a constant period, equal to the individual simple RT, from the 20th, 40th, 60th and 80th percentile scores of go-RT. As a result they covered approximately the same section of the inhibition function for young and older adults subjects, despite differences in variability of RT.

Another algorithm, known as the staircase-tracking algorithm (Levitt, 1970; cf. Logan, Schachar, & Tannock, in press; Osman et al. 1986, 1990) adapts to the response rate, and will therefore be referred to here as the *RR-tracking* procedure. This procedure inherently corrects for differences in the RT-distribution and for the tendency to postpone responses. An additional advantage is that the response rates remain almost constant across groups, despite differences in the efficiency of inhibitory control. After a period of adjustment, the SOAs vary around values that are most informative, and the mean of the SOAs within a level can subsequently be used for further calculations. For example, Osman et al. (1986) referred to three different SOAs as early, middle and late. If a stop-signal was presented on the middle SOA and the response was not inhibited, the next value of the middle SOA would be 50 ms earlier, so that the chance of inhibition was higher, whereas correct inhibition was followed by an increase of the delay by 50 ms, which made it harder to inhibit the response. As a result, only 50% of all responses were stopped at the middle SOA. For the early and late SOAs, the latencies were also adjusted, but asymmetrically for inhibition and response trials. For early SOAs, every failure to inhibit was followed by a decrease of the SOA by

50 ms, but the SOA was only increased after every second correct inhibition. This algorithm led to a response rate of 29%, and the late SOA evoked approximately 71% responses due to the converse algorithm (see Levitt, 1970, for a mathematical discussion of this procedure). Because the method worked so well for finding the SOA that evoked 50% responses, his later experiments were run with only the ‘middle’ SOA (Osman, 1990; see also Logan et al., in press). This procedure has the advantage that more data are acquired per condition, but it also carries a restriction. The exploration of the inhibition function in search of individual differences in, for example, variability of $SSRT_{in}$, requires several SOAs at some distance from each other, and this type of information is less readily available if the SOA moves around the center of the inhibition function.

The fact that the fixed-SOA, RT-tracking and RR-tracking procedures all yielded $SSRT_{Obs}$'s close to 200 ms for normal adults suggests that all these procedures are at least to some extent reliable. However, it was shown in the above that variation in the finishing time of the stop-process contributes to variance in the inhibition function. This observation argues against variable SOAs when the slope of the inhibition function is investigated. However, other arguments argue in favor of a tracking procedure, such as the economy of design and the equality of response rates. The simulations in this article help to choose between the SOA procedures, because they illustrate what the weight of the different arguments is.

The independence assumption of the horse-race model

Even though the horse-race model is a rather simple model, based on only a small number of assumptions, it is accepted as a useful model without obvious fallacies. For example, Jennings et al. (1992) listed four arguments that support the independence assumption of the race model. First, the horse-race model can explain empirical data quite well (cf. Logan et al., 1984). Second, there is sufficient similarity between observed and predicted signal RT. Third, Jennings et al. found no effect of SOA on $SSRT_{Obs}$. Finally, they state that the sufficiency of the model is supported by the observation that there is a consistent speed of stopping across tasks, ranging from 200 - 250 ms for a variety of primary tasks.

Calculation of $SSRT_{Obs}$, as well as other measures of inhibitory control, is based on the assumption that the go-process and the stop-process are running independently. The assumption of independence takes two related forms; context independence and stochastic independence. Context independence holds that the primary task is not affected by the presence of the stop-process and vice versa, and also applies to differences in $SSRT_{in}$ due to SOA manipulations. The second form of independence is the stochastic independence of the go- and stop-processes. It holds that variations in the go-process are not correlated to variations in the stop-process. To some extent, context independence and stochastic independence overlap. These assumptions make it possible to treat the nonsignal RT-distribution as the distribution of primary-process duration on stop-trials. It is easy to see that if the nonsignal RT distribution is different from the signal RT distribution, the $SSRT_{Obs}$ is miscalculated. For example, if the primary process is delayed with 50 ms by the presence of a stop-signal, all other things being equal, the $SSRT$ is underestimated by 50 ms.

Context independence

A common observation in stop-tasks is that the $SSRT_{Obs}$ decreases with SOA, suggesting that there is an interaction between the primary task and the speed of stopping. However, the decrease of $SSRT_{Obs}$ with SOA can be explained with the behavior of independent stochastic processes in a race model (Logan & Burkell, 1986), given that the $SSRT_{in}$ is variable. On early SOAs, the race between the average go-process and the stop-process is won with relatively slow $SSRT_{in}$'s, whereas the race can only be won with relatively fast $SSRT_{in}$'s on late SOAs. This explanation was confirmed by simulations of De Jong et al. (1990), who found a 24 ms decrease of $SSRT_{Obs}$ with SOA, even though RT and $SSRT_{in}$ were not correlated.

The assumption of independent processes appears to be in conflict with findings from the refractory-period literature (e.g. Pashler, 1993; Welford, 1952). This literature shows that the response to a stimulus is delayed if that stimulus follows shortly after the start of another stimulus, as if there is a bottleneck mechanism that can only handle one process at a time. Such a bottleneck mechanism would lead to a negative correlation of SOA and $SSRT_{in}$. An important difference between the stop-signal paradigm and other dual-task experiments, however, is that the second stimulus in dual-task experiments usually requires a response, whereas a stop-signal does not.

Logan and Burkell (1986) addressed this issue explicitly by comparing (a) a change task, in which the stop-signal carried the dual instruction to stop the first response, and execute an alternative response, with (b) a dual task, in which the second stimulus called for a second response but not for inhibition of the first response. Logan and Burkell showed that the refractory effect that was found on the dual task was also found on the change task if the original response could not be stopped, but not if inhibition had been successful. They concluded from these data that the bottleneck mechanism is bypassed by the inhibitory process, whereas it is used by a process involved in response execution. This conclusion contrasts with that of Pashler (1993), who held the stimulus-response translation accountable for the refractory effects, although it could be argued that stimulus-response translation and response execution are closely linked (Logan, 1994).

The opposite dependence between two competing tasks could also take place. For example, P. D. Jennings, Schell, Filion, and Dawson (1996) argue that during an increased cognitive load, access to the perceptual channels is suppressed. This would imply that detection of the stop-signal would be easier at the time when perception of the go-stimulus is going on (SOA is short) than during the central activity of selecting the correct response (SOA is long), which boils down to a positive correlation between SOA and $SSRT_{in}$. Although such a dependence would not be consistent with the observed decrease of $SSRT_{Obs}$ with SOA, a positive correlation can also lead to a decrease, provided that the influence of $SSRT_{in}$ variability is stronger than the influence of dependence.

Stochastic independence

A negative correlation of $SSRT_{in}$ and RT could occur theoretically if the go- and stop-processes suffer from limited resources or structural limitations. For example, Roberts, Hager and Heron (1994) used a combination of an anti-saccade task and a competing task that varied in mental load. In the antisaccade task, subjects were instructed to make a saccade away from a visual stimulus onset. An antisaccade is thought to require active inhibitory control of the ocular muscles, because the natural tendency is to make a saccade in the direction of stimulation. During the antisaccade, subjects either received no additional mental load, or performed on a number shadowing task or an arithmetic task. The efficiency on the antisaccade task was quantified by the proportion of trials where a reflexive saccade was given. Performance on the antisaccade task was lower if the competing task exerted a higher mental load, whereas performance on a prosaccade task was not affected by mental load. Roberts et al. concluded that the numerical tasks consumed working memory resources that were also required for inhibition of prosaccadic reflexes on the antisaccade task.

Other literature emphasizes that resource limitations only occur if the competing tasks tap the same resources, whereas tasks without resource overlap will always be perfectly time-shared (Wickens, 1984). The concept of one undifferentiated pool of resources is in conflict with some data that indicate perfect time-sharing between unrelated tasks. For example, Wickens (1976) found no interference from an auditory signal detection task on a response-based force-generation task. Although the similarity between the stop-signal paradigm Wickens' task is striking, the lack of interference from a secondary auditory signal onto a primary response task can not be generalized to the stop-signal paradigm.

In contrast, a positive correlation of RT and $SSRT_{in}$ could occur because energetic resources are variable in quantity (e.g. Sanders, 1983) due to time on task, sleep state and motivational factors. For example, if sustained attention is decreasing, both the go- and stop-process are likely to be impaired. Thus, there are also both theoretical reasons to expect a positive correlation of RT and $SSRT_{in}$ and reasons to expect a negative correlation.

Data that challenge the independence assumptions

The $SSRT_{Obs}$ has been shown to be robust against several factors that affect the speed on the primary task (Logan, 1981; Osman et al., 1986; 1990). Yet, there are two findings that suggest interdependence between the stop- and go-process. Kramer et al. (1994) compared stopping in two conditions of the Eriksen paradigm (B. A. Eriksen & C. W. Eriksen, 1974). In the Eriksen paradigm, a response needs to be based on a target stimulus element, which is flanked by elements that are associated with the opposite or same response. Conflicting flanker information causes a delay of RT, whereas redundant flankers cause a gain in RT. It is thought that flanker information activates response bias, that needs to be suppressed if the target indicates a different response (cf. Kornblum, Hasbroucq, & Osman, 1990). Kramer et al. found that responses to conflict trials could not be stopped as fast as responses to trials without conflict. They interpreted this finding as an interaction between stop-signal inhibition and the inhibition of incorrect response bias, which is part of the primary process.

A second primary-task factor that affected RT more than the inhibition function – which implies an effect on $SSRT_{Obs}$ – was repetition of stimulus-response pairs (Osman et al., 1986). Although several interpretations are possible for this interaction between the primary and inhibitory process, one of them is consistent with that of Kramer et al. (1994), i.e. that previous response activation primes the preparation for subsequent responses, and that suppression of incorrect priming interacts with stop-signal inhibition.

Although it seems like the effects of Eriksen congruence and stimulus-response repetition on $SSRT_{Obs}$ are violations of the independence assumption, there is an alternative interpretation in terms of the locus of inhibition. Several studies have addressed the question whether responses can be stopped throughout the whole responding process, or whether alternatively, part of the response process escapes this control and continues ballistically (Bartlett, 1958; De Jong et al. 1990; Logan, 1981; Osman et al., 1986, 1990). Under the assumption that there is no ballistic phase, the finishing time of the stop-process can be estimated with reaction times from the distribution of go-RT. However, if the locus of inhibition is not the moment of response execution, the $SSRT_{Obs}$ is an overestimation of $SSRT_{in}$, and effects on the duration of ballistic go-processes result in an artificial effect on $SSRT_{Obs}$. The effects of Eriksen-flanker conflict (Kramer et al., 1994) and response repetition (Osman et al., 1986) on $SSRT_{Obs}$ can thus be explained as an interaction between stopping and a go-process, but also as effects on the ballistic phase of the go-process. Both Eriksen congruence and response repetition effects are known to have a locus close to response execution (Kornblum et al., 1990; Sanders, 1990).

This interpretation is balanced by findings that do not support the existence of a large ballistic phase in the go-process. The manipulation of stimulus-response compatibility (Logan, 1981) and response complexity (Osman et al., 1990) for the go-process did not affect $SSRT_{Obs}$. As these manipulations are both interpreted as effects on response selection (Sanders, 1990), the locus of inhibition needs to lie close to the moment of response execution. This idea was supported by the finding of De Jong et al. (1990), that it was possible to stop responses for which muscle tension had already started to rise. Thus, it has been concluded that if there is any ballistic phase in responding, it is only a short phase, close to the point of execution (De Jong, et al., 1990; Logan, 1994; Osman et al., 1986; 1990).

De Jong et al. (1990) and Jennings et al. (1992) evaluated the plausibility of the independence assumption on the basis of the similarity between observed and predicted signal RT. Usually, the speed of signal responses is slightly higher than would be predicted from the race model, but this difference can be explained with variability in stop-speed (De Jong et al.). It was concluded on the basis of this test that there is no large violation of the context independence assumption.

In summary, the important assumption of independence between the stop- and go-processes is challenged by findings in the literature about resource limitations and dual-task interference, and about primary task manipulations that affected $SSRT_{Obs}$. These studies suggest that either the independence assumption is violated, or there is a phase in the go-process that escapes inhibitory control. Under both interpretations, calculations with the horse-race model would be biased.

Because most calculations with the horse-race model are based on the independence assumption, the simulations explore the influence of time-dependence and stochastic dependence on several measures. These two forms are implemented as a correlation between SOA and $SSRT_{in}$, and between RT and $SSRT_{in}$.

Indices of differences in inhibitory efficiency

Recent studies often compare stop-signal data between task conditions (e.g., De Jong, Coles, & Logan, 1995; Kramer et al., 1994), drug conditions (Tannock, Schachar, & Logan, 1994) or subject populations (Kramer et al., 1994; Schachar, Tannock, & Logan, 1993). For example, Logan et al. (in press) showed that impulsive subjects had longer $SSRT_{Obs}$'s than nonimpulsive subjects in a population of young adult students. Likewise, Kramer et al. (1994) showed that the $SSRT_{Obs}$ for older adults is increased relative to young adults, suggesting that the efficiency of inhibition decreases with age.

In some cases, dissociations in $SSRT_{Obs}$ are not the only difference in performance, and other measures need to be calculated to test whether the locus or loci of differences lies in the inhibitory mechanism. For example, Schachar and co-workers (Schachar & Logan, 1990b; Schachar et al., 1993; Schachar, Tannock, Marriott, & Logan, in press) were not satisfied by the observation that ADHD children were slower stoppers. They observed a large difference in the slope of the inhibition function and explored it to shed light on other characteristics of the inhibitory process than speed. By compensating for RT and SOA differences, the slopes of the inhibition functions could not yet be aligned between the groups (see Logan, 1994; Logan & Cowan, 1984). Because not even corrections for individual differences in $SSRT_{Obs}$ and variance of go-RT could eliminate the pattern that the inhibition function was flatter for ADHD subjects, Schachar and Logan (1990b) hypothesized that ADHD subjects differed from normal controls in other aspects of inhibitory control than what was compensated for. For example, ADHD subjects could be characterized by a higher variability of the inhibitory process or by an occasional failure to trigger the inhibition process. This logic for the interpretation of the ZRFT-slope (see Equation 4), has been used in several comparisons of clinical groups (e.g. Oosterlaan & Sergeant, 1995; Schachar & Logan, 1990b; Schachar et al., 1993; Schachar et al., in press; Tannock, Schachar, Carr, Chajczyk, & Logan, 1989; Tannock et al., in press).

However, groups may also differ in ways that have no relevance to stopping, or a clinical group may display dependence between the go- and stop-process while a control group does not. The implications of such differences for the inhibition function are not known. Thus, understanding inhibitory deficits requires a deeper and more detailed analysis, and it is necessary to rule out the contribution of irrelevant performance factors on the indices of inhibitory efficiency. The simulations in this paper can contribute to the insight in the locus of inhibitory deficiencies because they show what the observed pattern of data is as a function of go- and stop-process parameter

manipulations. With the simulations in Study 1, we show how sensitive the ZRFT-slope is to these two factors, and whether factor that are not related to inhibition also contribute to the ZRFT-slope.

Statistical power in the stop-signal paradigm

The horse-race model has contributed to the development of a large set of tools, but these tools are based on mathematical derivations from ideal data. In empirical situations, there are several possible threads to the reliability of observations, and the validity of the race model. The following simulation studies address the relationship between the refinement of inhibitory measures such as the ZRFT-slope, heterogeneity of subject groups and the reliability and power of statistics.

Similarity of scores

Calculations with the horse-race model yield an estimation of the speed, probability and variability of stopping based on a combination of observations. In many studies about normal stopping, task factors are manipulated in a within-subject design with normal young adults. As a result, the go-RT distribution is likely to be comparable between conditions, the speed of stopping is not subject to individual differences and the group is likely to be homogeneous. Therefore, the power of statistics is high.

For example, Osman et al. (1986) showed that stimulus-response repetition had a 58 ms effect on go-RT, versus a 34 ms effect on the inhibition function. The difference of 24 ms reached $p = .028$ significance with only six subjects. In contrast, Schachar and Logan (1990b) made a comparison between children and adults. They observed that the $SSRT_{Obs}$ decreased with age, but this decrease did not reach significance. The latter finding was based on four groups of 12 subjects, whose $SSRT_{Av}$'s were 326, 276, 253, and 264 ms. The contrast between the power of this study and Osman et al.'s illustrates the implications of design and heterogeneity for the power of statistics. This remark applies to other paradigms as well, but has particular relevance for the more complicated calculations that are used in the stop-signal paradigm.

Summation of error

The estimation of $SSRT_{in}$ is based on several measures; each with their own sources of error, and by combining these measures, the error variance of $SSRT_{Obs}$ increases. An even stronger argument can be made for the summation of error in derived measures such as the ZRFT-slope.

If the slope of RR as a function of ZRFT score is compared across conditions, the summation of error can become a problem for the power of statistics, especially with groups of subjects that are relatively heterogeneous. The ZRFT is a transformation of SOA that takes individual differences of mean $SSRT$, and mean and standard deviation (SD) of RT into account. As the $SSRT_{Obs}$ itself is based on RR and a percentile score of RT, the ZRFT has a total of four sources of variance. Finally, estimation of the slope of the ZRFT-corrected inhibition function reduces the data and adds RR as a fifth source of error. If the individual error contributions would be independent and equal, the slope would be measured with at least $(5)^{1/2}$ times as much error variance as a measure that is directly observable. In the simulations, the issue of refinement of calculations and the summation of error is addressed by calculating the confidence interval of the indices of inhibition (Study 2), and the sample size that is required to prove differences (Study 3).

Violations of the horse-race model assumptions

Logan and Cowan (1984) showed that the speed of stopping can be derived with a version of the horse-race model in which the speed of stopping is variable, provided that only the average speed of stopping is calculated. The influence of the independence assumption on estimations of the speed of stopping has been addressed by De Jong et al. (1990). They showed that the observed $SSRT$ was influenced by the correlation between $SSRT_{in}$ and RT, with larger disturbance away from the mean than around the mean of the inhibition function. Furthermore, increases in $SSRT_{in}$ enhanced the disturbance from the correlation between $SSRT_{in}$ and RT. De Jong et al. did not

calculate the consequences of violating the independence assumption for measures such as the ZRFT-slope.

Although a number of studies suggest that SSRT and RT are not related, and that the variability of SSRT is limited (e.g. De Jong et al., 1990), these assumptions are only validated for normal adults. It should be kept in mind that stop-performance is often compared between groups that have multiple performance differences. For example, in comparison with adults, young children respond with a higher variability (Hale, Fry, & Jessie, 1993), and are thought to have less mental resources available (Bjorklund & Harnishfeger, 1992). Moreover, children and subjects with ADHD suffer from more dual-task costs (Guttentag, 1989), and they either have greater refractory effects or have difficulty shifting capacity between competing tasks (Schachar & Logan, 1990a). These considerations can have implications for the assumption of independence, and the variability of $SSRT_{in}$.

The power of comparisons between inhibition scores depends on sample size, effect size, and population variance. Study 1 assesses the effect size of performance factors that are relevant or irrelevant to inhibitory efficiency, and the effect of violations of the race-model's assumptions on inhibition measures. In Study 2, it is calculated how accurate the estimation of inhibition scores is, so that a sufficient number of trials is run for demonstrating effects. Finally, different effects can be compared to each other, and to the outcome of Study 3, in which the sample size for a given power is calculated as a function of effect size and population variance.

Scope of the article

The preceding sections have listed four issues that adhere to the stop-signal methodology as well as to the validity of the horse-race model. We want to argue that the usefulness of dependent variables is restricted by the power and reliability of measurements. This reliability can be compromised on four levels. First, some differences in inhibitory efficiency may be relevant, but can not be observed. For example, a failure to trigger the inhibition process is hypothesized, but can not be observed, let alone be quantified. Simulations can be useful in this regard because they display the observable effects of a number of performance variables. A second issue is that the same pattern of effects that is caused by one parameter of inhibition, may also be caused by another parameter. For example, Schachar and Logan (1990b) were not able to conclude from their data whether ADHD subjects had a more variable $SSRT_{in}$, an occasional failure to trigger the inhibitory mechanism, or yet another difference relative to normal children. Third, some dependent variables are based on several other measurements and can thus accumulate measurement error, so that differences between conditions or groups are hard to prove. When subtle differences in trial-by-trial variability are assessed, the effects can be overruled by the accumulation of noise, stemming from irrelevant performance differences, error variance and violations of horse-race assumptions. Consequently, the signal-to-noise ratio is relatively small for such higher order measurements. Finally, some indices of inhibitory efficiency require more measurements than others. But especially for patient groups such as children with ADHD, there are limitations to the number of trials that can be presented before a subject shows signs of fatigue or inattentiveness. In the following simulations, the reliability of measuring a number of inhibition indices is compared between two types of SOA manipulation and with four ways to calculate $SSRT_{Obs}$. In addition the number of trials and subjects that are needed to reach a reasonable power level is summarized.

In the first study, the behavior of the horse-race model is tested under manipulation of eight parameters, in order to get hold of the observable effects of these variables. The result is a frame of reference for researchers who want to find out more about the possible causes of their data pattern. In the second study, the accuracy of measurements is discussed. In the third study, the relationship

between power, sample sizes and effect sizes is covered, through a choice of parameters that are analogous to those of stop-signal experiments in the literature.

Relation to previous work on the horse-race model

Although a formal mathematical description of the horse-race model and the indices of inhibitory control was already provided by Logan and Cowan (1984), a simulation of the performance on the stop-signal paradigm can make an important contribution to the design and interpretation of future stop-signal studies (cf. De Jong et al., 1990). In a number of respects, a simulation approach to the behavior of the horse-race model has advantages over an analytic approach. First of all, real numbers speak a clear language for researchers who have to decide between one form of testing and another form. Limitations to the financial resources and time that can be invested in an experiment oblige researchers to make a balanced decision about the number of subjects, the number of trials and the design of the experiment. This article provides numerical guidelines to make such decisions, whereas an analytic approach would be harder to translate to such guidelines. Second, covert characteristics of the inhibitory process may have multiple effects on the dependent variables of the stop-signal paradigm. For example, a given factor may have a small effect on $SSRT_{Obs}$, but a large effect on the slope of the inhibition function. This article enables researchers to recognize the similarity between their own data and the overall pattern of simulated effects of covert variables, whereas a mathematical analysis does not provide such a qualitative impression. Third, it is important to take the size of error and heterogeneity into account. Formally, influences such as subject variability can all be addressed analytically. However, as more parameters are involved, an analytic approach of the behavior of the race model becomes increasingly complicated, whereas Monte Carlo studies can provide a quick impression of the influence of all such parameters. The simulations illustrate how observed effect sizes relate to the power of statistics under different levels of performance variability.

The present simulations overlap with those of De Jong et al. (1990, appendix). Those simulations gave a concise overview of the effects of $SSRT_{in}$ variability and correlation between RT and $SSRT_{in}$. Thus, they addressed two important assumptions of the horse-race model, i.e. the simplifying but dispensable assumption of a constant speed of stopping, and the essential assumption that the speeds of the two processes are not related (see Logan & Cowan, 1984). De Jong et al. showed that especially the combination of violations could seriously affect $SSRT_{Obs}$. The correlation that they used was rather strong, however (-.5 and +.5), so it is possible that milder correlations do not cause problems for calculations.

Of course, an appendix can not cover the whole range of parameter effects and dependent variables. The following simulations are improvements relative to the De Jong et al. (1990) simulations. First of all, because the present simulations describe the effects of more parameters at more levels, on the behavior of the horse-race model. A second improvement is that this paper covers all dependent measures that are common in the literature about group differences, with two commonly used methods for the manipulation of SOA. The calculations of the relation between the power of statistics and the number of measurements are a third improvement, especially because they incorporate the influence of group heterogeneity.

GENERAL METHOD

Performance on the stop-signal paradigm was simulated on the basis of the horse-race model with randomization and regression procedures derived from Press, Flannery, Teukolsky, and Vetterling (1986). Under some parameter values, the model did not conform with the horse-race model as described by Logan and co-workers (Logan, 1994; Logan & Cowan, 1984), because the effects of violations of its assumptions were tested. The first study describes the behavior of the

horse-race model after 500,000 nonsignal trials, and 250,000 stop-trials for each of five SOAs. The second study deals with technical questions about the accuracy of measurements as a function of the number of trials, and the third study illustrates how the number of subjects that need to be tested depends on effect sizes and the choice of the dependent measure. These questions are approached with parameter values that are known from the literature or are estimated to be normal for an RT experiment. The size of the effect will be related to the power of a t -test.

Input parameters

Several control variables were used as input for assessing main effects in horse-race behavior. Go-RT and $SSRT_{in}$ were picked randomly from a normal (Gaussian) or an ex-Gaussian distribution. An ex-Gaussian distribution is a convolution of a Gaussian and an exponential distribution. Ex-Gaussian distributions are known to give a good fit of empirical choice-RT data (Ratcliff, 1979). In order to draw latencies from an (ex-)Gaussian distribution, we used the parameters μ (mean of the Gaussian component), σ (*SD* of the Gaussian component) and τ (the time constant of the exponential component, which is the mean as well as the *SD*). Throughout this paper $\text{var}(\mu, \sigma, \tau)$ refers to a variable var with a mean of $\mu + \tau$ ms and a variance of $\sigma^2 + \tau^2$ ms².

Speed parameters

The first variable was the go-RT; the reaction time of the primary process. Across simulations, the mean RT was held constant at 500 ms, as it is reasoned that the behavior of the horse-race model will not vary with mean RT as long as SOA is varied to the same extent (e.g. Logan, 1981; Logan & Cowan, 1984; Logan et al., 1984). The default values for RT were RT(440, 80, 60) so that mean and *SD* were 500 and 100 ms. Second, $SSRT_{in}$ followed an ex-Gaussian distribution with default values $SSRT_{in}(230, 46, 20)$, resulting in a mean of 250 ms and a *SD* of 50 ms. In Study 3, a population *SD* of 25 or 50 ms is added for $SSRT_{in}$, and a population *SD* of 50 or 100 ms is added for RT, both deviations were drawn from a normal distribution. On exceptional trials, the RT or $SSRT_{in}$ that was drawn with this method could be implausibly low or negative. We chose to redraw these variables only if they were negative, because a more conservative selection of input parameters would affect their distributions, and would be more arbitrary.

The selection of default values for processing speed is based on previous research with the stop-signal paradigm. As noted before, the mean RT is an arbitrary value, and different values would not affect the outcome. Furthermore, previous research (see Logan, 1994 for a review) suggests the stop-process is independent of the type of primary task. SD_{RT} decreased from 240 ms for 7-year-old children to 109 ms for adults in one study (Schachar & Logan, 1990b), and from 245 ms for 6-year-old children to 58 ms for adults in another study (Band, Van der Molen, Overtom, & Verbaten, see Chapter 2). The population *SD*s of RT in those studies decreased from 100 to 59 ms and from 158 to 30 ms respectively and in a different study it increased from 70 to 90 ms from young to older adults (Kramer et al., 1994). $SSRT_{Obs}$'s are between 200 and 300 ms for most age groups and can be as high as 437 ms for children with ADHD (Schachar & Logan, 1990b). The individual *SD* of $SSRT$ is not known, but the speed of response inhibition is often compared with a simple RT, for which 50 ms is a common *SD* (cf. De Jong, et al., 1990; Luce, 1986). The population *SD* of $SSRT_{Obs}$ was between 28 ms (De Jong et al., 1990) and 72 ms (Kramer et al., 1994) for young adults, and amounted to 112 ms for older adults (Kramer et al., 1994) and 183 ms for ADHD-subjects (Schachar & Logan, 1990b).

Other variables

The inhibition process could be triggered on all (1.0 = default value), or only a proportion of trials. It is noted by Tannock et al. (1994) that subjects with the ADHD-syndrome may differ from a control group with the same age by an occasional failure to trigger the inhibitory process. Because there was no clue in the literature about the size of a failure to inhibit, we chose to simulate with a minimum triggering rate of .75.

The effect of interdependence between the two processes that compete on the horse race was simulated with a variable correlation between RT and $SSRT_{in}$. Correlations between random scores were created by combining one shared source of covariance with two unique sources of variances for RT and $SSRT_{in}$. This procedure of creating a correlation can only be applied when the convolution of shared and unique contributions of variance belongs to the same family of distributions as the constituent parts. Whereas a convolution of two normal distributions follows a

normal distribution, a convolution of exponential distributions results in a gamma distribution. This was reason for us to restrict the assessment of the effect of correlations between variables to Gaussian distributions. It will be shown below that the simulations of the effect of skewness in $SSRT_{in}$ or RT only had small effects, which holds that the effect of dependence is not likely to be different with Gaussian vs. ex-Gaussian distributions. A positive correlation describes a generalized effect on processing speed, for example due to a variable arousal level (e.g. Sanders, 1983). On the other hand, a negative correlation models the effect of limited resources, in which the speed of one process hampers the speed of the other process. By default, the correlation of go-RT and $SSRT_{in}$ was zero.

$SSRT_{Obs}$ is usually reported to be faster on late SOAs than on early SOAs. Like Logan and Burkell noted, the negative relationship between SOA and $SSRT_{Obs}$ could be consistent with the horse-race model, as a result of variability in the $SSRT_{in}$. However, this pattern is also similar to the refractory effect that occurs if a subject has to respond to two stimuli in short sequence (e.g. Logan & Burkell, 1986; Pashler, 1993). It was shown in the above that there are theoretical reasons why a correlation between SOA and $SSRT_{in}$ would be expected. In order to show what pattern of data would be consistent with a positive or negative correlation, we simulated a correlation of SOA and $SSRT_{in}$.¹⁵ The default value of this correlation was zero.

The results with the fixed-SOA procedure are set off to the RR-tracking procedure. In order to avoid redundancy and to enhance the readability of this paper, results of the RT-tracking procedure are not reported. Simulations in which SOAs were selected relative to the mean RT of the previous 40 nonsignal trials yielded results that were comparable to the results of the fixed-SOA procedure. Although there is not always a difference in findings with fixed-SOA vs. RR-tracking, we do report the RR-tracking procedure, because with that procedure, input parameters can have a substantial effect on the changes to SOAs and their corresponding response rates.

Table 1: SOA procedures: The starting values (ms) for the fixed-SOA and RR-tracking procedure, and adjustment rules (ms) for the RR-tracking procedure, and the asymptotic rate of signal responses that would occur with default parameters.

SOA	Starting	After commission	After inhibition	Response Rate
A	150	-12	+3	.20
B	200	-10	+5	.33
C	250	-7	+7	.50
D	300	-5	+10	.67
E	350	-3	+12	.80

In the fixed-SOA procedure, five SOAs were fixed at the starting values. The RR-tracking procedure in this study held that for every one of five SOA levels, SOAs were adjusted after every presentation, so that they became shorter if the previous trial had evoked a signal response or became longer if the previous response was correctly inhibited. A more commonly used form of RR-tracking is to use constant step sizes for rises and decreases of SOA, but to make an adjustment in one direction more often than in the other direction. For example, if the SOA rises upon every correct inhibition and decreases upon every second signal response, the asymptotic RR is 71%. In practice, that procedure is easier to implement under limitations of computer clock ticks and has the

¹⁵As SOA was not randomly chosen, this correlation was established by letting the deviation in SOA determine a proportion of the deviation in $SSRT_{in}$.

additional advantage that it yields a restricted number of absolute SOA values. The specifications of the RR-tracking procedure are listed in Table 1.¹⁶

The basic principle for the implementation of the model is that if $(SOA_k + SSRT_k) < RT_k$ on trial k , the response is correctly withheld, whereas a response slips through when $(SOA_k + SSRT_k) > RT_k$. Furthermore, a failure of triggering the inhibitory process is implemented by letting the race be won by the primary task on a subset of signal trials, regardless of SOA_k , $SSRT_k$ and RT_k . In other words, on trials with a failure of the stop-process, the race is not considered, and the outcome of that trial is a signal response with an RT_k that is drawn from the nonsignal RT distribution.

Note that we did not manipulate the existence of a ballistic process. The effect of a ballistic process could be approached by assuming that the finish of the horse race does not lie at the moment of response, but Bal ms earlier, so that a response is only withheld if $(SOA_k + SSRT_k) < (RT_k - Bal_k)$. However, there is a number of reasons why simulating this would not be informative here. Several experiments have shown that the duration of a ballistic process, if it exists at all, is negligible. Furthermore, the pattern of results would not be strongly affected by the presence of a ballistic process. The term $(RT_k - Bal_k)$ could be replaced by RT_k minus a constant C , unless (a) Bal_k were related to other parameters of the race model or (b) Bal_k were highly variable. Theoretically, dependence between the duration of the ballistic process and another process is not very plausible, because it is an autonomous process. Furthermore, ballistic processes are thought to be rather rigid in their mode of execution, because they are what Schneider, Dumais, and Shiffrin (1984) would classify as automatic processes, which are not under conscious control. Therefore, the variance would be negligible compared with RT, and the ballistic process would not consume a large amount of mental resources. In conclusion, the effect of the presence of a ballistic process is too small to be of interest within the scope of this paper.

Dependent variables

In the following simulations, performance of the horse-race model is analyzed with the dependent variables that are used in the literature on the stop-signal paradigm. Some of the dependent variables are measured for every SOA, which would allow a multivariate approach. However, for the ease of exposition, all the power calculations are based on t -tests for the comparison of pairs.

The first dependent variable concerns the $SSRT_{Obs}$. For each SOA, the $SSRT_{Obs}$ can be calculated as the difference between SOA and the corresponding percentile score of the nonsignal-RT distribution. For example, if a given SOA evokes a RR of .60, the proper calculation is $SSRT_{Obs} = 60\text{th percentile RT} - \text{the corresponding SOA}$.

It is repeatedly observed that the $SSRT_{Obs}$ is different for every SOA, which can be the result of variability in $SSRT_{in}$. If variability of $SSRT_{in}$ is suspected, the speed of stopping can be expressed as one $SSRT_{Obs}$ across SOAs, that serves as a summary measure (e.g. Logan, 1994; Logan & Cowan, 1984). The first measure that we evaluated as a summary SSRT, was the average of those of the five $SSRT_{Obs}$'s for which $.15 < RR < .85$. The second measure (Logan & Cowan, 1984) is the distance between mean RT and the mean of the inhibition function ($SSRT_{Dist}$). To calculate this, one step is to subtract the response rate at SOA $(n - 1)$ from SOA (n) . This can not be done with the first SOA. In order to avoid that an asymmetric section of the inhibition function would be used, the fifth SOA was also rejected for the calculation of the $SSRT_{Dist}$. Thus, the $SSRT_{Dist}$ was calculated on the basis of SOA 2, 3, and 4 with the equation

¹⁶Because SOAs can change during the simulation, different SOA procedures cause unequal ranges of SOAs. The correlation was established on the basis of the standard deviation of the five initial SOAs, because the effect of tracking on the range of SOA could not be known in advance.

$$SSRT_{Dist} = \frac{\{[(RR_2 - RR_1) \cdot SOA_2] + [(RR_3 - RR_2) \cdot SOA_3] + [(RR_4 - RR_3) \cdot SOA_4]\}}{(RR_4 - RR_1)} \quad (5)$$

The distance SSRT ($SSRT_{Dist}$) is based on the same principle as the distance between the median RT and the central SOA ($SSRT_{Med}$). The $SSRT_{Dist}$ and $SSRT_{Med}$ procedures should lead to identical estimates if the go- and $SSRT_{in}$ distributions are symmetrical, because then the median is equal to the mean. The fourth summary measure is derived from the Colonius (1990) procedure. The cumulative distribution of $SSRT_{Obs}$ was calculated with Equation 1 for intervals of 10 ms between 0 and 800 ms. To remove spurious data points and create a monotonically rising cumulative function, the data were smoothed. Distribution functions were smoothed by averaging nonmonotonic values on a 10 ms interval with values of adjacent intervals, and density functions were smoothed by averaging large local probability differences with adjacent values. Smoothing continued until the function was monotonic or smooth, but there was a maximum of ten iterations, because more iterations would flatten the functions. The median $SSRT_{Col}$ was calculated with this method for every SOA, for each of the five $SSRT_{Obs}$'s. Theoretically the five measures should be equal if there is no relation between SOA and $SSRT_{in}$. The mean of these five values is reported as $SSRT_{Col50}$.

For the estimation of variability of $SSRT_{in}$, two measures were evaluated. The Colonius method yields an estimate of the $SSRT_{in}$ distribution. For every SOA, the interquartile distance, i.e. the difference between the 25th ($SSRT_{Col25}$) and 75th ($SSRT_{Col75}$) percentile score was calculated. This measure is less sensitive than the *SD* to the distortion of the estimated distribution at the tails, which is said to occur with the Colonius method (Logan, 1994). In addition, it is not as easy to extract the *SD* from a cumulative distribution as it is to calculate the interquartile distance. The second estimation of variability in $SSRT_{in}$ was calculated with Equation 3 (Logan & Cowan, 1984).

Signal RTs as predicted from the race model were compared with observed signal RT, because this comparison is used in the literature as a test of the independence assumption of the race model (De Jong et al. 1990; Jennings et al., 1992). For example, if a subject responds on 60% of all signal trials, the mean of the 60% fastest nonsignal RTs (predicted RT) is compared with the mean of signal responses (observed RT). This simulation study can reveal what the expected difference between observed and predicted RT is. Just as with $SSRT_{Obs}$, it can be calculated for every SOA.

RR is commonly depicted as a function of SOA, and the slope of the function is used as a window to the inhibitory process. However, the slope of this function depends to a large extent on the variance in go-RT (Logan & Cowan, 1984). As a result, differences between groups in the slope of the inhibition function can not be properly interpreted. In order to detect the locus of differences in stop-performance between groups, it is possible to make stepwise corrections for differences between groups in SOA and in the mean and *SD* of RT. When all these corrections are made, the ZRFT score replaces the SOA in the inhibition function (see Equation 4). Note that in the present simulations only one factor was manipulated at a time. The ZRFT transformation is calculated for all parameter manipulations throughout the paper, even though some parameters in the ZRFT-equation do not need to be compensated for. It is discussed because it is the most complete form of correction. In this way, effects on the ZRFT-slope can be compared between different manipulations.

In this study, the slope of the standard inhibition function, which relates RR to SOA, and the slope of the normalized inhibition function, which relates RR to ZRFT scores are determined through linear regression in the range of $RR = .15 - .85$. In this range, the inhibition function is

almost linear, whereas the sigmoid shape of the inhibition function becomes visible outside those bounds.

STUDY 1: ASYMPTOTIC BEHAVIOR OF THE HORSE-RACE MODEL

In this study, the behavior of the horse-race model is described as a function of parameter values mentioned in the above. Each parameter-setting is simulated with 100 virtual subjects who each performed on 5000 nonsignal trials and, for every SOA, 2500 stop-signal trials. The dependent measures are the means of the results with single parameter sets.

Measuring SSRT with the stop-signal paradigm

An obvious problem for researchers who want to measure $SSRT_{Obs}$ is the selection of SOAs (see section “When to present stop-signals”). For the calculation of several measures of inhibitory control, the range of the inhibition function should cover the SOA where $RR = .5$. As a rule of thumb, the mean RT minus mean of the anticipated $SSRT_{Obs}$ approximates this central SOA. Usually it is easy to estimate mean RT, but incorrect anticipation of the $SSRT_{Obs}$ can cause an experimenter to make a poor selection of SOAs. For information about the slope of the inhibition function, more SOAs can be chosen on both sides. Selection of more SOAs can make the assessment of the inhibitory control more reliable and increases the chance of employing a useful SOA. Unfortunately, however, there is a trade-off between the maximum number of SOAs and the number of stop-trial replications per SOA that can be measured within a given period of testing.

If the range of SOAs includes the central SOA, another problem can be that the range does not always cover both sides of the central SOA to the same extent. When the SOAs are asymmetric around the central SOA, the $SSRT_{AV}$ may be disturbed, as well as the regression-based $SSRT_{Med}$. A solution for the problem with finding a useful SOA and with choosing SOAs that are symmetric around the central SOA, is the RR-tracking procedure for the adjustment of SOAs. However, because this procedure causes the SOA to change from trial to trial, it may be the case that this procedure introduces a different problem; i.e. an increased variability of the finishing time of the stop-process. The following simulations can help to see to what extent a poor choice of SOAs may affect the results, whether the RR-tracking procedure can compensate for a poor choice, and whether the tracking procedure affects estimates in other ways.

In this section the accuracy of measurement is assessed in the situation where the initial SOA is not optimally adjusted to differences in $SSRT_{in}$. Mean $SSRT_{in}$ was varied between 210 and 300 ms in steps of 10 ms.

Results

As expected, the RR increased with SOA and with $SSRT_{in}$. Figure 2 shows clearly that the fixed-SOA procedure measured dissimilar sections of the inhibition function, whereas the RR-tracking procedure arrived at measurements on parallel sections of the inhibition functions for different levels of mean $SSRT_{in}$. The inhibition functions of the RR-tracking procedure were only moved to left or right and the RR values were equal. In addition, the fixed-SOA procedure resulted in a higher number of measurements close to $RR = \text{zero or } 1$, which are less informative and cause a less reliable $SSRT_{Obs}$. It is said (Logan & Cowan, 1984) that the inhibition function follows a sigmoid pattern. This is observable when extreme values of RR are included (such as $RR < .15$ or $> .85$), but for the middle range, the inhibition function hardly deviates from linearity. Thus, if the SOAs would be even less well adjusted to $SSRT_{in}$ than in the present simulations, the fixed-SOA procedure might yield data that are hard to interpret because they stem from a less representative section of the inhibition function, or because data in different conditions stem from different sections. Under the present values of mean $SSRT_{in}$, however, the inhibition function was not affected convincingly (see Table 2). The slope of the normal inhibition function was 3.3 or 3.4 s^{-1}

for all conditions, and was .33 or .34 for all ZRFT-transformed inhibition functions. Apparently, the variability of the finishing time of the stop-process, as caused by the RR-tracking procedure, did not affect the slope of the ZRFT-transformed function.

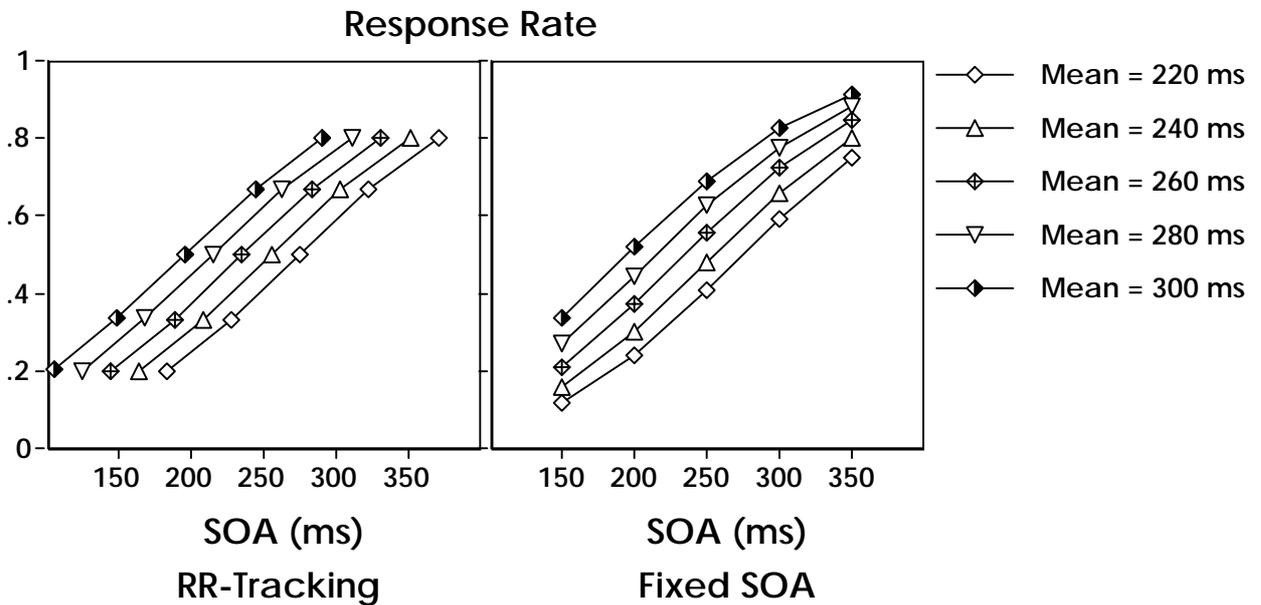


Figure 2. The effect of mean stop-speed and the SOA-procedure on the inhibition function. Note that for the RR-tracking procedure, the functions are parallel, whereas for the fixed-SOA procedure, the height of the function depends on mean stop-speed.

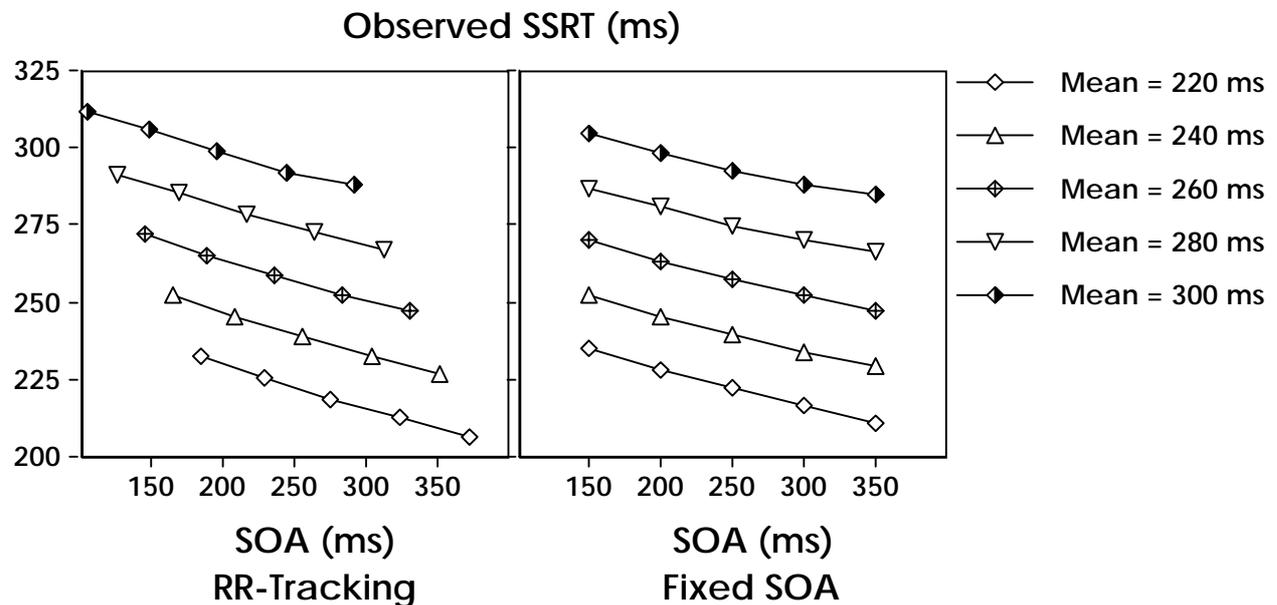


Figure 3. The effect of mean stop-speed and the SOA-procedure on observed stop-speed.

For all levels of mean $SSRT_{in}$, $SSRT_{Obs}$ decreased with SOA (see Figure 3). This pattern is in accordance with the common empirical finding, and can be explained by the fact that for early SOAs even slow $SSRT_{in}$'s lead to successful inhibition. In contrast, for later SOAs, only fast $SSRT_{in}$'s result in correct inhibition (see also, Logan & Burkell, 1986). A more technical explanation of the decrease of $SSRT_{Obs}$ with SOA, is that for early SOAs, the go-process duration intersects the distribution of $SSRT_{in}$'s on average at a later percentile than for later SOAs. The point where the $SSRT_{in}$ distribution is intersected is computed as the $SSRT_{Obs}$.

For the fixed-SOA procedure, there was a bias of $SSRT_{Obs}$ in the direction of 250 ms. As a result, the difference between $SSRT_{in}$ levels was underestimated by approximately 10%. This can be explained with the choice of SOAs: Identical SOAs corresponded to a higher RR for higher levels of mean $SSRT_{in}$, and a higher RR leads to a lower $SSRT_{Obs}$. This bias did not occur for the RR-tracking procedure, because in that situation the RRs, rather than the SOAs, were identical.

Table 2 shows summary measures of $SSRT_{Obs}$. The $SSRT_{Av}$ was within 5 ms accurate for all fixed-SOA conditions, and within 2 ms accurate for all RR-tracking conditions. The $SSRT_{Dist}$ was a reasonably accurate estimate of $SSRT_{in}$ when the RR-tracking procedure, but not when the fixed-SOA procedure was used. Information about the horizontal distance between the inhibition functions seems to be lost in Equation 4, so that the difference between $SSRT_{in}$ levels is not detected. $SSRT_{Med}$ was reliable for both SOA procedures, given that the 50th percentile $SSRT_{in}$ was 2 ms below the mean because of the positively skewed $SSRT_{in}$ distribution. The 50th percentile SSRT is also estimated with the Colonus method. Surprisingly, $SSRT_{Col50}$ largely underestimated $SSRT_{in}$ for both SOA procedures, although the differences between levels of mean $SSRT_{in}$ were reasonably accurate.

Table 2: Observed stop-speed as a function of input stop-speed and SOA procedure

Mean $SSRT_{in}$	$SSRT_{Av}$		$SSRT_{Dist}$		$SSRT_{Med}$		$SSRT_{Col50}$	
	Fix	RR-T	Fix	RR-T	Fix	RR-T	Fix	RR-T
210	210	209	242	211	208	208	191	200
220	220	219	243	221	218	217	200	207
230	228	229	245	230	228	227	208	219
240	240	239	246	240	238	237	217	225
250	249	249	248	250	247	247	228	236
260	258	259	249	260	257	257	240	248
270	269	269	251	270	268	268	248	260
280	278	279	252	280	278	277	257	269
290	287	289	253	290	287	288	267	276
300	296	299	255	300	297	297	277	286

Note: SSRT = stop-signal reaction time in ms. Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

Figure 4 shows the results of the independence test. The observed RT of signal responses was higher than predicted for all conditions. This difference was equal across different conditions of mean $SSRT_{in}$ for comparable SOA levels and was higher for early SOAs. For SOA = 250 ms, the difference amounted to between 4 and 11 ms, and for early SOAs the difference could be as high as 20 ms. These results suggest that there are no large differences between observed and predicted RT in a standard situation. Although it would be too conservative to test the independence assumption with the null hypothesis that observed RT = predicted RT, the absolute size of this difference may still be a valuable tool for the interpretation of data. However, it first needs to be shown that the independence test is robust against other factors that are irrelevant to the correlation of $SSRT_{in}$ and RT.

In conclusion, a poor choice of SOAs does not have large implications for the accuracy of $SSRT_{Obs}$ scores, or for the slope of the inhibition functions. Both measures can be derived with a fixed-SOA procedure as well as a RR-tracking procedure, although the latter gives a better estimate of differences in $SSRT_{in}$ on single-SOA measurements and allows the use of $SSRT_{Dist}$. The absolute size of $SSRT_{in}$ can not be estimated with $SSRT_{Col50}$. Because of the poor achievements, $SSRT_{Dist}$ for the fixed-SOA procedure and $SSRT_{Col50}$ for both procedures will not be evaluated in

subsequent sections. Although absolute values of $SSRT_{Col50}$ were not correct, there is no reason to dispose the Colonius method for estimating the interquartile distance of the $SSRT_{in}$ -distribution.

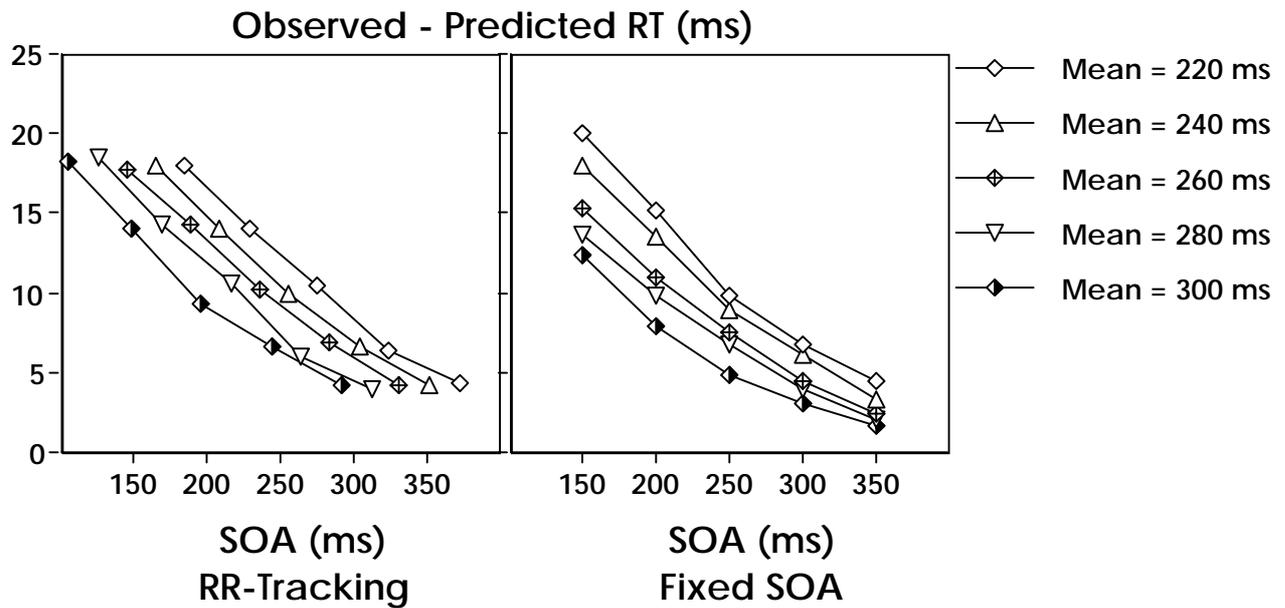


Figure 4. The effect of mean stop-speed and the SOA-procedure on the observed minus predicted reaction time of signal response trials. Note that for the fixed-SOA procedure the difference appears to rise with faster stop-speeds. The results of the RR-tracking procedure show that this is an effect of differences in the adaptation of SOAs to stop-speed.

The influence of a nonconstant $SSRT_{in}$

Logan (1994) noted that the assumption that $SSRT_{in}$ is constant, is bound to be wrong, but that in practice the correctness of the assumption is not very important for the estimation of $SSRT$. Although it is not a necessary assumption, a constant $SSRT_{in}$ comes handy, because it allows the estimation of $SSRT_{Obs}$ for every SOA. Logan and Cowan (1984) calculated the effect of a violation of the assumption of a constant speed of stopping. They concluded that $SSRT_{Obs}$ scores for single SOAs are spurious if $SSRT_{in}$ is variable, but that a summary $SSRT_{Obs}$ score for the whole inhibition function is warranted. Needless to say, the degree of variance determines whether estimating $SSRT_{Obs}$'s on single SOAs is warranted, and simulations can clarify what amount of variability is still acceptable. A first step in this direction was made by De Jong et al. (1990), who investigated the behavior of the horse-race model with two levels of variability in $SSRT_{in}$ (i.e. $SD = 27$ ms and 69 ms).

It is not general practice to quantify the variability of $SSRT$, even though there are methods for it. Logan and Cowan (1984) argued that if, for example, a normal distribution of $SSRT_{in}$ is assumed, the variance can be estimated from the slope of the inhibition function and the variance in RT (Equation 3). The variability of $SSRT$ can also be estimated with the Colonius method (Colonius, 1990), but this requires more, and high quality data.

Besides being interesting for the race model, variability of $SSRT$ has relevance for research on inhibitory deficiencies. It has been concluded in some studies (e.g. Schachar & Logan, 1990b) that the $SSRT_{Av}$ of two groups was comparable, while the variability in $SSRT_{in}$ across trials was larger for one group than for the other. Such a difference between groups is indicative of a less dependable inhibition system. Thus far, however, it is not common practice to estimate the variability of $SSRT_{in}$.

In the next simulations, the effect of variance in $SSRT_{in}$ is tested, and the accuracy of the two methods for estimating variability are evaluated. The standard deviation of $SSRT_{in}$ (SD_{SSRT}), could be 0, 10, 20, 50, 70 or 100 ms, and the proportion of exponential variance was fixed at .16.

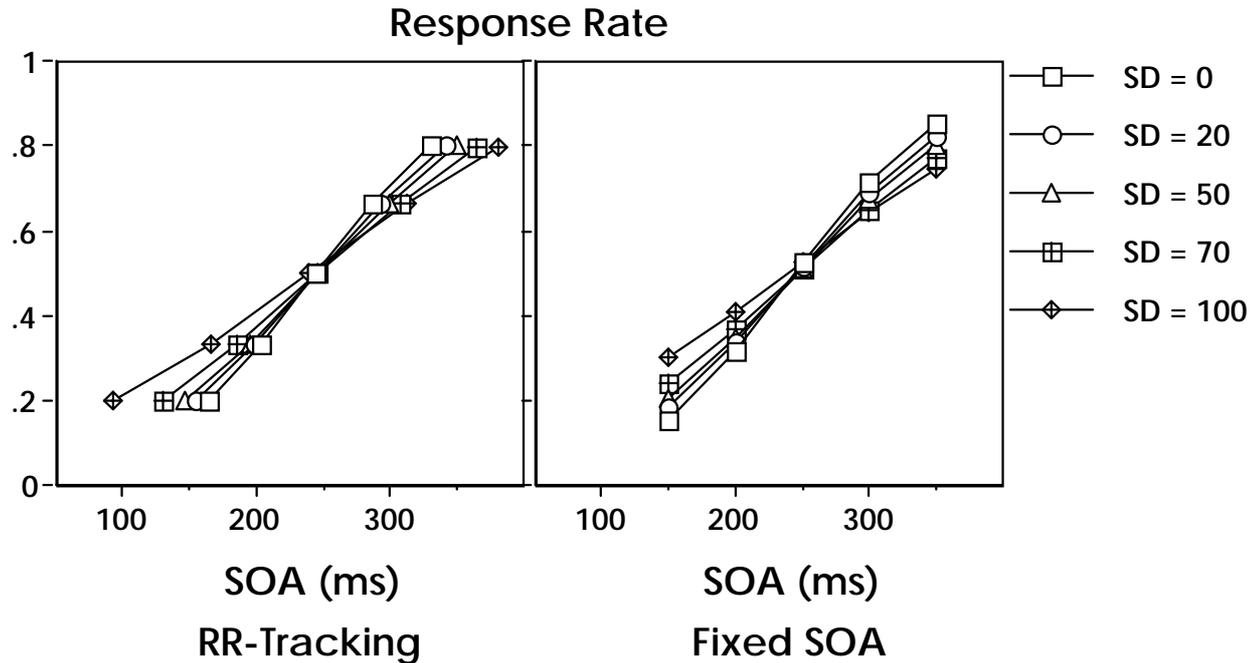


Figure 5. The effect of variance of stop-speed and the SOA-procedure on the inhibition function. An increase of variance in stop-speed causes a shallower slope. Note that the fixed-SOA procedure yields response rates close to zero or one, and that this makes the inhibition function less linear and more sigmoid shaped. This is not the case for the RR-tracking procedure.

Results

Figure 5 displays the inhibition functions for simulations with both SOA-procedures. It can be seen that the RRs at different SOA-levels are quite robust to the variance of $SSRT_{in}$ in the middle range of RR, but not towards the extremes. An increase of variability of $SSRT_{in}$ makes the inhibition function somewhat shallower, and just as was expected, this effect is not removed through ZRFT-transformation, as can be seen from Table 3 (cf. Schachar & Logan, 1990b).

A relatively large effect of $SSRT_{in}$ -variability is observed on $SSRT_{Obs}$ on single SOAs (see Figure 6). In general, with variance of $SSRT_{in}$, early SOAs lead to an overestimation of $SSRT$, whereas late SOAs lead to an underestimation (cf. De Jong et al., 1990). The estimation remains within reasonable bounds (± 13 ms) when $SD_{SSRT} \leq 50$ ms, but in empirical studies there may very well be a higher variability for some subjects. When $SD_{SSRT} = 100$ ms, the over- or underestimation of $SSRT_{in}$ can amount to 38 ms. However, most of the summary $SSRT_{Obs}$ measures did not change with variance in $SSRT_{in}$. Only when SD_{SSRT} was 100 ms, there was a small tendency towards overestimating $SSRT_{in}$.

Table 3: The inhibition function and observed stop-speed as a function of stop-speed variance and SOA procedure

SD_{SSRT}	ZRFT-slope		SOA-slope		$SSRT_{Av}$		$SSRT_{Dist}$	$SSRT_{Med}$		Est. SD_{SSRT}	
	Fix	RR-T	Fix	RR-T	Fix	RR-T	RR-T	Fix	RR-T	Fix	RR-T
0	-0.38	-0.37	3.8	3.7	250	250	252	249	249	36	41
10	-0.36	-0.36	3.6	3.6	250	250	252	248	248	48	46
20	-0.33	-0.33	3.3	3.3	249	249	250	247	248	69	69
50	-0.30	-0.30	3.0	3.0	248	248	249	247	247	85	89
70	-0.27	-0.26	2.7	2.6	247	249	249	247	247	109	117

100 -0.22 -0.21 2.2 2.1 251 258 255 255 256 147 160
 Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure. Est. SD_{SSRT} = the estimate of SD_{SSRT} (ms) based on Equation 3.

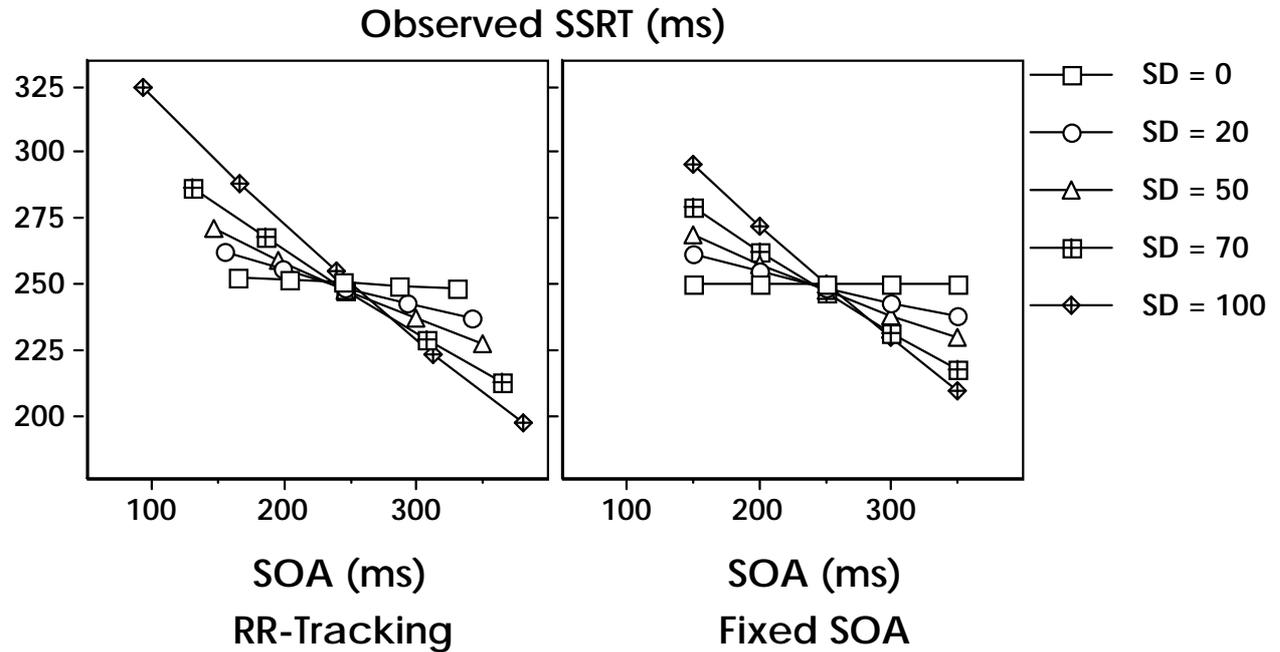


Figure 6. The effect of variance of stop-speed and the SOA-procedure on the observed stop-speed. A lack of variance results in observed stop-speeds that do not change with SOA. Extreme amounts of variance make single-SOA observations of stop-speed unreliable.

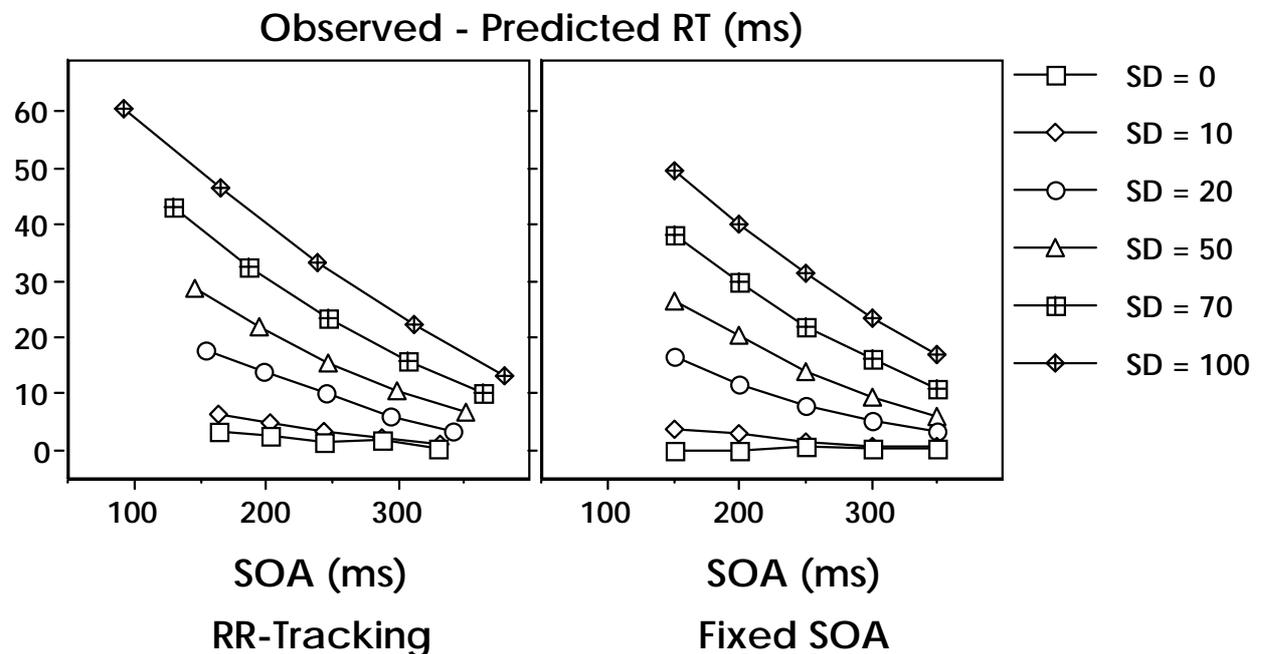


Figure 7. The effect of variance of stop-speed and the SOA-procedure on the observed minus predicted reaction time of signal response trials. The difference depends strongly on variance of stop-speed, and is close to zero if there is no variance.

A higher variability of $SSRT_{in}$ appears to be the major factor that determines the difference between observed and predicted signal RTs (see Figure 7). A SD_{SSRT} close to zero leads to a near-perfect match of signal responses and the predictions from the race model. However, if SD_{SSRT} is larger, it easily doubles the difference that was found under default parameter values. This means

that observed – predicted RT can reach higher values, despite the independence of the go- and stop-process.

The two methods for estimating variability in $SSRT_{in}$ both yielded disappointing results. Figure 8 shows that the Colonius method typically overestimated the distance between the 25th and 75th percentile score of $SSRT_{in}$ by about 10 - 50 ms; most strongly for early SOAs. This is a rather large overestimation if it is considered that the real distance was 0, 30, 69, 92, 132 and 190 ms for the six levels of SD_{SSRT} respectively.

Table 3 shows that Equation 3 also overestimated SD_{SSRT} . It is possible that the variance of the inhibition function can not be interpreted as a summation of SD_{RT}^2 and SD_{SSRT}^2 , because there is an interaction between both sources of variance. As a result, SD_{SSRT} can not be estimated, unless the interaction can be formalized.

Thus, it can be concluded that variability of $SSRT_{in}$ makes single-SOA measurements of $SSRT_{Obs}$ less accurate, but does not affect summary scores of $SSRT_{Obs}$. The SD_{SSRT} can not be estimated very accurately.

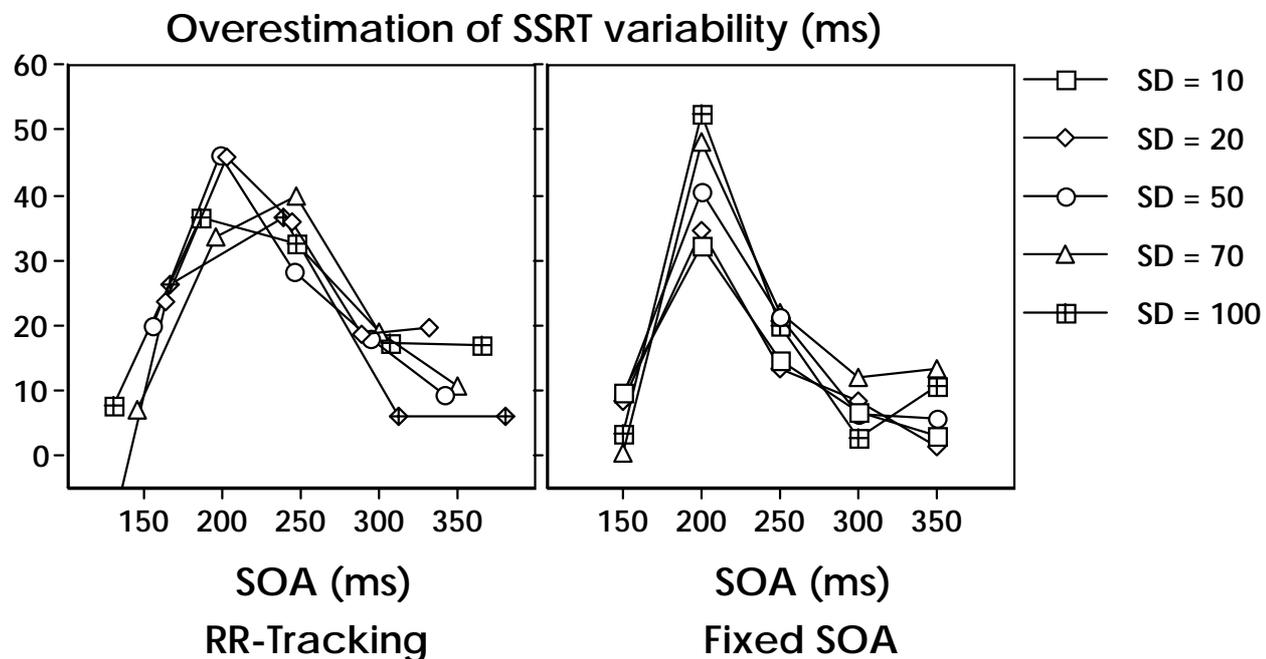


Figure 8. The effect of variance of stop-speed and the SOA-procedure on the measurement of variability of stop-speed. The Colonius (1990) procedure for estimating the difference between the 25th and 75th percentile of the stop-speed distribution is the least reliable for early SOAs, and is more reliable for later SOAs, given a high quality of data.

In the light of the strong dependence of $SSRT_{Obs}$ on the variability of $SSRT_{in}$, the next question is whether the distribution of $SSRT_{in}$ has an additional effect. It is known from the literature (e.g. Luce, 1986) that ex-Gaussian distributions describe simple and choice reaction times quite well, and it is only reasonable to assume that the $SSRT_{in}$ -distribution can also be described by an ex-Gaussian function. Logan and Burkell (1986) tried to explain why $SSRT_{Obs}$ decreased with SOA, and calculated the fit of exponential and Gaussian distributions of $SSRT_{in}$ onto empirical $SSRT_{Obs}$ as a function of SOA (cf. Figures 3 and 6). They found that the predicted decrease of $SSRT_{Obs}$ with SOA was almost identical for both types of distributions, but that the fit to empirical data was not always perfect. Thus, it could not be derived from their study what the distribution of $SSRT_{in}$ was.

A related issue is whether the methodology of the stop-signal paradigm is suited for all types of distributions. The full model of Logan and Cowan (1984) does not assume specific shapes of

either the RT or $SSRT_{in}$ distribution. Similarly, Colonius (1990) argued that his method for deriving SSRTs was distribution-independent.

The influence of skewness in the distribution of $SSRT_{in}$ was investigated with a fixed $SD_{SSRT} = 50$ ms. The proportion of variance that stemmed from the exponential deviation was 0; .04; .16; .36 or .64 because τ was 0, 10, 20, 30 or 40 ms. The skewness is more positive with increasing τ . For these values of τ , skewness of the $SSRT_{in}$ distributions did not have an effect that would be measurable under typical empirical conditions, on $SSRT_{Obs}$, the slope of the inhibition function or the independence test. In short, it is the variance of the $SSRT_{in}$ distribution, rather than the shape, that affects the average outcome of the horse race.

The influence of the go-process distribution

Under the present stop-signal methodology, variability of the nonsignal RTs is important for estimating $SSRT_{Obs}$. If RT were constant and $SSRT_{in}$ were not, there would not be a difference between, for example, the 20th and 80th percentile of the RT-distribution, so that calculations of $SSRT_{Obs}$ would be depending entirely on SOA. Conversely, as the variability of the go-RT increases, the resolution for the estimation of $SSRT_{Obs}$ increases. The methodology of the horse-race model does not make assumptions about the distribution of RT. However, differences between conditions in variability can obscure inhibitory performance. For example, the slope of the inhibition function is sensitive to variability in the go- as well as the stop-process. In order to remove the contribution of SD_{RT} from the inhibition function, Logan and Cowan (1984) have developed the ZRFT transformation. In the following simulations, it is explored in what way inhibition scores are affected by RT variability.

The effect of variability in the go-process was simulated with $SD_{RT} = 25, 50, 100, 150$ or 200 ms, with a fixed proportion of exponential variance = .36.

Table 4: The inhibition function and observed stop-speed (ms) as a primary-speed variance (ms) and SOA procedure

SD_{RT}	ZRFT-slope		SOA-slope		SSRT _{Av}		SSRT _{Dist}	SSRT _{Med}	
	Fix	RR-T	Fix	RR-T	Fix	RR-T	RR-T	Fix	RR-T
25	-0.16	-0.16	6.3	6.3	249	249	248	248	248
50	-0.26	-0.25	5.3	5.0	248	249	249	248	248
100	-0.33	-0.33	3.3	3.3	249	249	250	248	247
150	-0.37	-0.35	2.5	2.3	249	249	252	248	247
200	-0.39	-0.36	1.9	1.8	248	247	254	244	245

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

Results

Table 4 shows that the slope of the inhibition function depends to a large extent on the variability in RT. This is not surprising, given that under the assumption of a constant $SSRT_{in}$, the slope of the inhibition function is equal to the slope of the cumulative distribution function of RT, which is steeper when the SD is smaller. It is surprising to see, however, that the ZRFT-transformation, which is designed to compensate for the influence of noninhibitory processes, such as the mean and SD of the RT distribution, does not remove the slope differences in the inhibition function. The size of this effect is substantial, given that the ZRFT transformation is usually performed to shed light on differences in $SSRT_{in}$ -variability, by removing the contribution of RT variability. Table 4 shows that the effect of $SD_{RT} = 150$ vs. 50 ms can influence the ZRFT-transformed inhibition function just as much as the effect of $SD_{SSRT} = 0$ vs. 70 ms, but in opposite directions. The ZRFT-transformed inhibition function becomes steeper if SD_{SSRT} is lower or SD_{RT}

is higher. This pattern of findings suggests an explanation for the failure of the ZRFT-transformation to remove the influence of SD_{RT} . The variance of the inhibition function is the result of an interaction between SD_{RT} and SD_{SSRT} . Therefore, expressing inhibition as a function of a Z-score (see Equation 4) overcompensates for the influence of SD_{RT} . This finding challenges the usefulness of the ZRFT transformation as a method to remove primary task contributions and compare variability in $SSRT_{in}$ between groups.

SD_{RT} also has a substantial effect on $SSRT_{Obs}$, as can be seen from Figure 9. The $SSRT_{Obs}$ was quite consistent across the range of SOAs when $SD_{RT} \geq 100$, but deviated substantially when $SD_{RT} = 25$ ms. This effect was attenuated by the RR-tracking procedure, because the distance between SOAs was reduced. The cause of this effect can be found in the interaction of the go- and stop-process distributions. If the RT distribution is wider than the range of SOAs, the race can be won by some of the slow, and most of the fast stop-processes. In that case, both early and late SOAs result in the measurement of SSRT from the entire distribution, because both early and late SSRTs have a reasonable chance of winning a race. If, however, the RT range is narrower than the range of SOA, responses on trials with an early SOA only escape inhibition when the stop-process is extremely slow; whereas only extremely fast stop-processes stand a chance against the go-process if the SOA is late.

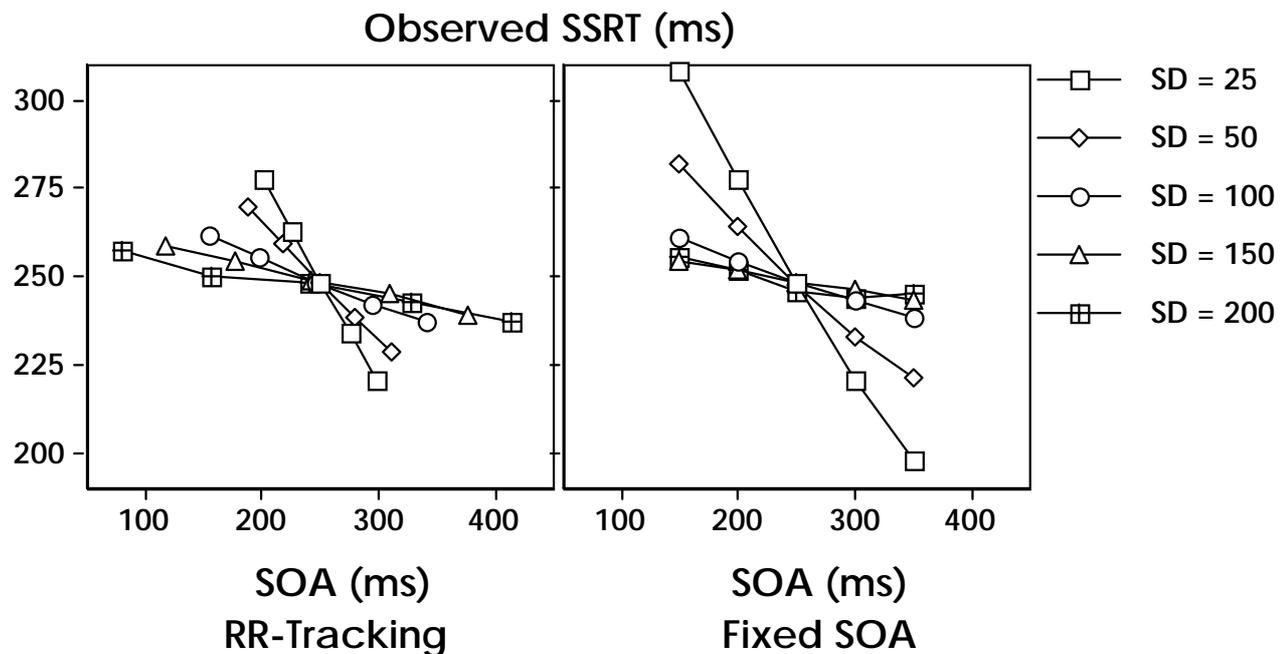


Figure 9. The effect of variance of go-speed and the SOA-procedure on the observed stop-speed. High variance of go-speed has limited effect on the estimation of stop-speed, but low variance makes measurements of stop-speed away from the central SOA unreliable.

Despite the effect of SD_{RT} on $SSRT_{Obs}$, summary $SSRT_{Obs}$ scores were able to remove this effect of single-SOA measurements. Table 4 shows that $SSRT_{Av}$, $SSRT_{Dist}$ and $SSRT_{Med}$ were between 244 and 254 ms for every level of SD_{RT} .

High variability of RT in large part removed the difference between observed and predicted RTs of signal responses. The difference increases if the variability of $SSRT_{in}$ is a relatively large, and RT is a relatively small contribution to the variability of the relative finishing time.

As the variance of go-RT had a considerable effect on the estimation of $SSRT_{Obs}$ on different SOAs, it is important to assess the influence of skewness as well. There is a specific reason for looking at the effect of skewness, which is related to the way how ZRFT is calculated. It can be shown that under the assumption of a constant $SSRT_{in}$, the ZRFT-score is functionally equivalent to

a Z-transformed score of a given go-RT. Plotting RR as a function of ZRFT would be functionally equivalent to plotting the percentile score of a given go-RT as a function of the Z-transformation of that RT. Logically speaking, this function can not depend on the mean or standard deviation of RT, but it could depend on higher order moments of the go-RT distribution, such as skewness (Logan, 1994).

The effect of a positively skewed go-RT distribution was simulated with a fixed SD_{RT} of 100 ms, and exponential proportions of variance amounted 0; .01; .04; .16; .36; or .81, because τ could be 0, 10, 20, 40, 60 or 90 ms.

Just like the skewness of $SSRT_{in}$, skewness of go-RT only had a small effect on all dependent variables, except when $\tau = 90$ ms: In that condition, $SSRT_{Obs}$ was higher for the earliest SOAs, and lower for the middle range of SOA. These situations were accompanied by a lower and higher RR respectively. The inhibition function with $\tau = 90$ deviated more from linearity, thus affecting the results of the $SSRT_{Av}$ and $SSRT_{Med}$ method.

Table 5: The inhibition function and observed stop-speed (ms) as a function of go-RT skewness (ms) and SOA procedure

τ_{RT}	ZRFT-slope		SOA-slope		$SSRT_{Av}$		$SSRT_{Dist}$	$SSRT_{Med}$	
	Fix	RR-T	Fix	RR-T	Fix	RR-T	RR-T	Fix	RR-T
10	-0.32	-0.32	3.2	3.2	249	250	247	250	250
20	-0.32	-0.32	3.2	3.2	250	250	247	250	250
40	-0.32	-0.32	3.2	3.2	249	249	247	249	249
60	-0.33	-0.33	3.3	3.3	249	249	250	247	248
90	-0.35	-0.36	3.5	3.6	247	247	260	239	240

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

Interdependence between SOA and $SSRT_{in}$

Several experiments have revealed a negative relation between SOA and $SSRT_{Obs}$ (e.g. De Jong et al. 1990; Logan & Burkell, 1986). Such a pattern mimics a refractory effect, as if a bottleneck process first finishes computations for the go-process before the stop-process can proceed. In such a case, there would be a negative correlation between SOA and $SSRT_{in}$. A negative correlation between SOA and $SSRT_{in}$ would also be expected if the preparedness of the inhibitory process increased after the presentation of the primary stimulus. Conversely, a positive correlation might be expected if the detection of the stop-signal would be hampered by response-related processing of the go-stimulus, for example because resources for the activation of the go-response would be allocated at the expense of resources that would arouse the perceptual mechanism for the analysis of a stop-signal (cf. Sanders, 1983). However, as shown in the above, a decrease of $SSRT_{Obs}$ with SOA can also be found as a consequence of $SSRT_{in}$ variance, and does not necessarily indicate a correlation. If a correlation would be present, this would be a violation of the independence assumption of the horse-race model.

The effect of interdependence between SOA and $SSRT_{in}(250, 50, 0)$ was tested at nine levels between $r = -.4$ and $r = +.4$ with $RT(500, 100, 0)$. As the exact implementation of these correlations would require knowledge about the influence of RR-tracking and about the success of creating covariance between the rectangular distribution of SOA and the Gaussian distribution of $SSRT_{in}$, the r 's in Table 6 represent input rather than output levels. The actual correlations were -.8, -.6, -.38, -.17, 0, .14, .27, .39, and .52 for the RR-tracking procedure and -.65, -.49, -.32, -.15, 0, .15, .32, .49, and .65 for the fixed-SOA procedure.

Results

It can be seen from Table 6, that the slope of the inhibition function increased with r , and that the difference remained after ZRFT-transformation. The effects on the slope of the ZRFT-transformed inhibition function could be almost as large as the effect from SD_{SSRT} . This holds that the transformed inhibition function can not differentiate entirely differences between groups in SD_{SSRT} from differences in dependence of $SSRT_{in}$ on SOA.

Table 6: The inhibition function as a function of time-dependence of stop speed, and SOA procedure

r	ZRFT-slope		SOA-slope	
	Fix	RR-T	Fix	RR-T
-.4	-0.22	-0.2	2.2	2.0
-.3	-0.25	-0.23	2.5	2.3
-.2	-0.27	-0.26	2.7	2.6
-.1	-0.30	-0.29	3.0	2.9
0	-0.32	-0.32	3.2	3.2
.1	-0.35	-0.35	3.5	3.5
.2	-0.42	-0.39	4.2	3.9
.3	-0.46	-0.43	4.6	4.3
.4	-0.49	-0.47	4.9	4.7

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

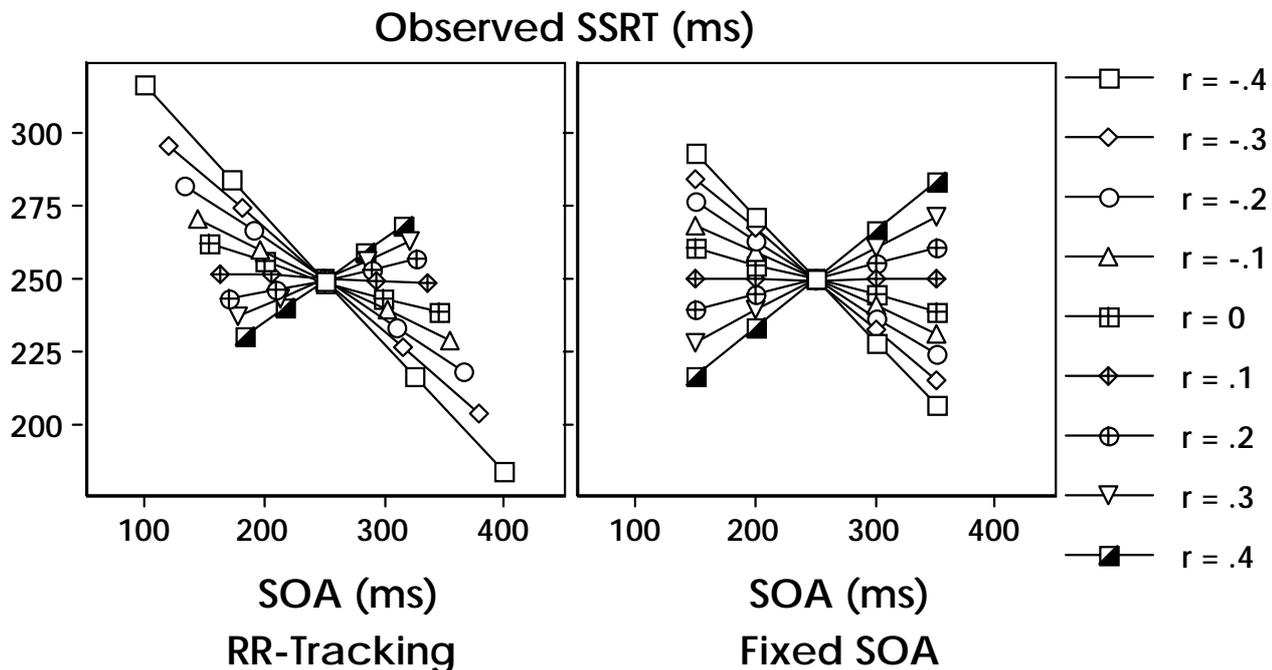


Figure 10. The effect of the SOA-procedure and correlation between SOA and stop-speed on the observed stop-speed. A negative or zero correlation yields the decrease of observed stop-speed with SOA that is known from the literature.

The $SSRT_{Obs}$ decreased with SOA when $r > .1$ and increased when $r < .1$, as can be seen from Figure 10. Empirical data usually show a decrease of $SSRT_{Obs}$ with SOA similar to what was found for negative or zero correlations. For example, Logan and Burkell (1986) found a decrease of approximately 40 ms in $SSRT_{Obs}$ between SOA = 160 and SOA = 320 ms when the probability of a stop-signal was 20%. This decrease would be comparable to what was found when the correlation

was -.2. In contrast, De Jong et al. (1990) found a much shallower decrease of $SSRT_{Obs}$ with SOA, which would be consistent with a zero correlation.

Although single-SOA measurements of $SSRT_{Obs}$ were affected by the interdependence with SOA, summary $SSRT_{Obs}$ scores in these simulations were rather constant, between 245 and 251 ms.

The observed signal responses deviated most from the predicted values when $r = 0$; the difference was then 10 ms at the central SOA. For all levels of dependence, the difference at the central SOA was between 0 and 10 ms. This finding holds that a correlation between SOA and $SSRT_{in}$ can not be detected with the independence test. However, observed – predicted RT is used to test for stochastic dependence; not time- or context-dependence. The effect of a correlation between $SSRT_{in}$ and RT can reveal whether the test is useful for the detection of stochastic dependence.

Of course the outcome of these simulations was determined by the choice of several parameters beside the correlation of SOA and $SSRT_{Obs}$. It would therefore not be justified to draw conclusions about the time-dependence of the speed of stopping from the similarity of empirical to simulated data. However, the data show that time-independence of the stop-speed is plausible in young adult subjects.

Dependence between RT and $SSRT_{in}$

The most important assumption of the horse-race model is that the process durations of going and stopping are independent. The literature on cognitive energetics and dual-task performance suggests that the durations may have a positive correlation due to the simultaneous effect from arousal on both processes (e.g. Sanders, 1983), or a negative correlation due to restrictions in mental capacity (Kramer et al. 1994; Logan & Burkell, 1986; Welford, 1952). De Jong et al. (1990) found in his simulation study that a correlation between RT and $SSRT_{in}$ of +.5 caused an increase of $SSRT_{Obs}$ with SOA, contrary to what is usually observed. Furthermore, a high positive relationship caused an increase in the difference between observed and predicted signal responses, and resulted in a steeper inhibition function. In contrast, a correlation of -.5 resulted in a shallower inhibition function and a sharper decrease of $SSRT_{Obs}$ with SOA.

In the present simulations, we decided to perform a more fine-grained analysis of the effect of violating the independence assumption, and used $RT(500, 100, 0)$, $SSRT_{in}(250, 50, 0)$ and r 's varying between -.4 and +.4 in steps of .1. In addition, this simulation allowed us to investigate the effects on other dependent variables.

Table 7: The inhibition function as a function of primary-task dependence of stop-speed

<i>r</i>	ZRFT-slope		SOA-slope	
	Fix	RR-T	Fix	RR-T
-.4	-0.29	-0.28	2.9	2.8
-.3	-0.29	-0.29	2.9	2.9
-.2	-0.30	-0.30	3.0	3.0
-.1	-0.31	-0.31	3.1	3.1
0	-0.32	-0.32	3.2	3.2
.1	-0.33	-0.33	3.3	3.3
.2	-0.34	-0.35	3.4	3.5
.3	-0.36	-0.36	3.6	3.6
.4	-0.41	-0.38	4.1	3.8

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

Table 7 shows that the inhibition functions are steeper when the correlation between RT and $SSRT_{in}$ becomes more positive, and this difference remains after ZRFT-transformation. The effect is again of almost the same size as the effect of moderate differences in SD_{SSRT} . This underlines that the interpretation of differences in slope of the ZRFT-transformed inhibition function is ambiguous.

Much like the pattern of Figure 10, values of $r > .2$ caused an increase of $SSRT_{Obs}$ with SOA. A likely explanation of this pattern is that when $r = 0$, stopping on late SOAs is only successful for the fastest $SSRT_{in}$'s. However, when RT and $SSRT_{in}$ are positively related, longer $SSRT_{in}$'s can still be successful because they are more often associated with slow go-processes. The reverse is also the case: A negative relation between the go- and stop-process amplified the decrease of $SSRT_{Obs}$ with SOA. Summary measures of $SSRT_{Obs}$ were not affected by the interdependence of the two processes.

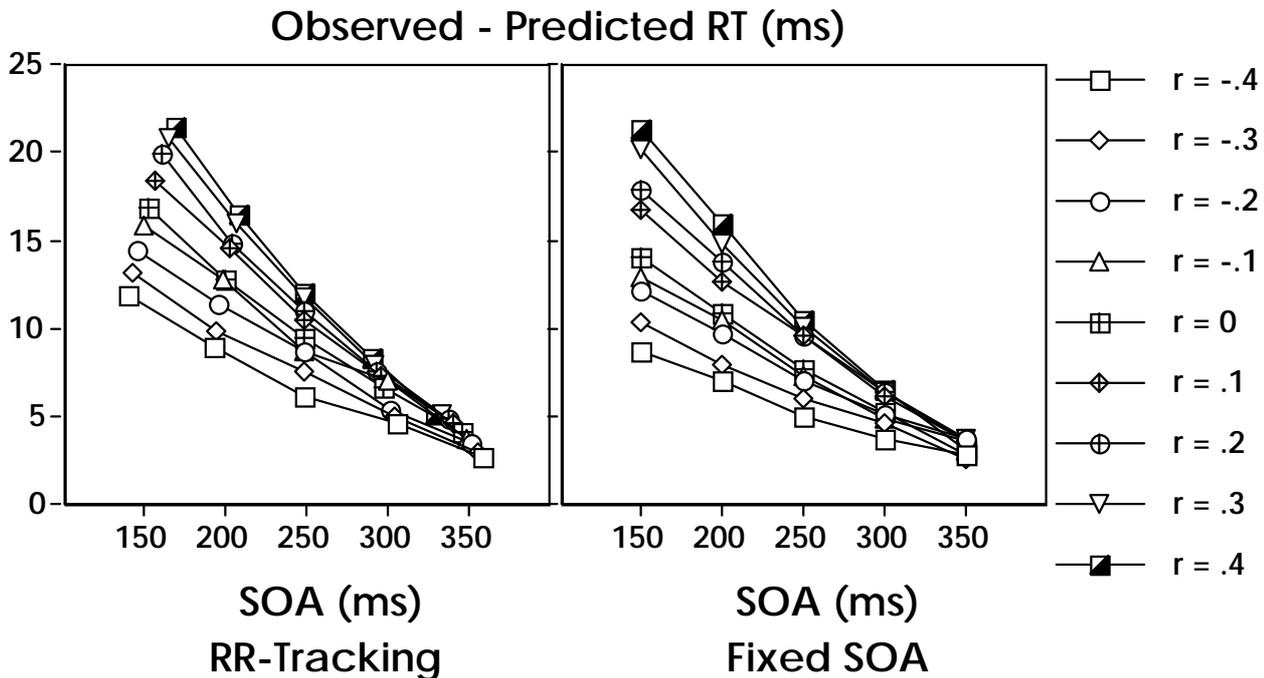


Figure 11. The effect of the SOA-procedure and correlation between go-speed and stop-speed on the difference between observed and predicted speed on signal-response trials. Although this measure is used as a test of independence between the two processes, dependence only has a small effect in comparison with variance of stop-speed or the measurement error.

The difference between observed and predicted signal RT (see Figure 11) increased as r became more positive, up to 12 ms on the central SOA. Given that this is approximately the difference that would occur if there is no dependence between RT and $SSRT_{in}$, the comparison does not have the power to detect a violation of the essential assumption that $SSRT_{in}$ and RT are independent. It should be noted however, that De Jong et al. (1990) found differences of 8 and 33 ms between observed and predicted RTs, in simulations with correlation of +.5. The difference between the present data and those of De Jong et al. can be explained in terms of the choice of SD_{SSRT} , which was 50 ms in our simulations, vs. 27 and 69 ms in De Jong et al.'s. It was shown in the above that the difference between observed and predicted RT primarily depends on SD_{SSRT} . Although it is outside the scope of this study, strategic effect are also likely to cause a rise in observed signal RT.

A decreased triggering rate of the inhibition process

Deficiencies in inhibitory control do not only apply to the speed or variability of the stop-process. For example, it has been noted by Schachar and Logan (1990b; see also Logan & Cowan, 1984) that children with the ADHD-syndrome may differ from normal control children in the triggering rate of the inhibitory process. However, the race model does not provide a tool to detect a deficient triggering. It is therefore important to infer from simulated data what pattern of inhibition scores should be found if this hypothesis were correct. Six levels of triggering rate were compared, decreasing from $p = 1.0$ till $.75$ in steps of $.05$.

Table 8: The inhibition function and observed stop-speed as a function of triggering rate of the inhibition process

p	ZRFT-slope		SOA-slope		SSRT _{Av}		SSRT _{Dist}	SSRT _{Med}	
	Fix	RR-T	Fix	RR-T	Fix	RR-T	RR-T	Fix	RR-T
1.00	-0.33	-0.33	3.3	3.3	248	249	250	247	247
.95	-0.31	-0.31	3.1	3.1	256	258	258	255	257
.90	-0.30	-0.28	3.0	2.8	263	269	266	264	267
.85	-0.28	-0.24	2.8	2.4	272	284	276	274	282
.80	-0.27	-0.14	2.7	1.4	281	329	287	286	327
.75	-0.25	-0.04	2.5	0.4	289	*	293	299	*

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

* = these values can not be computed reliably.

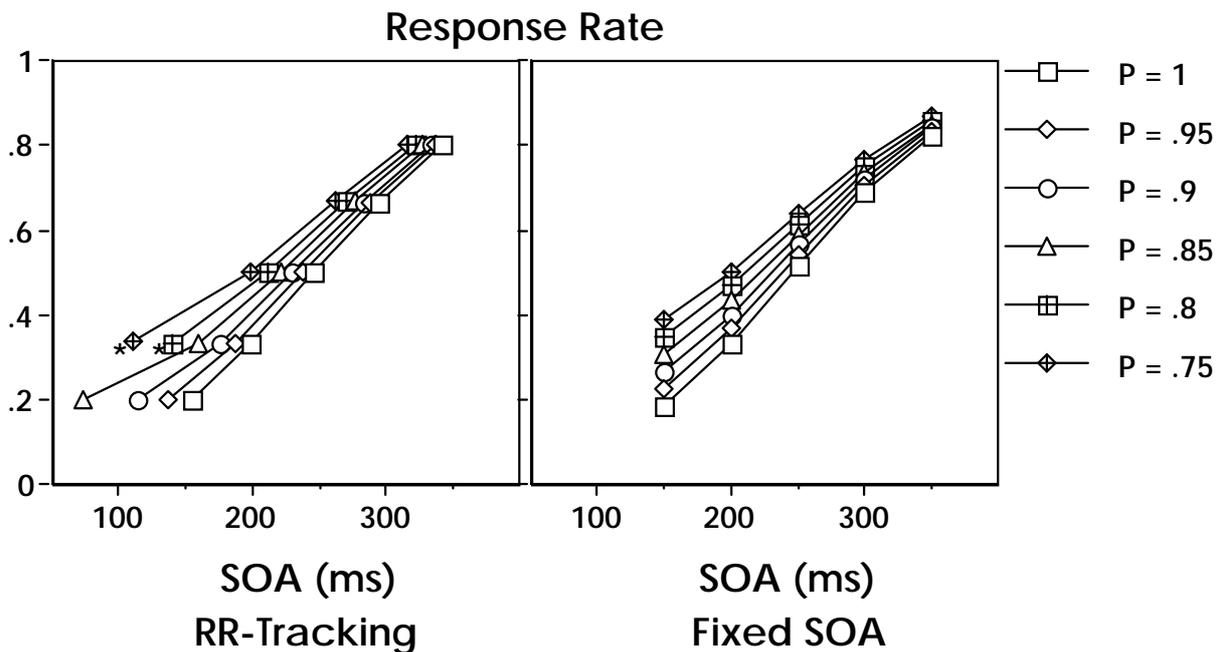


Figure 12. The effect of the SOA-procedure and a decreased triggering rate of the inhibition mechanism on the inhibition function. The slope of the function is shallower as the inhibition mechanism fails more often. * Note that the RR-tracking procedure causes the SOA to decrease to meaningless values if the triggering failure is more frequent than the aimed inhibition rate.

It can be seen from Figure 12 that early SOAs of the inhibition function are more sensitive to a decreased triggering rate, resulting in an increased RR. This is understandable, since the influence of a failing stop-mechanism can be best observed when the stop-mechanism often wins the horse race against the go-process. Note that some values of RR could never be reached with a RR-tracking procedure, because the success at stopping would need to be higher than the chance of the

stop-mechanism triggering. The result is that the SOA continues to drop to negative values. The outcome of these data points would not be very informative, so they are not reported here. ZRFT-transformation does not adjust the slope, which is slightly shallower when the inhibition process is triggered less often.

Even though early SOAs show the largest effect of a failure to trigger, the $SSRT_{Obs}$ is affected over the whole range of SOA (see Figure 13). Interestingly, this failure is the first factor in this paper that caused nonlinear relationships between $SSRT_{Obs}$ and SOA. All summary $SSRT_{Obs}$ scores yield considerable overestimations of $SSRT_{in}$ when $p < 1.0$; up to 40 ms (fixed-SOA procedure) or more (RR-tracking procedure). Because it is not possible to reach an asymptotic SOA that results in $RR = .2$ if the triggering rate is $p = .75$, the RR-tracking procedure yields extremely high summary $SSRT_{Obs}$'s. A comparison of observed and predicted signal RT shows a larger difference when the inhibition process is triggered less often. When $p = .75$, the difference was 18 ms at the central SOA.

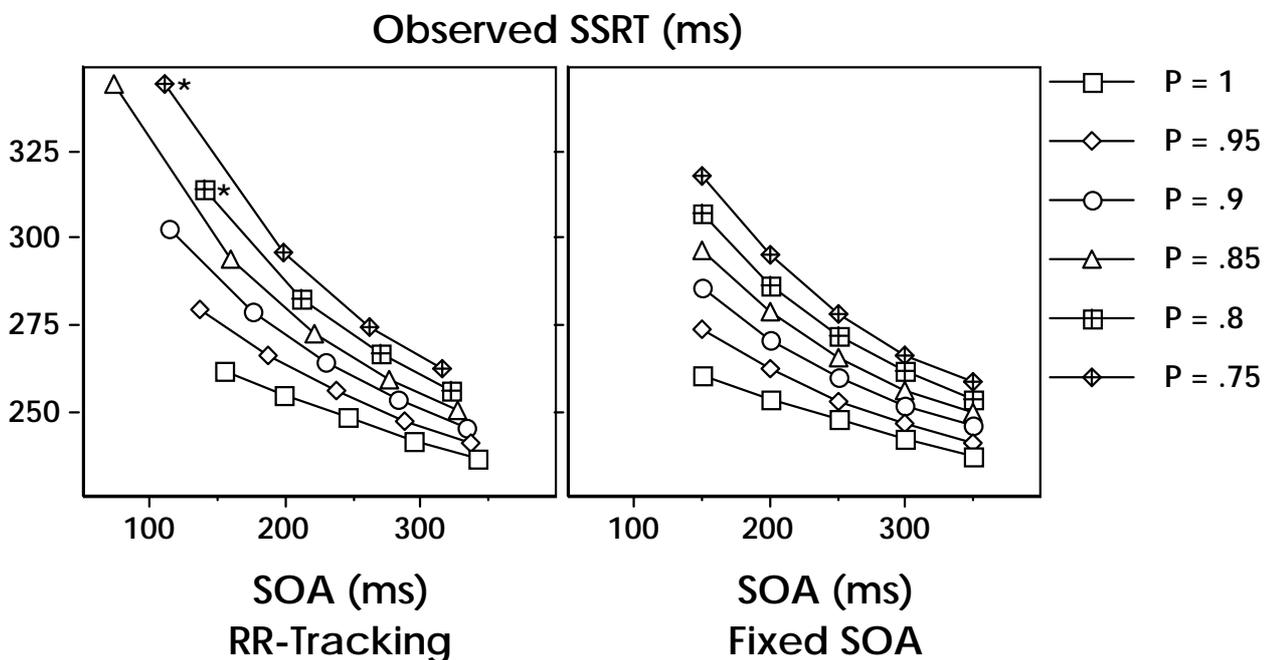


Figure 13. The curvilinear effect of a decreased triggering rate of the inhibition mechanism on the observed stop-speed, for both SOA-procedures. For neither of the SOA-procedures, the true stop-speed of 250 ms can be detected if the inhibitory mechanism occasionally fails to trigger.

* Note that the RR-tracking procedure causes the SOA to decrease to meaningless values if the triggering failure is more frequent than the aimed inhibition rate. For these SOAs it is not informative to calculate observed stop-speed.

Discussion

The preceding section suggests that the methods of the horse-race model (Logan & Cowan, 1984) are not all equally sensitive or suitable for their intended purpose, but that there are several reliable ways to estimate the speed of stopping.

Most importantly, there is good news for the measurement of $SSRT_{Obs}$. Even though $SSRT_{Obs}$'s of single SOAs overestimate $SSRT_{in}$ if $RR < .5$, and underestimate it when RR is higher, three different methods estimate $SSRT_{in}$ reliably over pooled data. If the SOAs cover a range where $.15 < RR < .85$, and if they are not primarily placed on one end of the inhibition function, the average $SSRT_{Obs}$ over different SOAs is reliable. The $SSRT_{in}$ is also estimated rather accurately with the median SSRT method. However, the distance SSRT (Logan & Cowan, 1984) only yields an accurate estimation with the RR-tracking procedure. The distance SSRT fails to

prove differences between $SSRT_{in}$ levels when the SOAs are fixed. Finally, the Colonius method (Colonius, 1990; De Jong et al., 1990) tends to underestimate the speed of stopping, and is therefore not recommended.

It was shown that the slope of the inhibition function is sensitive to many factors, such as SD_{SSRT} , SD_{RT} , a decreased rate of triggering the inhibition system, and a correlation of $SSRT_{in}$ with SOA or RT. In order to extract information about the variability of stop-speed from the inhibition function, several contributions to the position and slope of the inhibition function can be removed. However, the most rigorous transformation; i.e. the ZRFT-correction, is not able to isolate factors that are relevant to the inhibition process from primary task contributions. If in a given data set the slope of one condition is .25 and another is .33, it is not possible to distinguish where this effect stems from. Such an effect size can be the result of irrelevant differences in SD_{RT} (100 vs. 50 ms), as well as it can be the result of relevant differences in SD_{SSRT} (20 vs. 70 ms) or the triggering rate of the stop-process (.85 vs. 1). To a limited extent, dependence of $SSRT_{in}$ on RT or SOA affects ZRFT-slopes, but it is not plausible to suggest that the time or dual-task dependence can be very different for one group than for another. Of course a difference in SD_{RT} can be observed in the nonsignal trials. However the ZRFT-transformation is designed to remove the contribution of SD_{RT} , and this is not fully accomplished. Based on the present simulations it is not justified to conclude from a difference in the ZRFT-slope that the inhibition process in one condition differs from that in another, because it can not be ruled out that factors that are irrelevant to the inhibitory mechanism are responsible.

As the slope of the ZRFT-transformed inhibition function does not allow a conclusion about differences in SD_{SSRT} , it is important to find other ways to estimate the variability of $SSRT_{in}$. The two methods that are reported in the literature were tested, although, to our knowledge they have never been used on empirical data. The Colonius method (Colonius, 1990) would theoretically be able to recover the cumulative distribution of $SSRT_{in}$, and by this method the distance between the 25th and 75th percentile score could be used as a measure of $SSRT_{in}$ -variability. However, in the simulations, the interquartile distance was overestimated, despite the high numbers of signal and nonsignal trials. Another method for the calculation of SD_{SSRT} (see Equation 3) also overestimated the variability. In short, neither of the two measures was able to reveal the absolute variability of the stop-speed, although differences in variability might be detectable with both methods.

The results of the simulations with a correlation between RT and $SSRT_{in}$ and between SOA and $SSRT_{in}$ illustrated that a violation of the assumption that the speed of the stop-process on a given trial is (a) independent of the moment of presentation or (b) independent of the speed of the go-process, has strong implications. The inhibition function and measurements of $SSRT_{Obs}$ on single trials were affected, although the summary scores of $SSRT_{Obs}$ were not influenced. However, judging from the similarity between the data from several empirical studies and the data of the present simulations, there is no reason to conclude that either assumption is violated.

Logan and Cowan (1984) note that the race model makes predictions about the speed of signal-respond trials, and that the accuracy of these predictions can be used to test the validity of the race model. Specifically, it has been used to test whether the independence assumption was violated in the data (cf. De Jong et al., 1990; Jennings et al., 1992). However, the similarity of observed and predicted RT of signal-respond trials was not affected much by the manipulation of the dependence of $SSRT_{in}$ and RT, nor by the manipulation of the dependence of $SSRT_{in}$ and SOA. Moreover, a stronger effect was caused by the manipulation of SD_{SSRT} . These data indicate that the difference between observed and predicted RT is not sufficient as a test of the independence assumption.

The selection of SOAs was tested with the fixed-SOA method and the RR-tracking procedure. There were only a few differences between the results of these methods. However, there were considerable differences in the area of measurements. In general, the RR-tracking procedure was

able to select the SOAs from the entire inhibition function, whereas the fixed-SOA procedure could measure on a too narrow, too wide, too low or too high section of the inhibition function, depending on parameters such as the RT-distribution and the SSRT-distribution. Only in extreme cases, this characteristic could lead to biased estimations of $SSRT_{in}$ and its variability. On the other hand, the fixed-SOA procedure was able to restrict the range of SOAs under parameter conditions where the RR-tracking procedure led to spurious measurements. This advantage was illustrated when the chance of triggering the inhibitory mechanism was manipulated.

In conclusion, the simulations with high numbers of trials showed that the speed of stopping can be measured, but other characteristics of stopping remain disguised with the present state of stop-signal methodology. However, these were results with idealized data sets. The simulations were run with consistent parameter values, a constant shape of $SSRT_{in}$ and RT-distributions, large numbers of trials, no strategy effects and no measurement error. In empirical settings, the accuracy of measurements is likely to be restricted because a smaller number of trials is employed. In the following study, the accuracy of measurements as a function of the number of trials is addressed with the default parameter settings.

STUDY 2: ACCURACY OF MEASUREMENTS

In comparison with many RT-paradigms, the stop-signal paradigm requires a rather high number of trials. If an experimenter wants to keep subjects from anticipating stop-signals, it is imperative to employ a low percentage of stop-trials. In addition, if an experimenter wants to acquire information about inhibitory control on several SOAs, single SOA-conditions may end up representing less than 10% of all trials. In more extreme situations, if the subject population is not very attentive for the time that the session needs to last (as is the case for children with ADHD), it becomes tempting to accept a lower number of trials per SOA.

Study 2 and 3 are meant to provide an answer for researchers who want to know which method of testing is the most profitable and how many measurements need to be performed in order to test their hypothesis. This study will deal with the question how accurate a measurement can be as a function of the number of trials that are presented. An insightful way to do this, is through the calculation of confidence intervals. In order to estimate the number of trials that may be required in order to achieve a reliable estimation, we calculated the size of the 95% confidence intervals. In other words, any experiment with these parameters has a two-tailed probability of 5% of finding results that fall outside the indicated confidence interval. Note that the confidence interval is not distributed around the real values, but around the observed values. For example, $SSRT_{Obs}$ is likely to overestimate $SSRT_{in}$ for early SOAs, but nonetheless the confidence interval of this measure is centered around $SSRT_{Obs}$.

The simulations were run with the default input parameters and between 10 and 100 trials per SOA, combined with between 120 and 1200 nonsignal trials. The ratio of 1 : 12 for the number of stop-trials per SOA and nonsignal trials would approximately be reached if five different SOAs were distributed over 30% of all trials. The size of the 95% confidence interval was calculated as $2 * 1.96 * SE$, where 1.96 is $-t_{.025}$ or $t_{.975}$. To calculate the SE (standard errors), we performed groups of 600 simulated experiments with the indicated numbers of trials.

Conclusions about the required numbers of trials can not be drawn from a confidence interval alone. The maximum acceptable size of a confidence interval depends primarily on the effect size. We chose to determine the numbers of trials that would be required to prove a small and large effect size respectively (cf. Cohen, 1992). Experiments that do not lead to a larger than maximum acceptable confidence interval would not have the power to find significant differences between conditions or groups.

Results

Figure 14 shows the size of the confidence interval for $SSRT_{Obs}$, as a function of SOA and the number of stop-trials. It can be seen that there is rapid decrease with the number of trials in the size of the confidence interval, but that this gain in accuracy is strongest between 10 and 30 trials per SOA. Close to the central SOA, the $SSRT_{Obs}$ is measured most reliably and the confidence interval drops to < 20 ms within 30 trials and to < 10 ms within 50 trials. However, it requires 40 or 70 trials to bring measurements of noncentral SOAs to this level of accuracy. It was shown in Study 1 that the $SSRT_{Obs}$ at the central SOA is the most reliable estimate of $SSRT_{in}$.

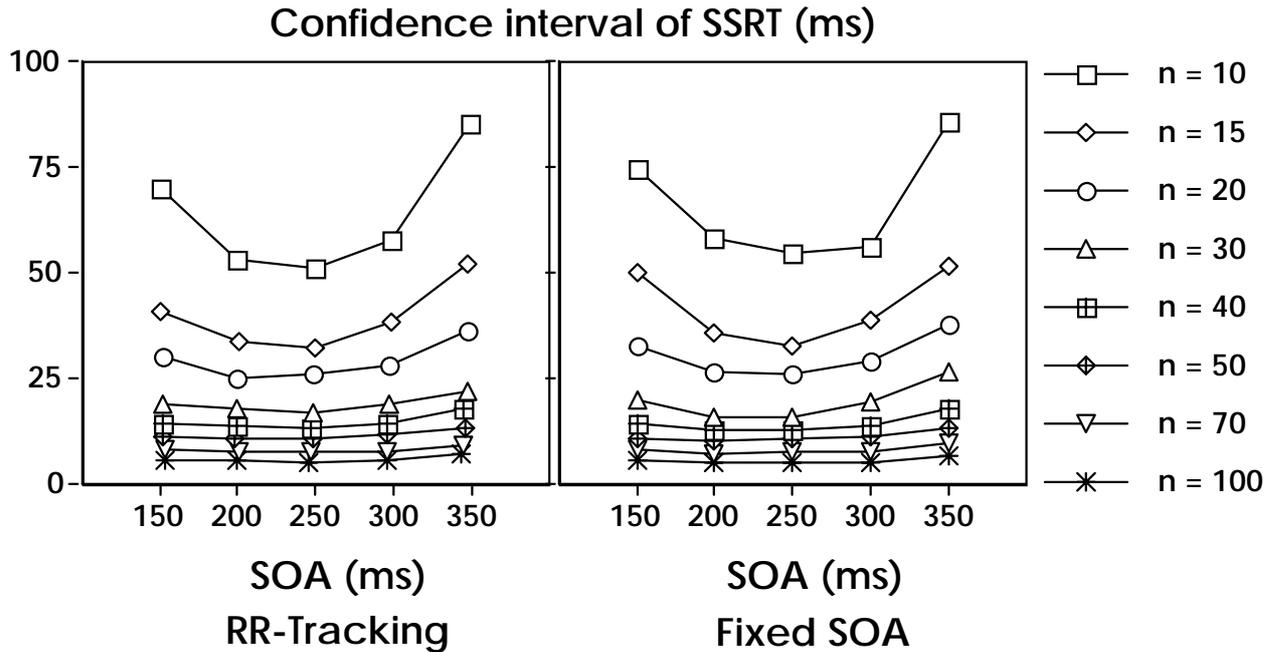


Figure 14. The size of the 95% confidence interval of observed stop-speed, as a function of SOA, the number of stop-trials per SOA, and the SOA-procedure. The effect of the number of stop-trials is large when less than 40 stop-trials are used. The observed stop-speed is more reliable if the SOA is closer to the central SOA.

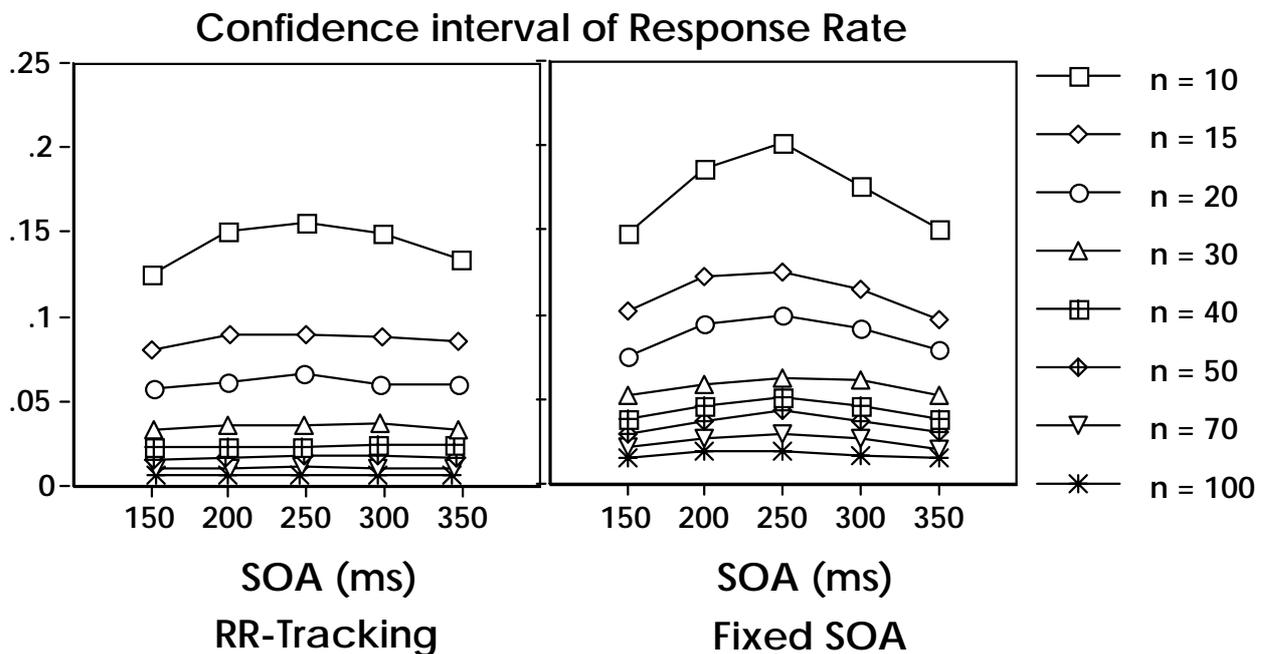


Figure 15. The size of the 95% confidence interval of response rate, as a function of SOA, the number of stop-trials per SOA, and the SOA-procedure. The effect of the number of stop-trials is large when less than 40 stop-trials are used. The response rate is more reliable if the SOA is away from the central SOA.

It can be seen from Figure 15 that for all single-SOA measurements of RR, a confidence interval $< .05$ was attainable with 50 trials for the fixed-SOA procedure or 30 trials for the RR-tracking procedure, provided that the starting values of SOA are well-chosen. A confidence interval $< .02$ was reached within 50 trials for the RR-tracking procedure, but could not be reached with less than 100 stop trials for the fixed-SOA procedure. The latter finding highlights an important characteristic of the RR-tracking procedure. It is possible to compare the inhibitory control in conditions with almost equal RR scores. Not only does the SOA adapt to parameter changes to arrive at an almost constant RR, the RR is also relatively consistent from one experimental session.

Confidence interval of Observed - Predicted RT (ms)

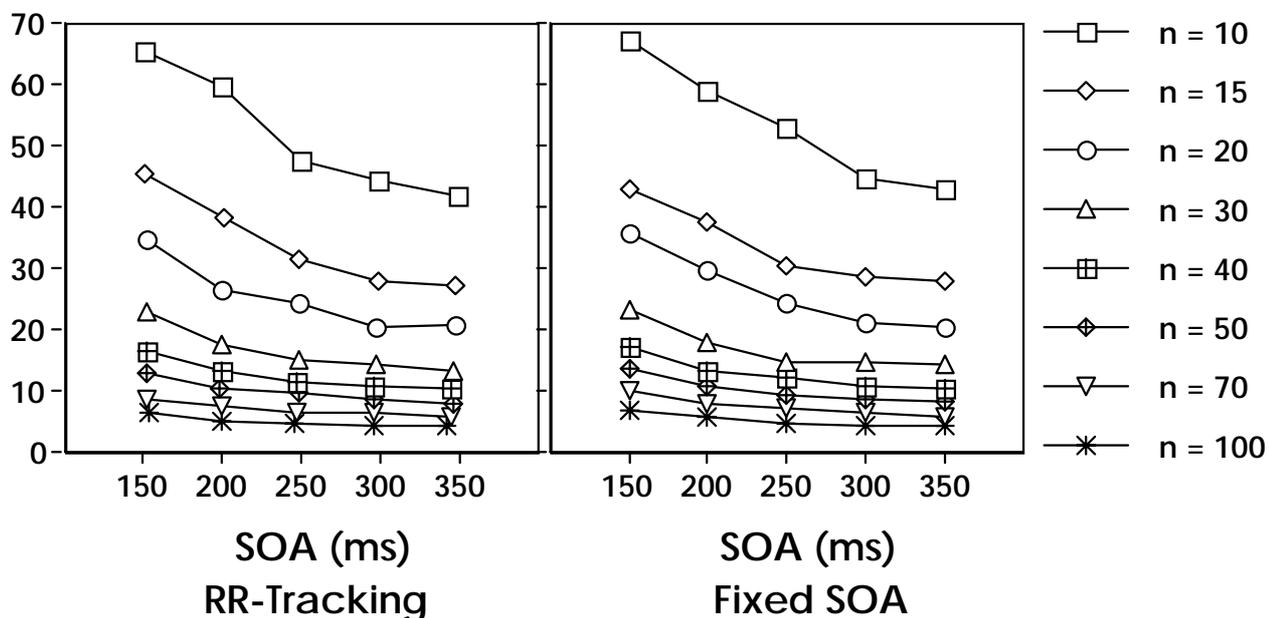


Figure 16. The size of the 95% confidence interval of observed minus predicted RT on signal-response trials, as a function of SOA, the number of stop-trials per SOA, and the SOA-procedure. The effect of the number of stop-trials is large when less than 40 stop-trials are used. The confidence interval remains close to 10 ms for 70 stop-trials per SOA.

The difference between observed and predicted RT of signal trials was not very consistent (see Figure 16), and had the largest deviation at early SOAs. A confidence interval of < 20 ms of this difference could be attained within 40 stop-trials per SOA, but a confidence interval of 10 ms required 70 ms. The same accuracy can be reached after 30 or 50 stop-trials at the central SOA, respectively. Effect sizes of 10 - 20 ms are large in comparison with the absolute size of the difference and in comparison with the effect of SD_{SSRT} . Just as in Study 1, these simulations render the interpretability of the comparison between observed and predicted RT questionable.

Table 9 shows how reliable summary measures are as a function of the number of stop-trials. For the summary inhibition scores, it should be noted that if less than five SOAs are used, the scores in Table 9 overrate the accuracy. Regression functions, which are used to determine the slope of the inhibition functions, are more reliable as they are based on more data points and as these data points cover a wider range of the control variable (SOA or ZRFT). It is easy to see that an

experiment with only two SOAs is likely to yield a less reliable slope of the inhibition function. In any case, linear regression requires at least two SOAs to interpolate or extrapolate inhibition results to the central SOA, but requires more SOAs to retain degrees of freedom for the estimation of an inhibition function.

The slope of the ZRFT is commonly used in order to detect differences in SD_{SSRT} or the triggering rate of the inhibitory mechanism. The simulations in the above (see Tables 3 and 8) show that a meaningful difference in either of these two variables has a small and large effect size on the slope of the ZRFT inhibition function of $< .02$ and $< .05$ respectively. We decided that the slope of the $RR = f(ZRFT)$ function should have these same sizes as their confidence intervals. With these criteria, a reliable comparison between conditions is possible with 30 trials per SOA for all SOA-procedures, but detection of small effects requires 70 trials. The same numbers of trials are required if effect sizes of $.2$ and $.5 s^{-1}$ need to be demonstrated for the regular inhibition function.

The summary scores of $SSRT_{Obs}$ were required to yield a confidence interval < 5 or < 10 ms. This score could be reached for $SSRT_{Av}$ with 30 - 50 stop-trials per SOA, or for $SSRT_{Dist}$ and $SSRT_{Med}$ after 40 - 60 trials. In contrast, $SSRT_{Col50}$ did not reach an interval < 27 ms in the first one hundred stop-trials per SOA.

Table 9: Size of 95% confidence intervals of the inhibition function and observed stop-speed (ms) as a function of the number of stop-trials per SOA and the SOA procedure

<i>n</i>	ZRFT-slope		SOA-slope		SSRT _{Av}		SSRT _{Dist}	SSRT _{Med}	
	RR-T	Fix	RR-T	Fix	RR-T	Fix	RR-T	RR-T	Fix
10	0.15	0.15	1.8	1.5	26	26	42	41	48
15	0.10	0.10	1.0	1.0	18	18	24	25	30
20	0.07	0.07	0.7	0.7	14	13	18	20	23
30	0.04	0.04	0.4	0.4	9	9	11	10	13
40	0.03	0.04	0.3	0.4	7	6	8	8	8
50	0.02	0.03	0.2	0.3	5	5	6	6	7
60	0.02	0.02	0.2	0.2	5	4	6	5	6
70	0.02	0.02	0.2	0.2	4	4	4	4	5
80	0.02	0.02	0.2	0.2	3	3	4	4	4
90	0.01	0.02	0.1	0.2	3	3	3	3	3
100	0.01	0.01	0.1	0.1	3	3	3	3	3

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.

Discussion

In summary, for the detection of large experimental effects on inhibition scores, 40 stop-trials per SOA should suffice. A small effect requires at least 70 stop-trials per SOA. The procedure for the choice of SOAs did not have large effects on the accuracy of measurements. RR-tracking was remarkably advantageous for the measurement of RR, but was only mildly more accurate on other measures.

Depending on the measure of interest, it is worth considering the option to decrease the number of SOAs. For a given number of SOAs, the $SSRT_{Av}$ score is a measure that requires relatively few trials. However, it is always based on a combination of SOAs and is less sensitive to measurement error as more SOAs are combined. Likewise, $SSRT_{Dist}$ can only be calculated from measurements on several SOAs. If, however, only the central SOA could be used and summary measures are ignored, an estimate of $SSRT_{in}$ with a 5 ms confidence interval can be acquired with 100 stop-trials and 300 nonsignal trials. The central SOA is not necessarily approximated in a fixed-SOA or RT-tracking procedure, although under some restrictions (e.g. no interdependence, a high

SD_{RT} and a low SD_{SSRT}) an SOA that is too early or too late would be reliable. The central SOA is easy to find with the RR-tracking procedure, however. The use of 100 stop-trials reduces the confidence interval of RR to $<.01$, so that the single SOA measurement of $SSRT_{Obs}$ becomes identical to $SSRT_{Med}$ (cf. Logan et al., in press; Osman et al., 1986). In such a way, it is possible to reach a high accuracy estimate of $SSRT_{in}$, but not of the inhibition function. Given the difficulties of interpreting the slope of the inhibition function, as shown in Study 1, this fast procedure for estimating the speed of stopping may be the optimal procedure for many purposes.

STUDY 3: POWER FOR THE DETECTION OF DIFFERENCES BETWEEN CONDITIONS

The previous results suggest that either 100 stop-trials with the central SOA or a minimum of 40 or 70 stop-trials per SOA are required for a reliable single-subject measurement of inhibitory efficiency. A logical next question for researchers is how many subjects are required before a reasonable power is reached to detect difference between averages. The power to detect a difference between two averages depends on four factors; (a) the effect size, (b) α , (c) sample size, and (d) variability of the population .

Table 10: Required sample size for a power of .8, as a function of heterogeneity for stop-speed at the closest-to-central SOA

Procedure	Pop. SD_{RT}	RR-T				Fix			
		50		100		50		100	
Pop. $SD_{SSRT_{in}}$		25	50	25	50	25	50	25	50
Effect size									
$SSRT_{Obs}$	10	190	613	213	635	179	490	233	592
	20	48	153	53	159	45	123	58	148
	40	12	38	13	40	11	31	15	37
	60	5	17	6	18	5	14	6	16

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure. Pop. SD = population standard deviation (ms). Effect size is in ms.

Of course, the effect size plays a crucial role (cf. Cohen, 1992), as well as the settings of most of the parameters that were discussed in Study 1. In order not to make the paper too long, we made our calculations with the default values, trusting that readers can extend the domain for their own purpose with the help of Study 1. With the three approved methods, we calculated the number of subjects that are required in order to reach a power ($1 - \beta$) of .8 for the detection of differences between two scores in a t -test, with an α of .05. The calculations are based on the assumption that two groups or conditions yield the same distribution of measurements, but a different mean. The number of subjects¹⁷ (n) that need to be tested to achieve a power of .8 was computed by $n = 24.73 * (SD/Effect\ size)^2$.

The following results were calculated on the basis of the standard deviations of dependent measures after 400 replications. Each replication consisted of an experiment with the default input parameters and an additional individual deviation of RT and SSRT_{in}, with 300 nonsignal + 100 signal trials for the central SOA; or 1050 nonsignal trials and 70 signal trials for each of five SOAs; or 600 nonsignal trials and 40 signal trials for each of five SOAs.

Not only effect size, also the variance in the sample affects the power of statistics. For the results of the stop-signal paradigm, the main sources of heterogeneity concern the population variance of go- and stop-speed. Therefore, in contrast with previous simulations, the mean RT and mean SSRT_{in} were different for every subject, with population SDs of 50 or 100 ms for RT and 25 or 50 ms for SSRT_{in}. Simulation that are not reported here indicated that manipulation of within-subject variability of SSRT_{in} and go-RT did not affect the measurements far beyond the effect of between-subject variability in go-RT and SSRT_{in}. This was reason to report only the influence on power of between-subject variability.

Results

Table 10 displays the number of subjects that are needed to detect differences in SSRT_{Obs} between conditions, on the basis of 100 stop-trials on the closest-to-central SOA, and 300 nonsignal trials, as a function of population variance of RT and SSRT_{in}. It is possible to detect differences of 40 ms in the SSRT_{Obs} at the middle SOA with 17 subjects, if the variability of SSRT_{in} is not too large. This is true for the RR-tracking procedure as well as for the fixed-SOA procedure. However, the RR-tracking procedure measures SSRT_{Obs} close to the central SOA, whereas RR is not necessarily close to .5 with a fixed SOA. It was shown that under some situations (e.g. low SD_{RT}) SSRT_{Obs} is less reliable away from the central SOA. The table also shows that the effect of the population variance of RT on the closest-to-central SSRT_{Obs} is not nearly as large as the effect of population variance of SSRT_{in}.

Table 11: Required sample size for a power of .8, as a function of heterogeneity for stop-speed and effect size with 70 stop-trials

Procedure		RR-T				Fix			
		50		100		50		100	
Pop. SD RT	Pop. SD SSRT _{in}	25	50	25	50	25	50	25	50
Effect size									
ZRFT-slope	0.03	55	63	69	76	109	161	189	205
	0.05	20	23	25	27	39	58	68	74
	0.08	8	9	10	11	15	23	27	29
	0.10	5	6	6	7	10	15	17	18
SOA-slope	0.3	53	59	65	75	108	159	188	199
	0.5	19	21	23	27	39	57	68	71

$$\mu_a + \sigma_a * \Phi^{-1}(.95) = \mu_b - \sigma_b * \Phi^{-1}(.80)$$

$$\sigma_a = \sigma_b = \frac{\sigma}{\sqrt{n}}$$

$$\frac{\sqrt{n}(\mu_a - \mu_b)}{\sigma} = 2 * (\Phi^{-1}(.80) + \Phi^{-1}(.95))$$

$$n = 4 * (\Phi^{-1}(.80) + \Phi^{-1}(.95))^2 * \frac{\sigma^2}{(\mu_a - \mu_b)^2}$$

$$n = 24.73 * \frac{SD^2}{Effect^2}$$

	0.8	7	8	9	10	15	22	26	28
	1.0	5	5	6	7	10	14	17	18
SSRT _{Av}	10	162	608	153	565	151	558	157	494
	20	41	152	38	141	38	139	39	124
	40	10	38	10	35	9	35	10	31
	60	5	17	4	16	4	15	4	14
SSRT _{Dist}	10	156	394	317	556	*	*	*	*
	20	39	98	79	139	*	*	*	*
	40	10	25	20	35	*	*	*	*
	60	4	11	9	15	*	*	*	*
SSRT _{Med}	10	167	613	161	589	215	669	329	648
	20	42	153	40	147	54	167	82	162
	40	10	38	10	37	13	42	21	41
	60	5	17	4	16	6	19	9	18

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure.
 Pop. SD = population standard deviation (ms). SSRTs are in ms.
 * The SSRT_{Dist} is not reliable for the fixed-SOA procedure.

The power of detecting differences in RR on the central SOA is strikingly better for the RR-tracking than for the other SOA-procedure. However, differences in RR can not be interpreted when the RR-tracking procedure is used without referring to the corresponding SOAs. The advantage of the RR-tracking procedure is illustrated by the results of the inhibition functions. The sample size that is required to show a significant difference between conditions, is displayed in Table 11 and 12.

It was illustrated in Study 1 that an effect size of .03 on the slope of the ZRFT-corrected inhibition function can be indicative of a meaningful difference in SD_{SSRT} or other parameters. However, detection of differences of this size would require more subjects than are usually tested – up to 70 per condition when 70 stop-trials per SOA are used, or up to 160 when 40 stop-trials are used. Similar numbers are required to show a difference of .3 on the normal inhibition function. Moreover, two or three times as many subjects are required for the detection of slope differences with the fixed-SOA procedure. Overall, the influence of heterogeneity on the power of slope effects is limited. Note that the ZRFT-transformation did not affect the accuracy of the data to the extent that the power for detecting slope differences was higher than the normal inhibition function.

Table 12: Required sample size for a power of .8, as a function of heterogeneity for stop-speed and effect size with 40 stop-trials

Procedure	Pop. SD RT	RR-T				Fix			
		50		100		50		100	
Pop. SD SSRT _{in}		25	50	25	50	25	50	25	50
	Effect size								
ZRFT-slope	0.03	99	126	164	136	240	221	335	312
	0.05	36	45	59	49	86	79	121	112
	0.08	14	18	23	19	34	31	47	44
	0.10	9	11	15	12	22	20	30	28
SOA-slope	0.3	173	123	162	368	228	218	339	295

	0.5	62	44	58	132	82	79	122	106
	0.8	24	17	23	52	32	31	48	42
	1.0	16	11	15	33	20	20	30	27
SSRT _{Av}	10	172	599	190	613	148	512	191	567
	20	43	150	47	153	37	128	48	142
	40	11	37	12	38	9	32	12	35
	60	5	17	5	17	4	14	5	16
SSRT _{Dist}	10	190	321	594	737	*	*	*	*
	20	47	80	148	184	*	*	*	*
	40	12	20	37	46	*	*	*	*
	60	5	9	16	20	*	*	*	*
SSRT _{Med}	10	187	653	227	695	226	684	501	708
	20	47	163	57	174	56	171	125	177
	40	12	41	14	43	14	43	31	44
	60	5	18	6	19	6	19	14	20

Note: Fix = fixed-SOA procedure, RR-T = Response-Rate tracking procedure. Pop. *SD* = population standard deviations (ms). SSRTs are in ms.

* The SSRT_{Dist} is not reliable for the fixed-SOA procedure.

Effects on summary scores of SSRT can be detected with smaller numbers of subjects. It can be seen from Table 11 and 12 that variability of RT does not affect the power much beyond the influence of variability of SSRT_{in}. With limited variability of RT and SSRT_{in}, it is feasible to show differences of 20 ms, both with 70 and with 40 stop-trials per SOA. The power does not differ much between the SOA procedures, and SSRT_{Av}, SSRT_{Dist} and SSRT_{Med} have a comparable power. It appears that SSRT_{Dist} – which is only calculated for the RR-tracking procedure – is more sensitive to RT-variability, whereas SSRT_{Av} and SSRT_{Med} are more sensitive to SSRT_{in}-variability.

GENERAL DISCUSSION

The stop-signal paradigm is a useful method for the assessment of inhibitory control. However, performance on this paradigm can only be interpreted with the help of the horse-race model, which holds that a primary response process takes part in an independent race against an inhibitory process. In this paper, we addressed four main questions about the design and interpretability of results on the stop-signal paradigm. Specifically, we investigated the outcome of experiments that were simulated with the help of the race model. Performance characteristics of the stop- and go-process were manipulated over a wide range of parameter values. The results were used to (a) compare two different procedures for timing the occurrence of stop-signals, (b) explore the consequences of a violation of assumptions of the race model, (c) assess the possibilities of the race model to detect the locus of differences in inhibitory efficiency, and (d) evaluate the power for detecting differences between conditions or between groups in inhibition scores. More in general, the simulations have considered the accuracy of measures of inhibitory efficiency that have been reported in the literature, and have summarized the effects of inhibition-irrelevant parameter changes on dependent measures of inhibition.

The selection of SOAs

The main difference between the RR-tracking procedure and the fixed-SOA procedure is that the former renders constant RRs and different SOAs for different conditions, whereas the latter

renders constant SOAs with different RRs. The advantage of a constant RR is theoretical as well as methodological.

Theoretically, performance in a stop-signal experiment is likely to be affected by the subjects' reflection on the proportion of signal-responses. Because the stop-signal paradigm combines the instruction to respond as fast as possible on the primary task with the instruction to withhold responses on signal trials if possible, subjects have to balance between the preparation for one process and another. It is known from dual-task literature that performance on a task can affect the performance on a competing task (e.g. Guttentag, 1989). Furthermore, literature about the trade-off of speed and accuracy teaches that the instruction to avoid errors obliges subjects to slow down (e.g. Meyer, Irwin, Osman, & Kounios, 1988). A comparison of speed between conditions is only justified if performance on the competing instruction is equal. Likewise, a valid comparison of conditions on the stop-signal paradigm requires equal response rates. An additional strategic consequence of nonadaptive SOAs is that the occurrence of a stop-signal is predictable, and subjects can await the occurrence in order to avoid signal responses (e.g. Logan, 1981). This problem can be bypassed by a response-rate tracking procedure. Finally, it can be argued that a subject who makes few signal responses due to a low SOA, develops a different attitude or strategy towards the instruction to stop, than a subject who is continuously frustrated by the unavoidable stop-signals.

Study 1 illustrates methodological reasons to keep response rates constant between conditions. If $SSRT_{in}$ is variable, the observed speed of stopping decreases with SOA. This pattern can be explained by differences in response rate between these SOAs, in so far that $SSRT_{in}$ is estimated most reliably around the central SOA, where the response rate is .5. Therefore, a comparison between conditions is only warranted if the corresponding response rates are similar, or better yet, close to .5. If the response rates differ between conditions, and the SOAs are different as well, it is difficult to see which condition is associated with a more efficient inhibitory mechanism. For the comparison of single-SOA measurements, the response rate tracking procedure solves the problem of unequal response rates (cf. Osman et al., 1986; Logan et al., in press). It is able to reduce the response rate margin to 5% with less than 30 trials, and yields the same response rates for different conditions. As a result of the flexibility, not many stop-signal trials are lost to SOAs that measure the tails of the inhibition function. It was shown in Study 2 and 3 that the speed of stopping can be derived with a combination of 100 stop-trials and 300 nonsignal trials, if the algorithm of RR-tracking is set to tracking a response rate of .5.

The fixed-SOA procedure employs time, rather than response rate as the constant. As a result, any difference in performance can be attributed to differences in primary task characteristics. A slight improvement is made if the SOAs are chosen relative to the reaction time on the primary task, as is the case with the RT-tracking procedure. By tracking nonsignal RT, any SOA can be expressed as a fixed distance to RT, but nonetheless the response rate differs between conditions, unless stop-speed is identical. Results with the RT-tracking procedure that were not reported in this paper, were not different from results with the fixed-SOA procedure.

To compare stop-speed, the fixed-SOA procedure requires more than one SOA. For this purpose the SOA at a desired response rate can be derived by interpolating or extrapolating from the linear section of the inhibition function, which is approximately delimited by response rates of .15 and .85. Whether or not several SOAs should be used depends on two factors. On the one hand a higher number of SOAs increases the accuracy of interpolation, regression or summary $SSRT_{Obs}$, but on the other hand there is a trade-off between the number of replications per SOA that can be measured, and the number of SOAs. Especially when subjects have difficulty sustaining their attention, it is better to invest in a large number of trials on a small number of SOAs. In this case, RR-tracking has shown to be a valuable procedure.

Because it can be reasoned that the RR-tracking procedure adds variability to the finishing time of the stop-process, the influence of this procedure on variables that are sensitive to variability was assessed. It turned out that the variability of SOAs did not affect the slope of the normal, or normalized inhibition function.

In the preceding simulations, fixed-SOA levels were selected around the central SOA based on knowledge about RT and $SSRT_{in}$. Because it is not possible for empirical studies to know in advance what the central SOA will be and how large the distance between SOAs should be in order to find sufficiently different response rates, tracking response rates is a cheap and fast solution. The only factor that the RR-tracking procedure could not compensate for entirely, is a decreased rate of triggering the inhibition mechanism.

If a summary measure of stop-speed is used, differences in response rate are disregarded, and the summary score is representative of the stop-speed at the central SOA, or close to it. It was shown that the summary measures $SSRT_{Av}$ and $SSRT_{Med}$ are robust against a selection of SOAs that is not optimally adjusted to mean $SSRT_{in}$ and RT. The fixed-SOA procedure provided a reliable estimation of $SSRT_{in}$, even when the majority of measurements was from one end of the inhibition function. $SSRT_{Dist}$, however, did not reveal the true speed of stopping when the inhibition function was not symmetric around the central SOA, so it could only be used with the RR-tracking procedure. Furthermore, the procedure for estimating the median stop-speed that Colonius (1990; see also De Jong et al., 1990) introduced did not stand up to the expectations. It is less accurate than $SSRT_{Av}$ and $SSRT_{Med}$, whereas it requires a high quality description of RT-distributions.

If several SOAs are used, the slope of the inhibition function can be interpreted in terms of variability in the go- and stop-process. Although it was shown that it is not possible to derive the variance of the stop-speed from a normalized inhibition function, this measure may still be used with some reservation. Study 3 showed that the accuracy of the RR-tracking procedure in finding a response rate also improved the power for detecting differences in the ZRFT-slope in comparison with the fixed-SOA procedure.

The assumptions of the horse-race model

The effect of violations of three assumptions that underlie the calculations with the horse-race model, was simulated in this paper. The first assumption of the horse-race model is that the speed of the inhibitory process is constant. Although Logan and Cowan (1984) also developed a version of the horse-race model that does not assume a constant speed of stopping, there are advantages to the model if the assumption would be met, or if the assumption would have limited consequences. The effect of variability in the stop-process duration was tested and it was found that most dependent variables are responsive to this violation. Most notably, the estimated speed of stopping showed a stronger decrease with the stop-signal delay as the variability of the speed of stopping increased (cf. Logan & Burkell, 1986). However, it was well possible to estimate the average speed of stopping, despite variability of the stop-process (Logan & Cowan, 1984). Variability of stop-speed also had a substantial effect on the slope of the inhibition function, that remained visible after normalization with the ZRFT-transformation. Finally, it is important to note that the difference between observed and predicted signal RT was close to zero when the stop speed was constant but rose considerably with variance in the speed of stopping.

It has been concluded by several researchers (e.g. Logan & Burkell, 1986) that a decrease of $SSRT_{Obs}$ with SOA in empirical data of normal adults was indicative of a variable $SSRT_{in}$, and this conclusion is supported by the present simulations. In addition, the fact that the slope of the inhibition function is shallower than the cumulative distribution function of RT, suggests that there is an additional source of variance, and this is probably the variance in $SSRT_{in}$. Given that children and clinical groups usually show more latency variance than normal adults (e.g. Hale et al., 1993), it

is fair to generalize this violation of constant stop-speed to other groups. Therefore, it does not seem possible with the present methods, to get a reliable estimation of the true $SSRT_{in}$ for single SOA measurements. Instead, only a summary estimate of $SSRT_{in}$, or an observation of $SSRT$ close to the central SOA is warranted. However, Study 1 showed that the reliability of $SSRT_{Obs}$ away from the central SOA is increased if go-RT is more variable.

The second assumption of the horse-race model is context-independence of the stop process. We evaluated the consequences of a variable stop-speed over time. In the simulations, a correlation between the speed of stopping and the delay of the stop-signal had a substantial effect on response rate as well as on the observed speed of stopping. Again, however, two summary scores of the observed speed of stopping ($SSRT_{Av}$ and $SSRT_{Med}$) were able to yield a reliable estimate of $SSRT_{in}$. It was shown that the slope of the normalized inhibition function was sensitive to time-dependence of $SSRT_{in}$. Furthermore, the difference between observed and predicted signal RT was not affected by the time-dependence of $SSRT_{in}$.

Third, the horse-race model is based on the assumption that the speed of stopping is not related to the speed of responding. A correlation between these two process durations had effects on the observed speed of stopping on single SOAs and on the response rate, but did not influence the average speed of stopping. Remarkably, the difference between observed and predicted RT on signal trials was not affected by the correlation of RT and $SSRT_{in}$, or the correlation of SOA and $SSRT_{in}$. In contrast, there was a large effect of SD_{SSRT} . This pattern of results contrasts with the way how observed – predicted RT is commonly used (e.g. De Jong et al., 1990; Jennings et al., 1992). These studies used the difference as a test of the independence assumption of the race-model.

Whether or not $SSRT_{in}$ decreases with SOA in empirical data, and whether $SSRT_{in}$ is correlated with RT, is still disputable, but the similarity between a number of data sets and the results with noncorrelated data suggests that there is no large violation of the independence assumptions.

Detecting the locus of inhibitory deficiencies

The speed of stopping can usually be determined with high accuracy. In addition to the mere speed of stopping, there are other aspects of performance that reflect a reduced inhibitory efficiency. Some researchers have argued, for example, that the variability of the stop-process duration is an important characteristic of inhibitory efficiency. Colonius (1990) and De Jong et al. (1990) developed a method for the estimation of the $SSRT_{in}$ distribution, on the basis of the distribution of reaction times of signal and nonsignal responses. Theoretically, it would be possible to derive the variability of the stop-process from this distribution. In order to bring this method into practice, however, a smooth probability density function is required of both reaction-time distributions. These can not be reached with the numbers of trials that we discussed in this paper, and even less so if the variability of the primary process is high. Moreover, the results of Study 1 suggest that even with smoothed and high-resolution estimations of the RT-distributions, this method largely overestimates the variability of stop-speed.

Another method, suggested by Logan and Cowan (1984) estimates the variance of the stop-process from the variance in go-RT and the slope of the inhibition function, with the assumption that the variance of the inhibition function is the sum of the variances of the two processes in the race. Study 1 shows that this calculation overestimates the variance of $SSRT_{in}$. Thus, neither of the two methods was able to estimate the absolute size of the variability. However, differences in variability may be detectable, because a higher SD_{SSRT} result in a higher estimated variability.

In the literature, direct estimations of SD_{SSRT} are not reported. Instead, the efficiency of the inhibitory mechanism is often analyzed through the slope of the inhibition function. Without correction, the slope of the inhibition function is primarily indicative of the variability in the speed

on the primary task, so that it does not reveal much about inhibition. By making stepwise transformations of SOA, the inhibition function should become increasingly informative about the efficiency of inhibitory control. Differences in the slope of the corrected inhibition function are said to be caused by other factors than what has been corrected for. Differences that remain after the most rigorous normalization of the inhibition function through the ZRFT procedure (Logan & Cowan, 1984) are hypothesized to reflect differences in the variability of $SSRT_{in}$ or triggering rate (Schachar & Logan, 1990b). The latter deficiency holds that, regardless of the speed and variability of stopping, the stop-process occasionally fails to be even started.

Study 1 shows that even the ZRFT transformation is not entirely able to remove the differences between subjects in primary task characteristics. More specifically, the slope is at least affected to the same extent by variability in the speed on the primary task and by interdependence of $SSRT_{in}$ with the SOA or with the speed on the primary task, as it can be affected by inhibition-relevant factors. Furthermore, Study 3 shows that medium-size differences in ZRFT-slope (0.03) can not be detected, unless a large sample size is used, in combination a large number of trials per subject. The combination of the poor power and the multiple interpretations of ZRFT-effects implies that it is hardly attainable with this method to prove differences between groups or conditions in variability of $SSRT_{in}$ or in the reliability of the stop-mechanism. In contrast, an inhibitory deficiency that is reflected in the speed of stopping should be easy to detect by several methods.

Sensitivity and power of inhibition measures

It was shown in the above that $SSRT_{Av}$, $SSRT_{Med}$ and to some extent $SSRT_{Dist}$ are robust against all the tested manipulations, except for an occasional failure to trigger the stop-process. Because research with the stop-signal paradigm often yields more data than just the average speed of stopping, it is useful to know how any pattern of stop-signal performance can be explained in terms of the underlying processes. This was reason for us to test more than just the effect of violations of the assumptions of the horse-race model. Specifically, we simulated the effect of variance in the primary-task duration, skewness of the distribution of reaction times and speed of stopping, and the failure to trigger the stopping process in a minority of trials.

The effect of the shape of the distributions on the most commonly used dependent variables was negligible. The variability of the primary-process duration, on the other hand, turned out to play an important role for the reliability of several inhibition measures. An increased variability makes it possible to estimate the speed of stopping over a wider range of stop-signal delays, so that a poor choice of delays can do less harm. At the same time, however, this variability decreases the accuracy of measurements, so that more trials or more subjects are required before an hypothesis can be tested.

The triggering rate of the inhibitory process was the only factor that we simulated, that affected the estimation of the summary speed of stopping and distorted the linearity in the midrange of the inhibition function. Of course it is possible to prove such a deficiency, because a subject who is not always able to trigger his inhibitory process would never be able to reach the asymptotic no-response level, even when stop-signals are presented well in advance of the primary task stimulus. However, if a deficiency in triggering the inhibitory process is suspected in a given data set, we do not know a way to correct the estimation of the stop-process duration.

It was shown that there were several factors in the race with comparable effects. For example, the effect of time-dependence was largely similar to the effect of stochastic dependence of RT and $SSRT_{in}$. Furthermore, RT-variability and $SSRT_{in}$ -variability resulted in opposite patterns of results. It was shown for the case of the ZRFT-slope that the similarity of results as an effect of different factors compromises the interpretability.

Study 2 showed that the accuracy of measurements was high for summary scores of $SSRT_{Obs}$, but that accurate assessment of the slope of the inhibition function requires a large number of trials. Given a sufficient number of stop-trials, the contribution of population variance in $SSRT_{in}$ and RT to the statistical power of inhibition scores is substantial. It was shown in Study 3, that the combination of the multiple interpretations of a difference in ZRFT-slope and the high influence of population variance on power makes it practically impossible to differentiate between the triggering efficiency or stop-speed variability of one group and another. The limited power of the ZRFT-slope also holds that there is an increased risk of ascribing differences in inhibitory control to stop-speed, rather than the variability of stop speed or triggering rate of the inhibitory mechanism (e.g. Schachar et al., 1993).

The sensitivity and power of the average speed of stopping was shown to be reasonable. It was demonstrated that an experiment with only one tracked SOA suffices for the estimation of stop speed, and that the number of stop trials only has a limited effect on power. Most importantly, differences in summary scores of stop-speed can only be explained in terms of the true stop-speed, or in terms of a substantial triggering deficiency.

Concluding remarks

The present findings have several implications for the design and interpretation of the stop-signal paradigm. First of all, the simulations show that the data that are collected with the stop-signal paradigm can be explained with the horse-race model of response inhibition, and that its assumptions are not challenged. Furthermore, clear advantages of a response-rate tracking procedure over fixed-SOA and RT-tracking were shown for the registration of stop-speed and the inhibition function. Unfortunately, the model does not yet have a method that can correct the inhibition function for inhibition-irrelevant factors, so that other inhibitory deficiencies than slow stopping can not be demonstrated. This implies that the rising interest in (a) more subtle forms of control (e.g. De Jong et al., 1995), (b) more fine-grained aspects of inhibitory control (e.g. Kramer et al., 1994) and (c) more concealed deficiencies of inhibitory control (e.g. Schachar & Logan, 1990b), should be gratified with converging measures. Possibly, methods from psychophysiology can complement the merits of the stop-signal paradigm.

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6 a General Discussion and Summary (English)

Throughout this dissertation, the people who performed in the experiments were referred to as subjects. However, the *Publication Manual of the American Psychological Association, 4th ed.* (APA, 1995, p. 49) warns psychologists that the word “subject” is impersonal, and recommends the word “participant” instead. The use of “subjects” in the passive voice suggests that individuals are acted on instead of being actors. Although this recommendation is not followed in the literature or in this dissertation, the arguments to do so deserve to be considered.

Without an instruction and motivation, a child who sees a clown jumping from a box on a computer screen is not likely to press a button on the side of stimulation. In this respect, the subject’s compliance is required for any experiment. However, this dissertation focuses on the subject’s role in the experiment at a more subtle level. For example, when a child is engaged in the task to respond on the side of the clown, the sudden onset of a tone on one ear causes a natural tendency to respond on that side. Because the child wants to comply with the instruction to give a fast response, but to strive for high accuracy, the response tendency that is induced by the tone needs to be tamed. In general, subjects are thought to be able to control their performance, for the improvement of speed or accuracy (Rabbitt, 1979).

The efficiency and point of impact for top-down control were investigated with choice reaction time tasks that were supplemented with factors that were helpful for a fast and correct response, or factors that interfered with the speed and accuracy of response processes. Chapter 2 tested the efficiency of response inhibition of children between the age of 5 and 12 years, and adults. The stop speed was surprisingly fast for children in comparison with adults. Although there was a slight decrease of stop-signal reaction time, this development was substantially more moderate than the development on other latencies. A second stop task instructed subjects to withhold the response that was in preparation, and execute a response with the opposite hand. The stop speed in this condition was more susceptible to development than regular stop speed, but again the difference between children and adults was smaller for stop latencies than for a variety of response latencies. For both tasks, an attempt was made to isolate the effect of stop-speed variability on stop performance. Consistent with the findings from Chapter 5, this exercise did not allow strong conclusions. After inhibition-irrelevant differences between groups were compensated for, a counterintuitive pattern remained, that suggested that the stop speed was less variable for children than for adults.

The same subject groups participated in an interference task. It was found that the response to the side of a visual stimulus was delayed by the presentation of an irrelevant tone to the opposite ear (cf., Simon, 1990). This delay is interpreted as an index of the efficiency of suppressing the incorrect response tendency that is induced by the tone. The delay was approximately constant for all age groups, which suggests that children as young as five years could resist interference efficiently. In an immediate-arousal experiment, the subjects received tones of variable intensity while responding to the side of a visual stimulus. Intense stimulation resulted in a faster response, even if the tone was presented quite late. These data were interpreted as support for Sanders’ (1980) model, in which the accessory stimulus results in increased readiness to respond. Furthermore, there was no difference between the age groups in the sensitivity to intense stimulation.

Together, the results in Chapter 2 show that there are large exceptions to the generality of the hypothesis that inhibitory efficiency changes with age (Bjorklund & Harnishfeger, 1990). The suggestion that the development of inhibitory efficiency serves as a unitary explanation of diverse expressions of cognitive development (Dempster, 1992) can not be maintained. The data in Chapter

2 also show exceptions to the global nature of age changes in processing speed as it was described in the global-trend hypothesis (Hale, 1990). Given a generalized effect of age on the speed of all component processes, the stop speed and the effects of the sound intensity and location should be have been larger for younger children. Their reaction times are typically more than twice as long as those of young adults, but the stop latencies were slower by a only fraction. The absence of a generalized pattern of speed changes suggests that there are other component processes than were tested in Chapter 2, that are more sensitive to age effects.

While Chapter 2 described effects of external stimulation after the start of a reaction process, Chapter 3 reversed the order of influence and activity. That is, subjects were instructed to prepare for a task situation on the basis of a prime stimulus. Subsequent differences in the target processing were interpreted in the light of that preparation. In this task, a prime stimulus suggested which response might be needed next, and the color of the prime indicated how reliable that response suggestion was. Then, a 1500 ms interval followed to allow the subject to interpret the information that was conveyed in the prime, and make proper preparations. Finally, a target stimulus was presented, consisting of a color that was mapped onto one of two response hands. The prime could be a valid suggestion of the response to the target on 80 or 50 percent of all trials, so that preparation for the primed response was useful in the former, but not in the latter case. The 20 percent invalid primes could be followed by either a no-go target, or a target that required a response with the nonprimed hand. The experiment allowed an analysis of 1) strategic preparation of target processing, and 2) adaptation and inhibition of incorrect preparation.

Strategic preparation of target processing took place on early processes, but did not lead to activation of the primed response before the target was presented. There was no hand-specific response activation during the foreperiod, as measured with the lateralized readiness potential (LRP). Soon after the presentation of the target, the LRP went in the direction of the primed response if the prime was informative, but not if it was noninformative. The latency of the P3 component reflected a substantial validity effect for informative primes, but a much smaller effect for noninformative primes. The validity effect on indices of motor processes did not show a large increase of the validity effect relative to the effect on P3. Because P3 latency is sensitive to the duration of processes before the start of response activation, a delay of a comparable size is likely to persist on the latency of later processes. Therefore, the absence of a considerable increase in the validity effect on LRP suggests that the strategic preparation effects were confined to processes preceding response activation, and lateralization for the primed hand just after target onset was interpreted as a consequence of preparation on earlier processes such as presetting motor programs.

Chapter 3 contained two task conditions that were designed to parallel the stop-all and stop-change conditions of Chapter 2. It was assumed that a selective form of inhibition of a subset of responses is required in the change condition, whereas no-go conditions allow a nonselective form of inhibition, which is more like freezing all activity (cf. De Jong, Coles, & Logan, 1995). The inhibition on no-go trials was slightly more successful than inhibition on change trials, judging from the overt error rate. However, psychophysiological indices were able to distinguish different patterns of inhibition for no-go and change conditions. There was a high proportion of no-go trials with EMG for the primed hand that did not result in a response, but a low proportion of correct change trials with incorrect EMG. It was concluded from these data that response inhibition could be exerted up till a later level of the reaction process on no-go trials than on the change trials.

In contrast to what De Jong et al. (1995) found for correct change trials, the LRPs for the primed response exceeded an amplitude level that was normally reached preceding an overt responses, but then decreased and changed into an LRP for the correct response. De Jong et al. interpreted the LRP level that precedes overt responses as a threshold level for the transmission of a command from central motor processes to executing structures. In this interpretative framework, the

presence of suprathreshold LRP for the primed response in combination with a low proportion of incorrect EMG trials suggests that inhibition on change trials was exerted between the level that is reflected in LRP, say response activation, and the level that is reflected in EMG, say response execution. This is in contrast with De Jong et al.'s (1995) hypothesis that response changes have to take place at a central level before or during the production of LRP.

Finally, the no-go LRP did not exceed the alleged threshold level. In contrast, De Jong et al. (1995) found that the LRP on correct stop trials did reach the threshold. Apparently, the activation of the primed response on no-go trials was already cancelled at an early level in a way that is comparable to freezing all responses. The same early inhibition may not have been possible on change trials because these trials required activation of an alternative response and the activation can not be cancelled selectively. However, on change trials it was possible to reduce activation for the primed response by activating a competing response. This type of reduction is not necessarily a direct effect of executive control. For example, there may be mutual inhibition between motor processes of the left and right hand.

The present findings are consistent with Logan and Cowan's (1984) model of executive control. In this hierarchical model, a higher-order mechanism monitors performance in subordinate processes and makes adjustments wherever necessary. A nonselective stop requirement is met by cancelling the support for subordinate response processes. This form of control can be exerted with high speed because the command has privileged access to the subordinate systems. Subjects are able to inhibit successfully just before the response, as was supported by the present EMG results. Logan and Cowan suggest that the reaction process can also be cancelled with high speed at earlier levels. This is supported by the attenuated LRP on no-go trials.

In contrast, Logan and Cowan (1984) argue that signals (i.e., the targets) that require an overt response that is sufficiently different from the response to the first signal (i.e., the prime) must wait for the subordinate processes to deal with them. Because the lower-level processes do not give precedence to changes in the response requirements, the success of inhibition in the change conditions depends on the interaction of competing response processes. As a result, response inhibition on stop-change trials is only successful if the change signal is presented relatively early. The estimated stop speed on change trials is therefore lower than that on stop trials, and the LRP amplitude remains small (cf., De Jong et al., 1995).

For prime-target sequences, the success of selective response inhibition is enhanced by a relatively late onset of reaction processes for the primed response. This is confirmed by the high error rate on change trials on fast response bins. If the primed response was not activated until the target was presented, the competition between the primed response and the correct response could result in an effective replacement of the response. In summary, the results fit Logan and Cowan's (1984) model, but are not entirely consistent with De Jong et al.'s (1995) distinction between central and noncentral inhibition.

Chapter 4 deals with the issue of temporal overlap between cognitive processes. In previous research it has been shown that response preparation can start on the basis of partial information about the stimulus, before the perceptual evaluation of the stimulus is finished (e.g., Miller & Hackley, 1992; Osman, Bashore, Coles, Donchin, & Meyer, 1992). This finding supports the existence of parallel processing and continuous transmission of processing output, which is a problem for serial stage models. However, the overlap between perceptual analysis and response preparation may be an exception, given the dual-task results that indicate that there are structural limitations to simultaneous processes. An important feature of the perceptual processes that have been shown to overlap with response preparation is that they all consisted of relatively simple analyses. In Chapter 4 the process of response preparation was investigated under the cognitive burden of a mental-rotation task. Character stimuli could be mirrored or normal, and could be

rotated over a variable angle relative to upright. The identity of a character was mapped onto a hand, and a response with that hand was required if the character was mirrored, but not if the character was normal. Because a mirror/normal judgement can not be made before the stimulus is mentally rotated to the upright position, there was a variable amount of time between the moment that the hand could be determined, and the moment that the go/no-go decision could be made. The presence of a small amount of motor preparation for the correct hand (LRP) indicated that subjects were able to make a separate translation of the identity and the mirror/normal status. This implies that information about the stimulus can be transmitted in parts (cf. Miller & Hackley, 1992; Osman et al., 1992), and that the response-preparation process could take place in parallel with mental rotation. However, the LRP was attenuated by the mental-rotation requirement. This conclusion was supported by a comparison between the LRP on the mental-rotation task, and a task without such a burden, that was designed to match the delay between the hand and go/no-go information.

If the LRP on no-go trials would not be accepted as sufficient evidence for hand selection, it could be argued that there was no activation of the correct hand because there was no identity-to-hand translation during mental rotation. This alternative explanation could be rejected, because responses to a probe stimulus that was presented during mental rotation were facilitated by the hand that was indicated by the identity of the rotated character. These results converge on the interpretation that it was possible to base the response decision on the identity of the character stimulus during mental rotation, but that there were limitations to the ability to activate this response. Either the execution was postponed in favor of the demanding rotation process, or there were structural limitations that caused the rotation process to delay response preparation.

The interference of mental rotation with response activation extends the findings of dual-task studies. It is shown in dual-task paradigms that the reaction to a second stimulus is delayed by processes that are engaged in the response to a first stimulus. While dual-task paradigms (cf. Pashler, 1993) have shown that the overlap of two *separate* response processes is structurally limited, the mental rotation experiments lead to the conclusion that there are limits on the ability of two processes of the *same task* to overlap in time. There is reason to consider the possible effects of such overlap for the stop-signal paradigm, because the stop-signal paradigm has similarities with dual-task paradigms. Both paradigms require the simultaneous interpretation of two stimuli. Logan and Cowan's (1984) hierarchical model posits that the executive control of responses is not involved in such structural limitations. But what would be the consequence of a violation of this assumption? This question was answered in Chapter 5.

In Chapter 5, the performance on the stop-signal paradigm was simulated with computerized data to test the strengths and weaknesses of this paradigm. The stop-signal paradigm is typically based on an RT task. A small proportion, say 25%, of all response stimuli is followed by an auditory signal that instructs the subject that the response on that trials should be withheld, if possible. Performance on this task can be described by the chance of inhibition as a function of the stimulus onset asynchrony (SOA) between the response stimulus and the stop signal. By itself, this inhibition function does not allow conclusions about the efficiency of stopping. However, the performance on the stop task can be interpreted with the help of the horse-race model (Logan & Cowan, 1984), that says that the stop process and the response process engage in an independent race. If the stop process finishes faster than the response process, the response is withheld, otherwise it is executed. This means that the finishing time of the stop process can be calculated from the combination of the chance of inhibition, and the distribution of RTs on trials without a stop signal. Because the starting time of the stop process is under the experimenters control, the total duration of the stop process can be calculated.

The calculations are all based on two important assumptions. First, the speed of the response process and the stop process are assumed to be independent. This assumption has received some

credibility from a comparison between a stop-change task and a dual-task (Logan & Burkell, 1986). These experiments showed that successful *inhibition* of the primary response does not cause a refractory effect for the changed response. However, the accidental *execution* of the primary response caused a delay of the secondary response that was comparable to the refractory effect on a dual-task. The simulations illustrated that a violation of the independence assumption causes substantial changes in the slope of the inhibition function. However, empirical inhibition functions do not deviate much from the data that were found with simulations of zero dependence. Violations of the independence assumption also affected the estimation of stop speed, although the estimation was not affected at the SOA where 50% of all responses was inhibited. Logan and Cowan (1984) described how the observed speed of commission errors on stop trials can be compared to the speed that would be predicted by the horse-race model. This comparison has often been interpreted as a test of the independence assumption of the race model (e.g., Logan & Burkell, 1986). However, the simulations revealed that this measure is sensitive to variability in the stop process, while it is virtually insensitive to violations of the independence assumption.

Second, the speed of the response process is assumed to remain unaffected by the presence of a stop process. It has been shown (e.g., Logan & Burkell, 1986) that an increase in the proportion of stop signal trials stimulates a more conservative strategy of the subject, leading to a longer RT on the primary task. However, overall changes in RT do not affect the measurement of the stop latency. It is important, however, to select SOAs that are well adjusted to mean RT and mean stop speed. Because these speeds can change during the experiment, the SOA should move along with the changes. In the simulations, the staircase tracking algorithm for adjustment of SOAs (Osman, Kornblum, & Meyer, 1986) yielded more reliable results than a fixed-SOA procedure.

The simulations showed that it is possible to estimate the speed of stopping reliably within a test session of reasonable duration. Although there are several methods that lead to an accurate estimation, there are some with poor results. An important notion is that the speed of stopping should be estimated at a point in the inhibition function where the chance of inhibition is approximately 50%. The stop speed at this point is found to be robust against a variety of influences, such as the speed distribution of the stop- and response-process, and SOA-dependence or primary-task dependence of the stop speed.

Of course, the speed of stopping is not the only defining characteristic of the efficiency of response inhibition. If an experimenter wants to test a subject or diagnostic group for inhibitory deficiencies, it is also important to know whether the stop process always works, and whether it works with a consistent speed. Logan and Cowan (1984) have illustrated that the inhibition function contains more information than just the speed of stopping, and that there is a way to isolate the contributions of other stop-related factors. They showed that the contribution of differences in primary task characteristics, the SOA and the speed of stopping can be removed. What remains is a normalized inhibition function, that is thought to contain only the contribution of trial-by-trial differences in stop performance. By comparing the slope of the normalized inhibition function between a test group and a control group, it is possible to detect differences in inhibitory efficiency.

The sophisticated calculations that Logan and Cowan proposed have, for example, been used by researchers (Schachar, Tannock, & Logan, 1993) who are interested in the control processes of children with Attention Deficit Hyperactivity Disorder (ADHD). They found that the speed of stopping of children with pervasive ADHD was lower than that of normal children. Furthermore, they found that the normalized inhibition function for ADHD children did not have a different slope than the inhibition function for control children. This led to the conclusion that the inhibition process of ADHD children was not more variable or less likely to be triggered. However, simulations in Chapter 5 showed that the correction procedure of the inhibition function is not flawless. The slope of the normalized inhibition function is affected to the same degree by

variability in the response speed or dependence between the stop and response processes, as it is affected by the variability of stop speed. This implies that it is not possible to draw strong conclusions from differences or the absence of differences in the slope of the inhibition function.

A related problem is that several sources of variance in the stop task contribute to the variance in the diagnostic tools to detect differences in the variability of the stop process. As more sources are included in the calculation of the dependent variable, the power to show differences between groups in the efficiency of inhibitory control is further decreased. Calculations based on group homogeneity, sample size and effect size have shown that it is workable to compare the speed of stopping, but the detection of differences in the slope of the normalized inhibition function requires too many subjects.

In conclusion, the simulations showed that stop speed can be estimated with several methods with a reasonable accuracy. However, there is no measure that is sensitive to stop-speed variability without being sensitive to inhibition-irrelevant factors. It is therefore recommended to invest experimenting time in replications of stop trials with an adaptive latency that tracks the SOA that corresponds with 50% inhibition. If that is done, a violation of the independence assumption does not affect the estimation of the stop speed.

This dissertation has contributed to the knowledge and methodology of response activation and inhibition, as well as the development of these functions. I believe that a reliable method for the assessment of executive control can become a tool in the diagnosis of behavioral disorders such as ADHD, and can help understand the deficiencies that underlie these disorders. In addition to these direct applications, I believe that a fundamental understanding of the response-execution process is one of the more manageable goals for cognitive neuroscience. But, of course, there is still much work to be done.

6 b Algemene Discussie en Samenvatting (Nederlands)

In dit proefschrift is consequent het woord 'subject' gebruikt voor de mensen die deelnamen aan de experimenten. De *Publication Manual of the American Psychological Association, 4th ed.* (APA, 1995, p. 49) waarschuwt echter dat het woord 'subject' in een artikel onpersoonlijk overkomt, en raadt aan het woord 'deelnemer' te gebruiken. Het woord 'subject' in de passieve vorm suggereert dat het experiment op individuen wordt uitgevoerd, terwijl ze in werkelijkheid zelf het experiment uitvoeren. Hoewel deze aanbeveling niet in de literatuur of in dit proefschrift wordt opgevolgd, verdienen de argumenten om dat wel te doen nadere aandacht.

Zonder instructie of motivatie, zal een kind dat een clown uit een doos ziet springen op een computerscherm niet geneigd zijn een knop in te drukken aan de kant van de stimulus. In dat opzicht is de medewerking van de proefpersoon in elk experiment vereist. Dit proefschrift benadrukt echter de rol van de proefpersoon op een subtieler vlak. Bijvoorbeeld, als het zelfde kind eenmaal bezig is met de taak om aan de kant van de clown te reageren, dan zal de plotselinge aanbieding van een toon aan één oor een neiging oproepen om aan die kant te reageren. Als het kind dan gehoor wil geven aan de instructie om snel maar accuraat te reageren, dan zal die neiging moeten worden getemd. In het algemeen zijn proefpersonen in staat hun prestaties te beheersen om snelheid en accuratesse te verbeteren (Rabbitt, 1979).

De efficiëntie en het aangrijppunt van controle-effecten zijn onderzocht met keuze-reactietijdtaken, waaraan factoren zijn toegevoegd die een snelle en correcte respons konden ondersteunen of juist verstoren. In Hoofdstuk 2 werd de efficiëntie getest van de responsinhibitie van kinderen tussen de leeftijd van 5 en 12 jaar, en van volwassenen. De stopsnelheid was verrassend hoog bij kinderen in vergelijking met volwassenen. Hoewel er een lichte afname van de stop-sigitaal reactietijd werd gevonden, was deze ontwikkeling aanzienlijk zwakker dan de ontwikkeling op andere latenties. Een tweede stop-taak instrueerde proefpersonen om een respons in voorbereiding tegen te houden, en met de tegengestelde hand een respons uit te voeren. De stopsnelheid in deze conditie was gevoeliger voor ontwikkeling dan de gewone stopsnelheid, maar wederom was het verschil tussen kinderen en volwassenen kleiner voor stoplatenties dan voor diverse responslatenties. Voor beide taken werd geprobeerd het effect van de variabiliteit van stopsnelheid op de stopprestaties te isoleren. Zoals te verwachten was op basis van de bevindingen in Hoofdstuk 5, stond deze poging geen krachtige conclusies toe. Nadat was gecorrigeerd voor inhibitie-irrelevante verschillen tussen groepen, bleef een onwaarschijnlijk patroon over dat suggereert dat stopsnelheid bij kinderen standvastiger is dan bij volwassenen.

De zelfde proefpersoongroepen namen deel aan een interferentietaak. Er werd gevonden dat een respons aan de kant van een visuele stimulus vertraagd werd door de presentatie van een irrelevante toon via het tegengestelde oor (vgl., Simon, 1990). Deze vertraging wordt geïnterpreteerd als een maat voor de efficiëntie bij het onderdrukken van de incorrecte responstendens die door de toon wordt opgeroepen. De vertraging was ongeveer constant over leeftijd, hetgeen suggereert dat kinderen van vijf effectief weerstand kunnen bieden aan interferentie.

In een immediate-arousal experiment kregen proefpersonen tonen van variabele intensiteit aangeboden, terwijl ze aan de kant van een visuele stimulus moesten reageren. Intense stimulatie had een snellere respons tot gevolg, zelfs wanneer de toon vrij laat werd aangeboden. Deze data werden geïnterpreteerd als steun voor Sanders' (1980) model, waarin een toegevoegde stimulus een toename van de responsbereidheid tot gevolg heeft. Verder werd tussen de leeftijdsgroepen geen verschil gevonden in de gevoeligheid voor intense stimulatie.

Gezamenlijk laten de resultaten van Hoofdstuk 2 zien dat er aanzienlijke uitzonderingen zijn op het algemene karakter van de hypothese dat de inhibitoire efficiëntie verandert met leeftijd (Bjorklund & Harnishfeger, 1990). De suggestie dat de ontwikkeling van inhibitoire efficiëntie als één verklaring van verscheidene uitingen van cognitieve ontwikkeling kan dienen (Dempster, 1992) is niet houdbaar. De data in Hoofdstuk 2 laten ook grote uitzonderingen zien op het algemene karakter van leeftijdsveranderingen in verwerkingssnelheid, zoals beschreven in de globale-trend hypothese (Hale, 1990). Aangenomen dat er een algemeen effect van leeftijd is op de snelheid van alle deelprocessen, zou de stopsnelheid en het effect van geluidsintensiteit en locatie groter moeten zijn voor jonge kinderen. Hun reactietijden zijn normaal gesproken twee keer zo lang als die van volwassenen, maar de stoplatenties waren slechts een fractie trager. De afwezigheid van een gegeneraliseerd patroon van snelheidsveranderingen geeft aan dat er andere procescomponenten zijn dan getest in Hoofdstuk 2, die gevoeliger zijn voor leeftijdseffecten.

Terwijl Hoofdstuk 2 de effecten beschreef van externe stimulatie na de start van het reactieproces, werd de volgorde van beïnvloeding en activiteit in Hoofdstuk 3 omgedraaid. Dat houdt in dat proefpersonen werd geïnstrueerd om zich voor te bereiden op een taaksituatie op basis van een prime-stimulus. De veranderingen in targetverwerking werden vervolgens geïnterpreteerd in het licht van die voorbereiding. In deze taak gaf de prime-stimulus aan welke respons mogelijk gevraagd zou worden, en de kleur van de prime gaf aan hoe betrouwbaar die suggestie was. In de daaropvolgende interval van 1500 ms werden de proefpersonen in de gelegenheid gesteld om de prime-informatie te interpreteren en om de gepaste voorbereidingen te treffen. Uiteindelijk werd een target-stimulus gepresenteerd, bestaande uit een kleur die hoorde bij een van de twee respons handen. De prime kon een valide suggestie bieden over de respons op de target op 80, dan wel 50 procent van alle trials, waardoor voorbereiding voor de geprimeerde respons in het eerste geval wel, maar in het tweede geval niet nuttig was. De resterende 20 procent invalide primes konden worden gevolgd door een no-go target, of een target die om een respons met de niet-geprimeerde hand vroeg. Het experiment stond een analyse toe van 1) strategische voorbereiding van de verwerking van target-stimuli, en 2) aanpassing en inhibitie van onjuiste voorbereiding.

Strategische voorbereiding van de targetverwerking werd gevonden op vroege processen, maar dit leidde niet tot activatie van de geprimeerde respons voordat de target werd gepresenteerd. Er was geen hand-specifieke responsactivatie gedurende de voorperiode, zoals gemeten met de gelateraliseerde responsbereidheidspotential (lateralized readiness potential, LRP). Kort na de presentatie van de target sloeg de LRP in de richting van de geprimeerde respons wanneer de prime informatief was, maar niet wanneer deze noninformatief was. De latentie van de P3-component gaf een aanzienlijk validiteitseffect voor informatieve primes weer, maar een veel kleiner effect voor noninformatieve primes. Het validiteitseffect op maten van motorische verwerking liet geen grote toename zien van het validiteitseffect in vergelijking met het effect op P3. Omdat P3-latentie gevoelig is voor de duur van processen die voorafgaan aan het begin van responsactivatie, ligt het voor de hand dat een vergelijkbaar effect nog meetbaar is op latere processen. De afwezigheid van een noemenswaardige toename van het validiteitseffect op de LRP geeft aan dat de effecten van strategische voorbereiding beperkt bleven tot processen voorafgaand aan responsactivatie. De lateralisatie voor de geprimeerde hand net na de targetaanbieding werd geïnterpreteerd als een gevolg van voorbereiding op vroegere processen zoals het klaarzetten van een motorprogramma.

Hoofdstuk 3 bevatte twee taakcondities die ontworpen waren naar analogie met de stop-alles- en de stop-wissel-conditie uit Hoofdstuk 2. Er werd aangenomen dat in de wisselconditie een selectieve vorm van inhibitie nodig is, terwijl no-go condities een nonselectieve vorm van inhibitie toestaan, die meer lijkt op het 'bevrozen' van alle handelingen (vgl. De Jong, Coles, & Logan, 1995). De inhibitie op no-go trials was iets succesvoller dan de inhibitie op wisseltrials, te oordelen naar observeerbare fouten. Echter, psychofysiologische indices konden verschillende

inhibitiepatronen onderscheiden voor no-go- en wisselcondities. Er was een groot aantal no-go-trials met EMG voor de geprimeerde hand die niet tot een respons leidden, en slechts een klein aantal correcte wisseltrials met incorrecte EMG. Uit deze data werd geconcludeerd dat responsinhibitie kon worden uitgevoerd tot aan een later niveau in het reactieproces van no-go-trials dan van wisseltrials.

In tegenstelling tot wat De Jong c.s. (1995) vonden voor correcte wisseltrials, overschreed de LRP voor de geprimeerde respons een niveau dat normaal alleen werd bereikt voorafgaand aan een observeerbare respons, maar daarna nam deze weer af en veranderde in een LRP voor de correcte respons. De Jong c.s. interpreteerden het LRP-niveau vlak voor een respons als een drempelwaarde voor de transmissie van een motorcommando van centrale motorische processen naar uitvoerende processen. Binnen dit interpretatiekader betekent de aanwezigheid van LRP boven het drempelniveau, in combinatie met een lage hoeveelheid incorrecte EMG, dat inhibitie op wisseltrials werd uitgeoefend tussen het niveau dat met LRP wordt gemeten, zeg responsactivatie, en het niveau dat met EMG wordt gemeten, zeg responsuitvoering. Dit staat in contrast met De Jong c.s.' (1995) hypothese dat responswisselingen plaats moeten vinden op een centraal niveau voor of tijdens de productie van LRP.

Tot slot bereikte de no-go-LRP niet het veronderstelde drempelniveau. De Jong c.s. (1995) vonden echter dat de LRP op correcte stoptrials dit niveau wel bereikte. Het lijkt er op dat de activatie van de geprimeerde respons op no-go-trials al op een vroeger niveau werd onderbroken, op een manier die vergelijkbaar is met het 'bevrozen' van alle responsen. Een zelfde vroege inhibitie was wellicht niet mogelijk op wisseltrials omdat deze trials de activatie van een alternatieve respons vereisten, terwijl de activatie niet selectief kan worden stilgelegd. Echter, op wisseltrials was het mogelijk om de activatie voor de geprimeerde respons te reduceren door een strijdige respons te activeren. Dit type reductie is niet noodzakelijkerwijs een direct gevolg van responsbeheersing. Zo kan er bijvoorbeeld wederzijdse inhibitie tussen motorprocessen van de linker- en rechterhand bestaan.

De huidige bevindingen zijn in overeenstemming met Logan en Cowan's (1984) model van responsbeheersing. In dit hiërarchische model houdt een hoger mechanisme de prestaties van ondergeschikte processen in de gaten en past deze aan waar dat nodig is. Aan een eis om nonselectief te stoppen kan worden voldaan door de ondersteuning van ondergeschikte responsprocessen te onderbreken. Deze vorm van beheersing kan met hoge snelheid worden uitgeoefend, omdat het commando geprivilegieerde toegang heeft tot ondergeschikte systemen. Proefpersonen zijn in staat nog vlak voor de respons succesvol te inhiberen, zoals wordt ondersteund met de huidige EMG-resultaten. Logan en Cowan suggereren dat het reactieproces ook al met hoge snelheid kan worden onderbroken op een vroeger niveau. Dit wordt ondersteund door de gedempte LRP op no-go-trials.

Aan de andere kant beargumenteren Logan en Cowan (1984) dat signalen (d.w.z., de targets) die een observeerbare respons vereisen die voldoende afwijkt van de respons op het eerste signaal (d.w.z., de prime), moeten wachten totdat ondergeschikte processen ze kunnen verwerken. Omdat de lagere processen geen voorrang geven aan veranderingen in de vereiste respons, hangt het succes van inhibitie op wisselcondities af van de interactie tussen strijdige responsprocessen. Als gevolg daarvan is responsinhibitie op stop-wisseltrials alleen succesvol als het wissel signaal relatief vroeg wordt aangeboden. De geschatte stopsnelheid op wisseltrials is daardoor lager dan op stoptrials, en de LRP amplitude blijft klein (vgl. De Jong c.s., 1995).

Voor prime-targetsequenties wordt het succes van selectieve responsinhibitie vergroot door een relatief late start van het reactieproces voor de geprimeerde respons. Dit wordt bevestigd door het hoge foutenpercentage op wisseltrials in snelle responsbins. Als de geprimeerde respons niet werd geactiveerd totdat de target werd gepresenteerd, dan kon de competitie tussen de geprimeerde en de

correcte respons ertoe leiden dat de respons effectief werd verwisseld. Samengevat passen de resultaten op Logan en Cowan's (1995) model, maar zijn ze niet geheel in overeenstemming met De Jong c.s.' (1995) onderscheid tussen centrale en niet-centrale inhibitie.

Hoofdstuk 4 heeft betrekking op de discussie rond de overlapping in tijd van cognitieve processen. In voorgaand onderzoek is aangetoond dat responspreparatie kan beginnen aan de hand van gedeeltelijke informatie over de stimulus, nog voordat de perceptuele evaluatie van de stimulus is afgerond (b.v., Miller & Hackley, 1992; Osman, Bashore, Coles, Donchin, & Meyer, 1992). Deze bevinding ondersteunt het bestaan van parallelle verwerking en continue overdracht van de tussenresultaten, hetgeen een probleem vormt voor seriële stadiamodelen. De overlapping tussen perceptuele analyse en responspreparatie kan echter een uitzondering zijn, gegeven de resultaten van dubbeltaak die aangeven dat er structurele beperkingen bestaan voor simultane verwerking. Een belangrijke eigenschap van de perceptuele processen waarvan overlapping met responspreparatie is aangetoond, is dat ze allen bestonden uit relatief eenvoudige analyses. In Hoofdstuk 4 werd de responspreparatie onderzocht onder de cognitieve last van een mentale-rotatietaak. Letterstimuli konden gespiegeld of normaal zijn, en konden zijn geroteerd over een variabele hoek ten opzichte van de rechtopstaande positie. De identiteit van een letter was gekoppeld aan een respons-hand, en de respons met die hand was vereist als de letter gespiegeld was, maar niet als deze normaal was. Omdat het onderscheid tussen gespiegeld en normaal slechts kan worden beoordeeld nadat de letter mentaal naar de rechtopstaande positie is geroteerd, was een variabele tijd beschikbaar tussen het moment waarop de respons-hand bekend werd en het moment waarop de go/no-gobeoordeling kon worden gemaakt. De aanwezigheid van een kleine hoeveelheid motorische voorbereiding voor de correcte hand (LRP) gaf aan dat proefpersonen in staat waren een afzonderlijke vertaling uit te voeren van de identiteit en het al of niet gespiegeld zijn. Dit betekent dat informatie over de stimulus in gedeelten kan worden overgedragen (vgl. Miller & Hackley, 1992; Osman c.s., 1992), en dat responspreparatie parallel met mentale rotatie kan plaatsvinden. Echter, de LRP werd gedempt door de noodzaak tot mentale rotatie. Deze conclusie werd gesteund door een vergelijking tussen de LRP op de mentale-rotatietaak en een taak zonder een dergelijke last, die was ontworpen met een even groot tijdsinterval tussen hand en go/no-go-informatie.

Als de LRP op no-go-trials al niet zou worden geaccepteerd als voldoende steun voor handselectie, dan zou kunnen worden geopperd dat er geen activatie van de correcte hand plaatsvond omdat de vertaling van identiteit naar hand was uitgebleven tijdens mentale rotatie. Deze alternatieve verklaring kon worden verworpen, omdat responsen op een probe-stimulus die tijdens mentale rotatie werd gepresenteerd gefaciliteerd werden door de hand die hoorde bij de identiteit van de geroteerde letter. Deze resultaten convergeren naar de interpretatie dat het mogelijk was een responsbeslissing te baseren op de identiteit van de letterstimulus tijdens mentale rotatie, maar dat er beperkingen waren aan het vermogen om die respons te activeren. Ofwel de uitvoering werd uitgesteld ten gunste van het belastende rotatieproces, of er bestonden structurele beperkingen die ervoor zorgden dat de preparatie werd vertraagd.

De interferentie van mentale rotatie met responsactivatie breidt de bevindingen van dubbeltaakonderzoek uit. Er is aangetoond met dubbeltaakparadigma's dat de reactie op een tweede stimulus wordt vertraagd door processen die bezig zijn met de respons op de eerste stimulus. Terwijl dubbeltaakparadigma's (vgl. Pashler, 1993) hebben laten zien dat de overlapping tussen twee *afzonderlijke* responsprocessen structureel beperkt wordt, leidden de mentale-rotatieexperimenten tot de conclusie dat er beperkingen zijn aan het vermogen tot overlapping van twee processen van de *zelfde* taak. Er is reden om de mogelijke effecten van een dergelijke overlapping te bezien in het kader van het stop-sigitaal paradigma, omdat het stop-sigitaal paradigma overeenkomsten heeft met dubbeltaakparadigma's. Beide paradigma's vereisen de simultane interpretatie van twee stimuli. Logan en Cowan's (1984) hiërarchische model stelt dat de

beheersing van responsuitvoering geen hinder ondervindt van zulke structurele beperkingen. Maar wat zou de consequentie zijn van een schending van deze aanname? Deze vraag werd beantwoord in Hoofdstuk 5.

In Hoofdstuk 5 werden prestaties op het stop-signaalparadigma gesimuleerd met gecomputeriseerde data, om de sterke en zwakke punten van dit paradigma te onderzoeken. Het stop-signaalparadigma is meestal gebaseerd op een reactietijds taak. Bij een klein deel van alle stimuli, zeg 25%, wordt een auditief signaal aangeboden dat de proefpersoon instrueert om de respons op die trial voor zover mogelijk tegen te houden. Prestaties op deze taak kunnen worden beschreven met de kans op inhibitie als functie van de interval tussen de primaire en de stopstimulus (stimulus onset asynchrony, SOA). Op zichzelf staat deze inhibitiefunctie geen conclusies toe over de efficiëntie van het stoppen. Echter, de prestaties op de stop taak kunnen worden geïnterpreteerd met behulp van het racemodel (Logan & Cowan, 1984), dat inhoudt dat het stopproces en het responsproces een onafhankelijke race aangaan. Als het stopproces eerder finisht dan het responsproces, dan wordt de respons tegengehouden; anders wordt de respons uitgevoerd. Dit betekent dat het moment van finishen van het stopproces kan worden berekend uit de combinatie van de kans op inhibitie, en de distributie van reactietijden op trials zonder stopsignaal. Omdat het moment van starten van het stopproces bepaald wordt door de proefleider, kan de totale duur van het stopproces worden berekend.

De berekeningen van het racemodel zijn gebaseerd op twee aannamen. Allereerst wordt aangenomen dat de snelheid van het responsproces en het stopproces onafhankelijk zijn. Deze aanname is aannemelijker geworden door een vergelijking tussen de stop-wisseltaak en een dubbel taak (Logan & Burkell, 1986). Deze experimenten toonden aan dat succesvolle *inhibitie* van de primaire respons geen refractair effect op de gewisselde respons had. Daarentegen had de onbedoelde *uitvoering* van de primaire respons een vertraging van de secundaire respons tot gevolg die te vergelijken was met het refractaire effect op de dubbel taak. De simulaties illustreren dat een schending van de onafhankelijkheidsaanname aanzienlijke veranderingen in de helling van de inhibitiefunctie tot gevolg heeft. De helling van empirische inhibitiefuncties verschillen echter niet veel van de helling die werd gevonden bij simulaties zonder afhankelijkheid. Schendingen van de aanname van onafhankelijkheid beïnvloedden ook de schatting van de stopsnelheid, hoewel de schatting niet werd verstoord op de SOA waarop 50% van alle responsen werden geïnhibeerd. Logan en Cowan (1984) beschreven hoe de geobserveerde snelheid van commissiefouten op stop trials kunnen worden vergeleken met de snelheid die op grond van het racemodel worden voorspeld. Deze vergelijking is vaak geïnterpreteerd als een test van de onafhankelijkheidsaanname van het racemodel (b.v., Logan & Burkell, 1986). De simulaties lieten echter zien dat deze maat gevoelig is voor variabiliteit in het stopproces, terwijl het vrijwel ongevoelig is voor schendingen van de onafhankelijkheidsaanname.

Ten tweede wordt aangenomen dat de snelheid van het responsproces niet wordt beïnvloed door de aanwezigheid van het stopproces. Het is aangetoond (b.v., Logan & Burkell, 1986) dat een toename van het aantal stop trials tot gevolg heeft dat proefpersonen een conservatievere strategie aannemen, waardoor een langere reactietijd op de primaire taak wordt veroorzaakt. Globale veranderingen in reactietijd beïnvloeden echter niet de meting van de stopsnelheid. Het is dan wel van belang om SOAs te kiezen die goed zijn aangepast aan de gemiddelde stop- en reactiesnelheid. Omdat deze snelheden kunnen veranderen tijdens het experiment zou de SOA mee moeten schuiven. In de simulaties leverde het trapsgewijze-volgalgorithme voor de aanpassing van SOAs (Osman, Kornblum & Meyer, 1986) betrouwbaarder resultaten dan een procedure met vaste SOAs.

De simulaties lieten zien dat het mogelijk is de stopsnelheid betrouwbaar te schatten binnen een testsessie van een redelijke lengte. Hoewel er verschillende methoden zijn die tot een nauwkeurige schatting leiden, zijn er ook enkelen met slechte resultaten. Een belangrijke

opmerking is dat stopsnelheid zou moeten worden geschat op een punt op de inhibitiefunctie waar de kans op inhibitie ongeveer 50% is. De stopsnelheid op dat punt bleek robuust tegen diverse invloeden, zoals de verdeling van de stop- en responsnelheid, en de afhankelijkheid tussen de stopsnelheid enerzijds en de SOA of de primaire taak anderzijds.

Natuurlijk is de stopsnelheid niet de enige karakteristiek van de efficiëntie van responsinhibitie. Als een proefleider een proefpersoon of diagnostische groep wil onderzoeken op inhibitorische deficiënties, dan is het ook belangrijk te weten of het stopproces altijd werkt, en of de snelheid ervan redelijk constant is. Logan en Cowan (1984) hebben geïllustreerd dat de inhibitiefunctie meer informatie bevat dan alleen de stopsnelheid, en dat het mogelijk is andere stop-gerelateerde factoren te isoleren. Zij lieten zien dat de bijdrage van verschillen in primaire-taakeigenschappen, de SOA, en de stopsnelheid kunnen worden verwijderd. Wat overblijft is een genormaliseerde inhibitiefunctie, die verondersteld wordt alleen de bijdrage te bevatten van verschillen in stopprestaties van trial tot trial. Door de helling van de genormaliseerde inhibitiefunctie te vergelijken tussen een testgroep en een controlegroep kunnen verschillen in inhibitorische efficiëntie worden gedetecteerd.

De verfijnde berekeningen die Logan en Cowan hebben voorgesteld zijn bijvoorbeeld gebruikt door onderzoekers (Schachar, Tannock, & Logan, 1993) die geïnteresseerd waren in de beheersingsprocessen van kinderen met de aandachtstekortstoornissen en hyperactiviteit (Attention Deficit Hyperactivity Disorder, ADHD). Zij vonden dat de stopsnelheid van kinderen met pervasieve ADHD lager was dan die van normale kinderen. Verder vonden zij dat de genormaliseerde inhibitiefunctie van de ADHD kinderen en die van de controlekinderen niet afweken in helling. Daaruit leidden zij af dat het inhibitieproces van ADHD kinderen niet variabel was of minder goed op gang werd gebracht. Simulaties in Hoofdstuk 5 lieten echter zien dat de correctieprocedure voor de inhibitiefunctie niet vrij is van gebreken. De helling van de genormaliseerde inhibitie wordt in de zelfde mate beïnvloed door variabiliteit in de responsnelheid of afhankelijkheid tussen het stopproces en het responsproces, als door variabiliteit in het stopproces. Dit impliceert dat het niet mogelijk is om sterke conclusies te trekken uit de aan- of afwezigheid van verschillen in de helling van de inhibitiefunctie.

Een gerelateerd probleem is dat verschillende variantiebronnen in de stopmaak bijdragen aan de variantie in de diagnostische maten waarmee verschillen in de variabiliteit van het stopproces worden getoetst. Naarmate meer bronnen worden toegevoegd in de berekening van de afhankelijke variabele, neemt de power verder af om verschillen tussen groepen in inhibitorische efficiëntie aan te kunnen tonen. Berekeningen aan de hand van groepshomogeniteit, steekproefgrootte en effectgrootte hebben laten zien dat het haalbaar is om stopsnelheid te vergelijken, maar dat het aantonen van verschillen in de genormaliseerde inhibitiefunctie te veel proefpersonen en metingen vergt.

Concluderend laten de simulaties zien dat stopsnelheid kan worden geschat met verschillende methoden met een redelijke accuratesse. Er is echter geen maat die gevoelig is voor variabiliteit in stopsnelheid zonder ook gevoelig te zijn voor inhibitie-irrelevante factoren. Het wordt daarom aangeraden om experimentatietijd te investeren in replicaties van stoptrials met een adaptieve SOA die het punt met 50% inhibitie volgt. Als dat wordt gedaan, dan zal een schending van de onafhankelijkheidsaannname geen invloed hebben op de schatting van de stopsnelheid.

Dit proefschrift heeft een bijdrage geleverd aan de kennis en methodologie van responsactivatie en -inhibitie, zowel als de ontwikkeling van deze functies. Ik geloof dat een betrouwbare methode om uitvoeringsbeheersing in kaart te brengen ook als gereedschap kan dienen bij de diagnose van gedragsstoornissen zoals ADHD, en kan helpen om de onderliggende deficiënties van deze stoornissen te begrijpen. Maar naast deze directe toepassingen geloof ik dat

een fundamenteel begrip van het reactieproces een van de meer hanteerbare doelen is van de cognitieve neurowetenschappen. Maar, uiteraard moet er nog veel werk gebeuren.

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