Control failures, error processing, and cognitive aging

Nieuwenhuis, S.T.

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Chapter 1

Introduction

1.1. Cognitive control

1.1.1. What is cognitive control? Research paradigms

Well, then, what is it?

Cognitive control refers to a set of cognitive processes that "organize" and "oversee" the operation of more specialized cognitive processes. Here, "organization" refers to the ability of our cognitive system to configure itself for the performance of specific tasks through adjustments in perceptual selection, biasing of response selection, and the maintenance of contextual information over temporally extended periods. The idea that specialized "lower-level" processes can only function in accordance with behavioral goals when under control of "executive" control processes has been very influential in psychology. A number of theoretical accounts has been proposed that try to specify the nature of the influence that control processes exert, and that try to characterize the situations in which control processes are called into play (e.g., Baddeley, 1986; Cohen, Dunbar, & McClelland, 1990; Meyer & Kieras, 1997; Norman & Shallice, 1986; for a review, see Monsell, 1996). Among others, these are situations in which the task is novel, in which multiple tasks need to be managed at the same time, or in which information in the environment threatens to trigger an inappropriate action.

"Overseeing" refers to the ability of the cognitive system to monitor the internal and external environment for signals that indicate the demand for increased executive control. For example, upon detecting competing response tendencies, the system may bias goal-relevant "response pathways" in order to minimize such conflicts on subsequent occasions. Monitoring of external feedback signals may also lead to adaptive changes in control settings, such that the system learns to select the appropriate information or actions through interaction with the external environment. Detailed accounts of how the intervention of executive control processes is triggered by the outcome of such monitoring processes are relatively new (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001; Braver & Cohen, 2000; see also Schneider & Detweiler, 1987).

Executive and evaluative control functions, such as error monitoring, interference control, task-set reconfiguration, or context updating, should not necessarily be considered as basic mental functions, supported by specific dedicated neural circuits. They might well be conceived of as emergent functions, being established by the configuration and tailoring of existing subordinate processes in such a fashion that a 'new', unique function emerges. For instance, it has been demonstrated that monitoring for response conflicts might provide a computationally simple approximation to error detection (Yeung, Botvinick, & Cohen, submitted). Thus, error detection might be achieved by the cognitive system without a dedicated error detection function. Likewise, interference control might be accomplished by combining mechanisms of selective attention with the active maintenance of memory representations (Braver & Cohen, 2000). And endogenous task-set reconfigura-
tion (see Chapter 4) might be an emergent property of instruction-driven activation and automatic decay of task sets in memory (Altmann, & Gray, in press). An important task for theories of cognitive control is to identify the basic functional (and neural) mechanisms needed to carry out higher-order control functions and to describe how these basis mechanisms give rise to complex control functions.

Neuropsychology. Frontal lobes and control

The existence of control processes which are dissociable from other, more specialized, cognitive processes has perhaps first been inferred from the pathological failures of control resulting from damage to the frontal lobes. Frontal lesions often result in failures to prevent inappropriate actions, particularly when a task requires the patient to override a habitual response. For instance, some frontal patients are impaired at looking away from, rather than towards, a target stimulus, as is required in the antisaccade task (Fischer & Everling, 1998). A special case of failures to prevent inappropriate actions constitutes a behavioral phenomenon commonly known as "utilization behavior": the inability, often associated with frontal lobe damage, to inhibit action patterns from being triggered by the sight of the object with which they are habitually associated. Other behavioral deficits (see also Monsell, 1996) include the tendency to perseverate, which is manifest in the patient persisting in a now-inappropriate response pattern; failures to voluntarily initiate an appropriate action; distractibility, an impairment in the control of attention; and failures to carry out a specific action despite repeated verbal acknowledgement of the intention to act. These behavioral deficits indicate that damage to the frontal lobes may result in selective impairments not in any specific cognitive or behavioral domain, but rather in the organization and monitoring of a whole range of cognitive skills (see reviews by Duncan, 1986; Shallice, 1988). However, for all the efforts to characterize the behavioral deficits thought to be attributable to "executive dysfunction" in frontal lobe patients, the search for a specific test that is uniquely sensitive to frontal lobe damage has generally been unrewarding (Della Sala, Gray, Spinnler, & Trivelli, 1998); many of the tests often referred to as "frontal" tests appear also sensitive to damage in other areas in the brain.

Experimental psychology. Construct validity

A likely reason for the poor discriminant validity of many "frontal" tests is that they tap executive functions as well as multiple non-executive functions, such that task performance may be disrupted in many ways (see Burgess, 1997; Pennington, Benetto, McAleer, & Roberts, 1996). To arrive at a relatively pure measure of executive function, one can attempt to eliminate the contribution of construct-irrelevant influences through task manipulations and various types of experimental control. Using this isolation approach, experimental psychologists have developed several tasks with the aim of measuring executive processes. For example, many measures have been claimed to measure the ability to inhibit processing of perceptual items and production of responses that are irrelevant to the current goal. These measures include the number of intrusions from external items in free recall, the amount of interference from previously ignored items, the performance cost in detecting targets at a recently attended location, and the famous Stroop effect. However, Rabbitt (1997; 2001) and others have pointed out that the "inhibition" concept has been overextended to cover an implausibly wide range of functions or measures. That is, according to Rabbitt (2001), "... imprecision of the common-language use of the word "inhibition" has encouraged misleading analogies between quite disparate functional processes" (pp. 7; see also McDowd, 1997). Some of these effects may actually arise as a result of a reflexive (or reactive, cf. Logan, 1994) rather than executive, type of
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inhibition, by which we mean a non-intended side effect or residual effect of executing some process that must be overcome by concurrent or subsequent processes. However, even some skills that at a phenomenological level appear to require the intentional, top-down suppression of perceptual information or habitual responses, such as the ability to minimize Stroop interference and the ability to generate saccades away from a sudden onset, can be formally modeled without incorporation of any process that resembles "top-down inhibition" (e.g., Cohen et al., 1990; Kimberg & Farah, 1993; Trappenberg, Dorris, Munoz, & Klein, 2001). This raises questions about the construct validity of a construct such as "top-down inhibition". On the other hand, top-down inhibition may be a valid construct in specific cases, such as the "stop-signal inhibition" process proposed to play a crucial role in withholding a response when necessary (Logan, 1994).

Another aspect of the construct validity of measures of executive processes is addressed by examining the intercorrelations between performance scores on tasks designed to measure the same executive process. Construct validity would be established if different variables constructed to measure the same executive process would correlate significantly with each other but not with variables hypothesized to measure other constructs. Unfortunately, there is not much evidence for this type of construct validity for measures of executive processes, since the correlations among these measures are predominantly low and usually not higher than the correlations with variables hypothesized to measure other types of processes (e.g., Duncan, Johnson, Swales, & Freer, 1997; Rabbitt, 1997). This may even be true for correlations between scores on different versions of the same task. For instance, as described in more detail below, Shilling, Chetwynd, and Rabbitt (in press) gave four different versions of the Stroop task to a group of older participants and found no evidence that a specific participant's amount of Stroop interference in one task version was predictive of this participant's Stroop interference in another task version.

Thus, even with the controlled procedures available to experimental psychologists, it turns out to be difficult to develop a specific task that yields a relatively pure measure of inhibition or other executive processes. Despite many valuable contributions, the neuroscientific approach to the study of executive processes (i.e., studying brain activation patterns as a function of experimental manipulations) is often subject to very similar limitations. As is the case for behavioral measures, differential patterns of brain activity are often directly interpreted in terms of the cognitive processes reflected by these patterns. Nonetheless, the nature of these processes is not rarely inferred on the basis of a rather superficial task analysis, rather than on the basis of a detailed, mechanistic model of processing in that particular task. Without the support of a process model, it is also difficult to draw firm conclusions about whether a brain area activated in an "executive task" implements the actor of executive control or is a target of the effects of control, which is itself exerted elsewhere (see, e.g., Cohen, Botvinick, & Carter, 2000).

Two potential solutions to these methodological and conceptual problems are (1) the development of formal models of executive processes, in which processing principles are made computationally explicit; and (2) the consideration of the possibility -- representing a recurrent theme in this thesis-- that executive processes do not become manifest as a systematic pattern of purported absolute limitations --as measured by average performance on specific "executive tasks"--, but rather as within-task or between-task variability of performance due to repeated failures of control initiation. These two topics will be discussed next.
1.1.2. Models of cognitive control

Verbal models of cognitive control

The first models of cognitive control were rather descriptive by nature; they primarily identified the need for a control mechanism and tried to characterize the situations in which control is recruited (e.g., Baddeley, 1986; Norman & Shallice, 1986; Shiffrin & Schneider, 1977). For instance, Norman and Shallice (1986) proposed a model of executive control based on the production-system architecture. The to-be-controlled entities in this model are called "thought and action schemata", specialized routines for performing individual tasks that can be triggered into action by the presence of appropriate conditions as defined in a database with if-then statements. Multiple schemata may be activated simultaneously by different trigger conditions, suggesting the need for a mechanism to prevent response conflict and errors. Norman and Shallice's model uses a "contention-scheduling" mechanism to help prevent such conflicts. This mechanism operates by means of lateral inhibition between competing schemata, as a result of which the strongest activated schema takes control in a winner-takes-it-all fashion. However, to enable the system to react flexibly, that is in ways other than those defined in the if-then trigger database, Norman and Shallice propose a second, higher-order, level of control called the Supervisory Attentional System (SAS). The SAS has access to the overall goals of a person, and, in novel, dangerous, or highly competitive situations, may intervene by inhibiting or activating the appropriate schemata. In other words, executive control of the SAS is necessary in situations in which the automatic processes of the contention-scheduling mechanism are inadequate with respect to goal-directed behavior.

Computational models of control. Getting rid of the "homunculus"

Although conceptions of cognitive control as the SAS or other powerful unitary control mechanisms as the Central Executive (Baddeley, 1986) have strongly influenced the way of thinking about information processing in general, they do not provide very detailed accounts of the mechanisms by which control processes exert their influence over information processing and the mechanisms by which control is recruited. At a certain cost of generality, some more recent models of control manage to be much more computationally explicit about the principles of control recruitment and intervention. These models are often constructed to account for performance characteristics in a specific task (i.e., rather than in the whole domain of tasks requiring executive processes), although some of these models can, with a minimal amount of adjustments, account for performance across a range of seemingly different tasks (see for example Kimberg & Farah, 1993). Naturally, a detailed discussion of many computational models of control is beyond the scope of this chapter, so below I discuss a few models which illustrate some important computational principles.

A particularly illustrative model for understanding cognitive control in conflict situations is Cohen, Dunbar and Servan-Schreiber's (1990) connectionist model of the Stroop task. The model consists of two partially overlapping, feedforward processing pathways: one for processing color information, and one for processing word information. The two pathways, each modeling one set of S-R mappings, have separate input and intermediate units but converge on the same response units. A stimulus is modeled as a pattern of activation across the input units, which then propagates forwards through the network as a function of the connection weights. A response occurs when the activation of one of the response units reaches a pre-set threshold. The relative strength of the S-R
mappings (i.e., word reading is a habitual response to a presented word whereas naming its ink color is not) is modeled by stronger connection weights in the word-reading pathway. Thus, in the absence of modulatory output, the model responds preferentially to the word input. However, to overcome this tendency in the critical condition of a Stroop experiment, the model incorporates a set of "task demand" units, which are connected to the hidden units in both pathways, and whose contribution is to add additional activation to the relevant (i.e., color naming) pathway. As a result, a signal presented to the color-naming pathway is able to overcome the otherwise dominant response mediated by the word-naming pathway. Moreover, the degree of modulatory input from the task demand units is modeled in a way to ensure that the relative strength of the word-naming pathway is still such that there will be substantial interference at the response unit level -- resulting in the Stroop effect.

In Cohen et al.'s (1990) Stroop model, control is exerted by a module which somehow knows what the relevant task is, and which biases the usual flow of information processing. Note that these are some of the properties of the SAS in Norman and Shallice's (1986) model. However, the specific benefit of Cohen et al.'s computational model is that it can quite accurately simulate a host of benchmark phenomena from the Stroop literature including the well-known pattern of asymmetrical interference and the effect of practice. Another attractive feature of the model is that contextual information (i.e., about the relevant task) is represented in the same way as other information, namely as a pattern of activation across a set of units. Likewise, modulation of processing by this contextual information and other information processing is all accomplished via the same type of pathways. In other words, the "control module" has no special status in the model. Importantly, models with essentially the same computational principles have been successful in simulating performance in other cognitive control tasks such as the Eriksen flanker task (Cohen, Servan-Schreiber, & McClelland, 1992).

Kimberg and Farah (1993) simulated the Stroop effect using a production-system model. Their simulation consisted of two production rules, one for color naming and one for word naming. If, as in the case of a Stroop stimulus, both production rules are activated (as is likely since the two stimulus attributes match with conditions in each of the production rules), the most active production rule is executed. The activation of each production rule is partly determined by its baseline activation, which is set higher for the rule associated with the highly practiced task of word naming, so that in the absence of control input the word-naming rule is always executed. In addition, activation is added from working-memory elements that match with conditions specified in the production rules. These working-memory elements, which themselves gain more activation through their connection with other working-memory elements, consist of stimulus-attribute elements (i.e., the stimulus is in red ink) and goal elements (i.e., name the color attribute). In the critical Stroop condition, control is exerted by strengthening the connection between the relevant stimulus-attribute elements and goal elements. As a result of the strengthened connection, the goal element of word naming will receive more activation from activation of the color-attribute element, and this increase in activation will tend to sufficiently prime the word-naming production rule. Although this model is much simpler and less powerful than the connectionist model discussed above, there is a striking similarity in the conceptual logic of the two models. Furthermore, the simplicity of the production-system model allows it to be easily adjusted for the simulation of a wide range of "executive" tasks (Kimberg & Farah, 1993).

The two models discussed above, and in fact most computational models of control, focus on the nature of the influence exerted by control. They are not explicit about
the way in which control is recruited; the model somehow "knows" that control is required, as for instance on incongruent trials in the Stroop task and Eriksen flanker task. As Botvinick et al. (2001; see also Braver & Cohen, 2000) have argued, the lack of an account of how the intervention of control is brought about is problematic, for it implies a "homunculus" — a little person, residing in the brain, that does all the important decision-making. In order to deal with this homunculus problem, Botvinick et al. have proposed that the cognitive system determines the need for control, at least in part, through monitoring for conflicts in information processing. Such conflicts occur, for instance, when a stimulus affords two competing response tendencies. It is important to note that conflict is not detected by a new homunculus but is instead computed as a simple, multiplicative function of the activation in competing response pathways (see for a more detailed discussion the section 'Error detection or conflict monitoring?' below). Botvinick et al. report a number of simulations in which the conflict signal is utilized by the cognitive system in order to guide the allocation of executive control. For instance, if in the Eriksen flanker task (see Chapter 6 for a description of this task) attention is not sufficiently focused on the central stimulus element, then incongruent flanker elements will tend to activate the incorrect response. If this results in the concurrent activation of two competing responses, the resulting conflict signal will be used to intensify the focus of attention on the subsequent trial. This mechanism --conflict leads to increased control which in turn leads to reduced conflict-- accounts for systematic variations in control, such as the finding that interference effects tend to be smaller on trials following incongruent trials, and the trial-type frequency effect in the Stroop task, that is, the finding that the Stroop effect is reduced if incongruent trials are frequent relative to congruent trials.

1.1.3. Control failures

Absolute vs. probabilistic control limitations. An illustration

De Jong, Berendsen, and Cools (1999) have suggested that many "interference effects" (i.e., the performance cost associated with conflict between competing response tendencies or task sets) that have traditionally been interpreted in terms of absolute limitations to what cognitive control can achieve, may actually be caused by failures to consistently utilize cognitive control when it is needed. I will refer to this theoretical distinction as absolute vs probabilistic control limitations. An interesting illustration of the relevance of this distinction can be taken from the task-switching literature. In the typical version of the task-switching paradigm, subjects switch back and forth between two tasks afforded by the same stimulus. For instance, subjects may be required to alternate between responding to the ink color and word identity of a classical Stroop stimulus (Allport, Styles, & Hsieh, 1994). The robust performance cost that is associated with a change of task, compared to a task repetition, is called the "switch cost". The switch cost is reduced by the opportunity to prepare for the change of task in advance of the task stimulus (Meiran, 1996), but there remains a "residual cost" that resists reduction by further opportunity for preparation (Rogers & Monsell, 1995).

At first sight, this stubborn interference effect of having recently performed a competing task, seems to demonstrate an absolute limitation to the ability to achieve a prepared state by fully endogenous means. Indeed, Rogers and Monsell (1995) have attributed the residual cost to a component of the preparatory control process that can only be initiated by exogenous means (i.e., when triggered by the stimulus). However, on the basis of properties of the RT distribution of trials thought to be associated with a consistent residual cost, De Jong (2000) has proposed a fundamentally different explanation.
According to De Jong, these distribution properties suggest that people do not prepare on a considerable proportion of the trials. Obviously, this hampers performance. But if they prepare and have sufficient time to do so—that is, on the remaining proportion of the trials—RTs are no different than those obtained on task-repetition trials. Thus, according to De Jong, the residual switch cost does not reflect an absolute preparatory limitation, but rather the (probabilistic) contribution of trials on which people do not initiate the preparatory control process. For modeling and empirical results that support De Jong's claim, I refer to De Jong (2000; see also Nieuwenhuis & Monsell, in press; see Chapter 4). Other interference effects that have been attributed, at least in part, to probabilistic control limitations include the Stroop effect (De Jong et al., 1999) and the increased latency of antisaccades (Nieuwenhuis, Ridderinkhof, De Jong, Kok, & Van der Molen, 2000; see Chapter 2).

**Intention activation, goal activation**

Following Duncan and colleagues (Duncan, 1995; Duncan, Emslie, Williams, Johnson, & Freer, 1996), De Jong et al. (1999) proposed an intention-activation account of probabilistic control limitations. According to this account, effective recruitment of cognitive control requires an explicit goal or intention (i.e., to exert control) to be added to the basic goal structure that governs task performance (Duncan, 1995), and retrieval and the carrying out of this intention at the proper time. Failures of this process, which is here referred to as intention activation (Chapter 2) or goal activation (Chapter 3), may result in a behavioral phenomenon called goal neglect, defined by Duncan et al. (1996) as disregard of a task requirement even though it has been understood and remembered. This phenomenon is characteristic of patients with frontal lobe damage, but can, as is argued in Chapter 3 (see also Duncan et al., 1996), also be observed in the normal population. Goal activation failures may also explain performance problems in prospective-memory tasks (Brandimonte, Einstein, & McDaniel, 1996; Duncan et al., 1996), in which subjects are explicitly required to carry out previously formed intentions at a later time.

If probabilistic control limitations are due to failures of a single goal-activation process, we may ask whether it is possible to stimulate this process, for instance by increasing the benefits and reducing the cognitive effort associated with goal activation and maintenance. The answer seems to be 'yes'. In the task-switching paradigm, the estimated probability of failures to engage in advance preparation is reduced by conditions that should minimize cumulative fatigue (De Jong, 2000), and by performance-related incentives (Nieuwenhuis & Monsell, in press; see Chapter 4). The degree of probabilistic control limitations is also reduced substantially by the use of an explicit response deadline (Eenshuistra, Wagemakers, & De Jong, 1999), explicit prompts (Duncan et al., 1996), and instructions (Nieuwenhuis, Broerse, Nielen, & De Jong, submitted; see Chapter 3). This suggests that the purported goal-activation process can also be stimulated by external feedback implicitly or explicitly stressing its importance. These and other factors that may influence goal activation are extensively reviewed and discussed in Chapter 3. For now, it suffices to note that according to the goal-activation account, executive (dys)function should be most apparent in the form of variability of task performance both within and between experimental tasks or task conditions. Within-task variability may arise as a result of occasional goal activation failures. Between-task variability—even between seemingly highly similar tasks—may arise because of task differences in factors influencing goal ac-

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1 Note that De Jong (2000) refers to probabilistic control limitations as "failures to engage" [in advance preparation for a switch of task].
tivation, such as feedback, instruction and other easily neglected procedural and design features.

1.2. Error processing

1.2.1. Types of errors

The importance of cognitive control becomes especially apparent in those instances when performance breaks down. Reason (1990) made a useful distinction between two types of errors: mistakes and slips. That is, people commit errors either because they are not sufficiently aware of the right action to perform (mistake), in which case they may realize that they have made an error only through interaction with the environment; or they commit errors because for some reason they fail to carry out the intended action and instead carry out a wrong action (slip). In case of a mistake, efficient feedback may enable one to learn from one's error and to prevent similar errors in the future. But, of course, even after a long period of learning one may still occasionally experience a "slip of action" (e.g., a "slip of the tongue" despite knowledge of how to pronounce the word or phrase). Experiment 2 in Chapter 6 can be taken as an illustration of the dynamical trade-off between mistakes and slips in the course of learning. Another useful distinction can be made between (1) slips as a result of lapses of attention or intention. (As discussed above, this type of slips is associated with failures of goal activation); and (2) slips as a result of system limitations. One way of evoking such slips is by asking subjects in a choice RT task to respond as quickly as possible. Whereas under normal circumstances, subjects would have had no problems to choose the correct response on each trial, the speed regime requires the subject to respond before his cognitive system has accumulated sufficient information about the presented stimulus and/or required response. It is this type of action slip that constitutes the focus of Chapter 5 and 6 and of most of the research reviewed in the present section.

1.2.2. Theory and database before the error-related negativity was discovered

The existence of an error-monitoring mechanism in the brain has often been assumed, either implicitly or explicitly, in theories of cognition (e.g., Cooke & Diggles, 1984; Levelt, 1989; Norman & Shallice, 1986; Rumelhart, Hinton, & Williams, 1986). However, until the 1990s there was very little evidence for the existence of an error-detection or error-correction mechanism in the brain. The existence of such mechanisms was primarily inferred from the demonstration of rapid error correction following errors of choice in a RT task (e.g., Rabbitt, 1966) and from apparently compensatory behavior following errors, such as a slowing of response RT immediately after an error (Rabbitt, 1966). However, the fact that we are able to correct our errors rather quickly, and are able to adjust our subsequent behavior in an appropriate manner does not necessarily entail the existence of a specific error-detection mechanism. As Gehring and colleagues (Gehring, Goss, Coles, Meyer, & Donchin, 1993) have pointed out, this error-related behavior could occur without the presence of a specialized error-processing system: "The apparent correc-

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2 It is important to note that the two categories of slips are not mutually exclusive; system limitations may be encountered earlier if attention is not focused or the intended action not sufficiently activated.
tion could simply be a correct response produced in parallel with, but more slowly than the error. Furthermore, a response on a trial after an error could be slow because of a persistence of the processing problem that caused the error” (pp. 385).

1.2.3. The error-related negativity (ERN)

*What is it? Phenomenology*

The interest in the existence of a specialized error-monitoring system was highly stimulated by the discovery of a scalp potential that is elicited when human subjects commit errors in choice RT tasks (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring et al., 1993). This EEG potential is especially evident in the ERP waveform, derived by averaging EEG epochs that are time-locked to the erroneous response. It was labeled "error-related negativity" (ERN) by Gehring, Coles, Scheffers, and colleagues (the Illinois group) and "error negativity" (Ne) by Falkenstein and colleagues (the Dortmund group). The ERN is a rather sharp negative potential with maximum amplitude over fronto-central recording sites. Its peak amplitude is reached about 100 ms following the onset of EMG activity leading to an erroneous overt response, but its onset may coincide with the first incorrect EMG activity (Gehring et al., 1993). An ERN may also be observed after partial errors, that is, erroneous response activation that is corrected before it leads to an overt error (e.g., Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). An ERN-like wave may even be observed on correct trials, but Coles et al. (2001) have argued that this "CRN" either represents the influence of stimulus-evoked components in the response-locked ERP, or can be attributed to error processing on correct trials (e.g., when subjects have an incorrect representation of the correct response). Interestingly, an ERN-like negativity has also been reported after feedback signaling that an error was made (Holroyd & Coles, submitted; Miltner, Braun, & Coles, 1997; Nieuwenhuis et al., submitted; see Chapter 6). This feedback-locked ERN peaks around 200-300 ms after the onset of the negative feedback stimulus. Using source localization techniques, several researchers (e.g., Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998) have localized the source of the ERN (response-locked and feedback-locked) in or very near the anterior cingulate cortex (ACC), which is part of the brain's limbic system. Neuroimaging studies (Carter et al., 1998; Kiehl, Liddle, & Hopfinger, 2000) and single-cell recordings in behaving monkeys (Gemba, Sasaki, & Brooks, 1986) have provided corroborating evidence for the activation of the ACC in association with errors.

*The ERN: Antecedent conditions*

The ERN is elicited after errors irrespective of response modality (Holroyd, Dien, & Coles, 1998; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; see Chapter 5) and occurs after both errors of choice (e.g., incorrect response button) and errors of action (i.e., respond when you're not supposed to; Scheffers, Coles, Bernstein, Gehring, &Donchin, 1996). ERN amplitude varies with the person's awareness of the required response (Scheffers & Coles, 2000), but appears to be unaffected by the degree of awareness of the actual erroneous response (Nieuwenhuis et al., in 2001). There is also evidence that the amplitude increases with increasing muscle activity in the incorrect response hand (Scheffers et al., 1996; but see Gehring et al., 1993), and with increasing number of incorrectly chosen response parameters (Bernstein, Scheffers, & Coles, 1995). The Illinois group (e.g., Coles, Scheffers, & Holroyd, 2001) claims that the size of the ERN is positively correlated with the degree of "remedial action" as evident in post-error slowing and immediate error correction, but the evidence for this claim seems to be somewhat limited (Ge-
Gehring et al., 1993; a trend in post-error RT in Scheffers et al., 1996), and discounted by conflicting evidence (e.g., Gehring & Knight, 2000; Nieuwenhuis et al., 2001).

Luu, Collins, and Tucker (2000) have found that ERN amplitude also correlates with certain affective personality measures. At the initial stage of their experiment, ERN amplitudes were larger in subjects who scored high on measures of negative-emotionality and affect, suggesting that ERN amplitude may be related to the degree of distress experienced during error detection. The finding that ERN amplitude is higher in conditions in which instruction emphasizes accuracy over speed (Gehring et al., 1993) could also be taken as evidence for an affective dimension of the ERN (see Luu, Collins et al., 2000), but as will be explained below, there are at least two different, "cognitive" explanations for this finding (Coles et al., 2001; Yeung, Botvinick, & Cohen, submitted). Finally, abnormal ERN amplitudes have been observed in several psychiatric patient groups: higher ERN amplitudes in patients with obsessive-compulsive disorder (Gehring, Himle, & Nisenson, 2000), and lower amplitudes in Parkinson (Falkenstein, Hielscher et al., 2001) and schizophrenia (Ford, 1999) patients. Schizophrenia patients also show a substantial ERN on correct trials. Decreased ERN amplitudes in the context of a substantial "CRN" have also been reported for patients with frontal lesions (Gehring & Knight, 2000) and, but to a lesser extent, for healthy individuals in older age (Band & Kok, 2000; Gehring & Knight, 2000).

Verbal and computational "mismatch" accounts of the ERN

It has been argued (e.g., Bernstein et al., 1995; Coles et al., 2001; Falkenstein et al., 1991) that the ERN reflects a process that compares a representation of a correct response with the actual response. The ERN amplitude reflects the degree of mismatch between these representations. This type of account is usually referred to as the "mismatch" account of the ERN. The representation of the actual response is likely to derive from a central feedback system, since latencies of the ERN onset are too short to allow for external feedback. Obviously, we do not always wait with responding until all possible information about the appropriate response is available; in order to obey speed instructions, we use whatever information is available at the time of the response. However, the mismatch detector makes use of the fact that continued stimulus processing tends to lead to an increasingly reliable representation of the correct response. Motor commands leading to subthreshold incorrect response activation (followed by the correct overt response) may also be "efference copied" to the mismatch detector. This is one of the explanations for the occasional observation of an ERN on correct trials. Furthermore, Coles et al. (2001) assume that the representation of the correct response may also include temporal parameters of the response, for instance when very fast responses are required. This assumption is needed to account for the finding that correct responses that exceed a response deadline are also followed by an ERN-like component in the EEG (e.g., Luu, Flaisch, & Tucker, 2000). Finally, the mismatch signal is hypothesized to form the input to a remedial action mechanism, which is responsible for immediate error correction and strategic adjustments that reduce the likelihood of further errors.

As mentioned above, this (verbal) mismatch account explains why an ERN occurs on incorrect trials but also sometimes on correct trials. It also offers an explanation of the correlation between size of the error and ERN amplitude. The generic nature of the proposed mismatch detector explains why the ERN occurs for several response modalities and types of errors. The relation between ERN amplitude and various other variables (e.g., fatigue, speed-accuracy regime, stimulus degradation) is explained in terms of the effect of these variables on the quality of the representation of the correct response.
A considerably more developed and refined version of the mismatch theory of the ERN has recently been proposed by Holroyd and Coles (submitted). Importantly, their theory provides an integrated account of both response-related and feedback-related ERNs. According to Holroyd and Coles' theory, the basal ganglia learns to predict the expected outcome--in terms of reward and punishment--associated with various events (e.g., stimuli, responses, or stimulus-response ensembles) on the basis of past experience with those events. Violations of these predictions lead to a phasic alteration in firing rate in the mesencephalic dopamine system: a phasic increase if ongoing events are better than predicted, and a decrease if they are worse than predicted. These error signals serve as a reinforcement learning signal that is used by the basal ganglia to improve the quality of its predictions. It does so according to the "temporal difference learning" rule (cf. Braver & Cohen, 2000; Sutton & Barto, 1998; see Chapter 6), a heuristic rule for learning the earliest predictors of future reward or punishment. However, the dopaminergic error signal is also transmitted to the ACC and other brain areas, where it is used to reinforce response settings and stimulus-response mappings.

The ERN is assumed to be associated with the arrival of a "negative" dopaminergic error signal at the ACC. The larger the violation of the prediction, the larger the signal, the larger the ERN. Thus, according to this view, if ongoing events are suddenly worse than expected (e.g., because you emit an incorrect response to a stimulus that usually elicits the correct response), the mesencephalic dopamine system carries a negative error signal--reflecting the size of the error--to the ACC, where it elicits the ERN. In a choice-response task with trial-to-trial feedback, feedback-locked ERNs can be observed after negative feedback when a person does not know the stimulus-response mappings and hence cannot predict the negative feedback on the basis of the response. However, as the person learns the mappings (i.e., knows what response to give to each stimulus in order to obtain positive feedback) the system transmits the negative error signal as soon as it detects that the wrong response has been selected. Holroyd and Coles have formalized their theory in a neural network model which can account for the pattern of human response- and feedback-related ERN amplitudes in a probabilistic learning task and in the Eriksen flanker task (see Chapter 6 for a description of these tasks). Chapter 6 reports a replication of many of these results in younger adults and an application of the model to age differences in ERN amplitudes.

Error detection or conflict monitoring?

It should be emphasized that error detection is not regarded as a function outside the scope of cognitive control -- nor in this thesis nor in the many publications on the ERN. In contrast, it is treated as a special form of monitoring the demand for executive control, and probably the single most important reason why it has received so much attention is the discovery of the ERN. However, not all cognitive control researchers believe that the ERN reflects error detection per se (e.g., Botvinick et al., 2001). Indeed, there is even doubt as to whether the brain houses a specialized system for detecting errors at all!

Cohen and co-workers (Botvinick et al., 2001; Yeung et al., submitted) have recently proposed an alternative theory of the ERN, according to which the ERN is a psychophysiological marker of response conflict. Using a simple connectionist network model of the Eriksen flanker task, Yeung et al. argue that errors are usually associated with conflict in the period following the erroneous response, the time window of the ERN. More specifically, incorrect responses are characterized by the initial activation--as a result of noise or input from flanker stimulus units in incongruent trials--of the response unit associated with the incorrect response. This initial incorrect activation, which leads to the erro-
neous response, is followed by the activation of the correct response unit due to continued processing of the stimulus. Importantly, in the model there is usually a brief period following erroneous responses during which both the correct and the incorrect response units are quite strongly activated. During this period, response conflict, which is defined as the scaled product of the activation of the two response units, is high, resulting in a large conflict signal. This post-error conflict signal is the proposed account of the ERN. Finally, the conflict signal (giving rise to the ERN) is computed in the ACC, an assumption that is consistent with source modeling of the ERN and with a large neuroimaging database supporting the ACC's involvement in evaluating conflict situations (Botvinick et al., 2001).

Yeung et al.'s (submitted) conflict-monitoring model can account for several empirical results, including the relation between the ERN amplitude and speed-accuracy instruction, and the finding that the ERN tends to be larger on congruent than on incongruent flanker trials (Scheffers & Coles, 2000; see also Chapter 6). For instance, a greater emphasis on accuracy is modeled as an increase in the response threshold of the response units and a relative increase in the activation of the input unit associated with central stimulus element (reflecting greater attentional focus). Simulations show that a greater attentional focus leads to more rapid post-error build-up of activity in the correct response unit and hence a larger ERN (consistent with Gehring et al., 1993). Despite the success in re-evaluating the corpus of response-related ERN research in the context of the conflict-monitoring model, a major limitation of the conflict-monitoring model is that it does not explain the feedback-related ERN.

It turns out to be difficult to devise a method for testing between the conflict-monitoring hypothesis and the mismatch hypothesis of the response-locked ERN. The mismatch hypothesis has to assume that the system “knows” that one of the responses is correct, while the conflict-monitoring hypothesis does not require such an assumption, and thus is in a sense more parsimonious. But apart from that, the predictions of the two hypotheses seem very similar. Importantly, proponents of the mismatch hypothesis reason that if there is conflict, then there is activation of the incorrect response, and hence you should observe an ERN. Also, because, unlike the conflict-monitoring account, the mismatch account has not been formalized in its original form, it is hard to determine exact predictions regarding the occurrence, size, and latency of the ERN under various circumstances. But Coles et al. (2001) have proposed a critical test between the two hypotheses. This test involves creating two categories of trials that are equal in the amount of conflict, defined as the product of EMG activation in the two response hands, but different with respect to the correctness of the response. The conflict-monitoring hypothesis would appear to predict that there should be a similar ERN in both categories of trials, but Coles et al. report data showing a larger ERN after incorrect responses, suggesting a special status for errors. However, this argument is based on the nontrivial assumption that EMG is an acceptable single-trial measure of response activation, as featuring in the conflict-monitoring model.

1.2.4. The error positivity (Pe)

The ERN is often followed by a slow positive potential with a centro-parietal scalp distribution. This potential has been labeled "error positivity" and usually extends from around 200-500 ms after the response (e.g., Falkenstein et al., 1991). The Pe has received much less attention than the ERN/Ne, probably because there has been some debate as to whether the Pe may in fact be a delayed stimulus-related P3 visible in the response-locked ERP (see Falkenstein, Hohnsbein, & Hoormann, 1995). If average response
latencies are short (as is the case in many RT experiments), and a stimulus-related positivity is indeed extended on incorrect trials (see Donchin, Gratton, Dupree, & Coles, 1988, for some evidence), then the extended part of the P3 may indeed appear as a positive wave in the response-locked ERP. However, Nieuwenhuis et al. (2001; see Chapter 5) have argued that there is sufficient evidence indicating that the P3 and Pe represent two separate components. Yet, the scalp distribution, timing with respect to a significant event (i.e., an error), and morphology of the Pe are closely similar to those of the P3. This similarity has led several researchers to propose that the Pe and P3 may reflect the same functional and biological process, but one is a reaction to a significant stimulus and the other a reaction to a significant response event (e.g., Falkenstein, Hoormann, Christ, & Hohnbein, 2000; Leuthold & Sommer, 1999).

Not much is known about the functional significance of the Pe. It is not related to immediate remedial action, because it is visible on corrected and uncorrected error trials (Falkenstein et al., 2000). However, as Falkenstein et al. (1991) have originally hypothesized, the Pe seems somehow related to awareness of an error having been made. Initial, indirect support for this hypothesis comes from studies showing that the Pe (but not the ERN) is absent in subjects who are under hypnosis (Kaiser, Barker, Haenschel, Baldeweg, & Gruzelier, 1997), and is strongly reduced following subthreshold (i.e., partial or EMG) errors (Vidal et al., 2000). Nieuwenhuis et al. (2001; see Chapter 5) have more directly confirmed the "awareness hypothesis" by showing that the Pe (but again not the ERN) is reduced or absent after subjectively unrecognized response errors. Interestingly, Nieuwenhuis et al. also found that --like the Pe-- a consistent degree of post-error slowing is present after recognized errors but absent after unrecognized errors. This between-conditions correlation between the presence or absence of the Pe and the presence or absence of post-error slowing led us to suggest that the processes reflected in the Pe, but not the ERN, may be causally related to the activity of a remedial action system.

1.3. Control failures, error processing, and cognitive aging

1.3.1. "Complexity effects" in cognitive aging research

There are specific problems with the interpretation of results from between-group comparisons, such as the comparison between a group of younger and older adults. I will briefly discuss one of these problems before turning to studies of cognitive control in older age.

Aging is associated with a slowing of mean response times and an increase in the variability of mean response times (e.g., Rabbitt, 1993). An increase in task difficulty or complexity due to experimental manipulation increases response times more for older adults than for younger adults. This effect can be nicely demonstrated by means of a Brinley plot, a scatter plot showing mean response time for older adults against mean response time for younger adults for a range of tasks or task conditions which differ in complexity (Brinley, 1965; Ratcliff, Spieler, & McKoon, 2000). Across a wide range of conditions, the mean response times for younger and older adults are generally linearly related, and the function relating the two has a slope around 1.5 and a negative intercept. A slope greater than 1 indicates that each next level of difficulty slows older adults' response times more than it does younger adults' response times, and thus reflects an age-related increase in variability of mean response times (Ratcliff et al., 2000). Furthermore, such Brinley plots indicate that mean response time for older adults can be accurately predicted from
mean response time for younger adults without reference to the specific processes involved in any individual task. Indeed, the finding that response time pairs can often be nicely fitted by a simple one-parameter function has led some researchers to suggest that nearly all of the age differences in response times are attributable to the operation of a single "global slowing" factor (or "generalized slowing" factor; Cerella, 1985; Myerson, Hale, Wagstaff, Poon, & Smith, 1990; see Ratcliff et al., 2000, for a critique).

The Age X Complexity interaction illustrated by Brinley plots has important consequences for the interpretation of age differences in performance on most tasks designed to measure cognitive control functions. To take up the example of "inhibition tasks" once more, in such tasks it is usually expected that older adults display a larger interference (e.g., Stroop) effect than do younger adults. However, the nature of the Age X Complexity interaction will ensure that age will always increase response times in the (more difficult) interference condition more than it will increase response times in the (easier) baseline condition. Thus, across many paradigms used to measure cognitive control functions, evidence for a control deficit is also qualitatively consistent with global slowing (cf. Spieler, 2001). This problem is often dealt with by using parameters of the Brinley function as estimate of global slowing, and then testing whether performance of older adults on individual (e.g., control-related) variables deviates significantly from these parameters. In other words, the Brinley function is taken as null-hypothesis. Although this approach may in many cases be reasonably appropriate and conservative, there are several ifs and buts. For instance, some researchers (e.g., Meyerson et al., 1990) have suggested that the Age X Complexity interaction may be best described by a power function rather than a linear function. To the extent that this is the case, taking a linear function as null-hypothesis will increase the probability of accepting the alternative hypothesis (i.e., disproportional slowing of older adults), especially for tasks with high-difficulty levels. Furthermore, the power of taking the Brinley plot to derive a conservative null-hypothesis is dependent on the quality of the fit of a linear function and of the number of variables on which the fit is based (see Perfect, 1994).

In Chapter 2, an approach is used that circumvents many of these limitations. Using hierarchical regression analysis, the proportion of age-related variance in our variable of interest that is not shared by age-related variance in a variable hypothesized to measure basic speed of processing is examined. As the use of proportional response times, this approach serves to control for global slowing of older adults' response times, but in a way that is more theory-neutral. In Chapter 2, the technique is described in more detail. For an extensive comparison of techniques such as hierarchichal regression analysis and structural equation modeling in the context of developmental and aging research, the reader is referred to Span, Ridderinkhof, and Van der Molen (submitted).

1.3.2. Aging and cognitive control. Issues in interpreting experimental data

In this section, I briefly discuss three (of many) theoretical issues that are of relevance to the interpretation of experimental data regarding age differences in cognitive control.

Specific control deficits versus global slowing

Given the nature of the Age X Complexity effects discussed above, an important question is whether the increased interference effects reported for older adults are the consequence of specific control deficits or should be largely attributed to global slowing. For instance, Salthouse and Meinz (1995) found that age-related variance in the Stroop effect...
largely overlapped the variance associated with measures of processing speed, suggesting that age-related decline in Stroop performance may not reflect decline of inhibitory control mechanisms but rather of basic processing speed. Verhaeghen and De Meersman (1998) reached a similar conclusion in a meta-analytic study of the Stroop effect in aging. Using data from 20 relevant Stroop studies, Verhaeghen and De Meersman found no age differences in the Stroop effect expressed as mean standardized difference. Several other studies have nevertheless reported evidence in favour of a specific-deficit account of age differences in Stroop performance (e.g., Hartley, 1993). The critical issue seems to be the method that is used to assess the source of age differences (see for a discussion McDowd & Shaw, 1999). Brinley analyses, hierarchical regression analyses, and other methods each lead to different conclusions, suggesting that the primary need is for theorists to identify the method that is most suited to study the issue of global slowing versus “local” slowing. For other task domains, there is similar confusion as to whether older adults’ performance deficits are most adequately characterized as originating from specific or general cognitive deficits (see, e.g., McDowd & Shaw, 1999). Of course, the truth may lie somewhere in between with variation in a single cognitive factor being responsible for most of the age-related variance in executive function measures, but age-related variability in other, more specific factors responsible for the remaining “unique” age-related variance -- provided that methods are sufficiently sensitive.

General intelligence (g) as mediator of age effects

Apart from basic processing speed, general intelligence or g has also been proposed as an important mediator of age-related changes in executive task performance. For instance, Rabbitt (1997) reports a study in which a group of more than 600 adults, aged 60-86, was tested on a battery of tests of executive function. The significant though modest (i.e. \( r = .3 \) to .45) correlations between scores on logically similar tests were almost all reduced to non-significance after variances associated on intelligence tests, such as the Culture Fair Test, were partialled out. Thus, in this group of healthy, independent older adults, associations between scores on executive tests could be almost entirely accounted for in terms of their common loadings on intelligence test scores or with their common loadings on Spearman’s g-factor (see Duncan, 1995). Similarly, Shilling et al. (in press), as mentioned above, evaluated younger and older adults’ performance on four different versions of the Stroop task. They found that if individuals were grouped in terms of intelligence test scores, then a particular group’s performance on one of the Stroop task versions was predictive of this group’s performance on the other Stroop task versions. This was not true if individuals were grouped in terms of their biological age. Importantly, intelligence-test scores are also a powerful mediator of age effects on simple tests of memory and information-processing speed (see Rabbitt, 1993). This suggests that a large part of the age-related variance in performance on any cognitive task may be explained in terms of a single cognitive factor -- a factor that is significantly associated with successful performance on general intelligence tests (see Duncan et al., 2000).

Goal activation

Duncan and colleagues (Duncan et al., 1996; 1997) reported that individual differences in g could well account for interindividual variation in a measure of goal neglect obtained in a prospective memory task. On the basis of this finding, they proposed that g may be closely associated with the efficiency of the goal-activation function discussed above. If this is the case, then the finding that general intelligence scores pick up most of the age-related variance in performance on a wide variety of cognitive tasks supports the
hypothesis --central in this thesis-- that the goal activation concept is crucial to understanding age differences in cognitive function (see also De Jong, 2001). The evidence in favor of this hypothesis, suggesting that age differences on many executive tasks are due to probabilistic control limitations that can, in principle, be overcome, will be reviewed extensively in Chapter 3.

1.3.3. Aging and cognitive control. Neurobiological models

In this section, I briefly discuss two, related, neurobiological models of cognitive aging that are especially relevant to the development of cognitive control processes in older age.

The frontal cortex model of cognitive aging

Particular parts of the brain "age" faster than others. The clearest evidence for this relates to age-related changes in the frontal cortex (see for reviews Raz, 2000, Van der Molen & Ridderinkhof, 1998; West, 1996). In older adults, prefrontal cortex shows a greater loss of cortical volume and greater cell loss than other brain areas. These losses have been attributed to either neuronal shrinkage or reduction in synaptic density. In addition, the older brain shows more pronounced decreases in blood flow in prefrontal cortex than in posterior regions of the cortex, as measured with brain-imaging techniques. For example, reduced prefrontal activation has been observed in working-memory tasks and attention tasks. This type of neurobiological evidence has often been cited to support behavioral results of older adults that suggest more pronounced processing impairments on tests thought to rely on the integrity of the frontal lobes (see West, 1996). But we should be wary about this kind of theorizing. First, as discussed above, the construct and discriminant validity of many of these so-called "frontal" tasks is unclear (e.g., Nieuwenhuis et al., submitted; see Chapter 3; Rabbitt, 1997), leaving the possibility that the age differences should be (partly) ascribed to aging of other brain areas. Second, gradually a more sophisticated and differentiated picture of frontal lobe structure and function is emerging in the literature. It may well be that aging affects some frontal structures and functions more than others (see, e.g., Phillips & Della Sala, 1996). And third, there are also many examples of neuroimaging experiments that indicate age-related reductions in frontal-lobe activation that are not more frequent or severe than age-related reductions in activation elsewhere in the brain (for review, see Grady, 2000). Indeed, there are also reports of greater activation in frontal cortex in older than in younger adults. These age-related increases in frontal activation in some (mostly working-memory) tasks have been attributed to increased recruitment of executive control functions to compensate for an age-related loss of efficiency of other cognitive functions (such as memory storage; see, e.g., Reuter-Lorenz et al., 2001). Thus, although the frontal cortex model of cognitive aging is well-supported by several lines of evidence and will continue to play a dominant role in cognitive aging research, the reality is likely to be more complex than is often thought, with differential reductions in older adults' brain activity dependent on the type of task and on other brain areas interacting with frontal cortex.

A dopamine model of cognitive aging

Normal, healthy aging is associated with changes in a variety of neurotransmitter systems, including dopaminergic, cholinergic, and serotonergic systems. The age-related decline in dopaminergic function seems to be best-documented. For instance, Backman et al. (2000) found an age-related deterioration of dopaminergic receptor binding in striatal
structures. Importantly, statistical control of this variable eliminated the age-related variation in performance of a number of cognitive tasks, suggesting that dopaminergic transmission is an important factor in age-related cognitive decline. Furthermore, Kaasinen et al. (2000) reported significant age-related loss of dopamine receptors in various brain areas, especially in the frontal cortex. In monkeys, age-related decreases in neurotransmitter concentration are most pronounced for dopamine in the prefrontal cortex (see Goldman-Rakic & Brown, 1981). Thus, the decline with age in functional efficiency of prefrontal cortex may be partly mediated by reduced efficiency of the dopaminergic system, in prefrontal cortex and in brain areas that interact with prefrontal cortex.

Given the important role of dopamine in cognitive control processes (see, e.g., Braver & Cohen, 2000; Cohen & Servan-Schreiber, 1992), it is not surprising that recently there have been some attempts to relate cognitive-control inefficiency in older adults to changes in dopaminergic function. For instance, Braver et al. (in press) used the Braver and Cohen model of the AX-CPT task to generate predictions about the performance of older adults in this task. The results were consistent with a theory posing that older adults are impaired at the updating and active maintenance of context information. In the Braver and Cohen model, these processes are driven by dopaminergic reinforcement learning signals, and in similar models (Cohen & Servan-Schreiber, 1992) degradation of context representations has been modeled by a model parameter corresponding to the neuromodulatory effects of dopamine in prefrontal cortex. Also, Nieuwenhuis et al. (submitted; see Chapter 6) have recently shown that the age-related decline in learning rate, and in response- and feedback-related ERN amplitudes in a probabilistic learning task can be modeled by manipulation of a single parameter in Holroyd and Coles’ (submitted) model of error processing. This parameter corresponds to a weakening of the dopaminergic reinforcement learning signal which is used to select and reinforce adaptive behaviors. Finally, Li and Lindenberger (1999) have reported a set of computational simulations that relate aging-induced deterioration of catecholnergic systems (among which the dopaminergic system) to several benchmark phenomena of aging, such as increases in mean response time and interindividual variability of response times, and the dedifferentiation of ability structures (see Li and Lindenberger, 1999).

Because dopamine theories are relatively new in cognitive aging research, it remains to be seen to what extent these theories will become subject to similar criticisms as frontal-lobe theories of cognitive aging. However, a virtue of dopamine theories is that they do not focus on a single structure in the brain, but instead place emphasis on the interaction, via dopaminergic pathways, between brain structures. Furthermore, although the neuromodulatory effects of dopamine may be similar in different parts of the brain, the functional consequences of such effects may be quite different in each context. If it turns out that many cognitive aging phenomena can be explained by a single neurobiological system, it seems feasible that such a system will, like the dopamine system, be complex both in structure and function.

1.3.4. Aging and error detection

Virtually nothing is known about possible age differences in error-related processing. In choice-reponse tasks and other rather simple tasks such as the Eriksen flanker task, older adults detect and correct as many of their errors as young adults (Falkenstein, Hoormann, & Hohnsbein, 2001; Rabbitt, 1979). Despite the absence of age differences at the behavioral level, several studies have reported reduced response-related ERN amplitudes for older adults (Band & Kok, 2000; Falkenstein, Hoormann et al., 2001; Gehring &
Knight, 2000). Falkenstein, Hoormann et al. (2001) emphasized that older adults' other ERP components generally showed no reduction in amplitude (see also Chapter 6). Furthermore, an analysis of ERN measures in single-trial epochs, led these authors to conclude that "the [Ne] amplitude effect [of age] may be slightly enhanced by a larger latency variance in the elderly, but it is certainly not due to or enhanced by the absence of the Ne in the elderly in some of the trials, since the amplitude variance was lower in the elderly than in the young" (pp. 261). For lack of a real explanation of the ERN amplitude effects, Falkenstein and colleagues conclude that their data suggest an alteration of error detection in the elderly. Likewise, Band and Kok conclude that older adults discerned a smaller proportion of their errors than younger adults. However, the relation between ERN amplitude and subjective recognition of an error may not be as straightforward as is often assumed. Chapter 6 offers a "neurocomputational" explanation for age differences in response- and feedback-related ERN amplitudes. As mentioned above, it is proposed that dopaminergic reinforcement learning signals, corresponding to a phasic alteration in firing rate of the mesencephalic dopamine system and generating the ERN at their arrival in the ACC, are weakened in older age.

1.4. Outline of thesis. Publications and co-authors

Apart from this introduction (Chapter 1) and a short summary (Chapter 7), this thesis consists of five chapters reporting empirical and modeling work (Chapter 2-6). Chapters 2, 3, and 4 focus on executive control processes and probabilistic limitations of control. Chapter 2 reports a study of age differences in antisaccade task performance and provides an initial attempt to distinguish between absolute "inhibitory" limitations and failures of intention activation in healthy older adults. In Chapter 3, these results are reviewed together with other aging studies in the field of oculomotor control. This review serves as illustration for the explanatory power of the concept of goal activation in the study of executive function. The "goal activation" theoretical framework is further evaluated on the basis of antisaccade performance results from two DSM-IV groups: first-order schizophrenia patients and patients with obsessive-compulsive disorder. Using modeling of data from two task-switching experiments, Chapter 4 tests an hypothesis regarding probabilistic limitations to the endogenous configuration for a switch of task. Each of these three empirical chapters focuses on one or several experimental factors that may influence the proportion of goal activation failures.

Chapters 5 and 6 deal with the evaluative side of cognitive control and in particular with the monitoring and processing of errors. Chapter 5 tries to further our understanding of the functional significance of the error-related negativity and error positivity by examining how these ERP components are related to awareness that an error was made. The relation between these error-related components and adjustments in response behavior after errors is also discussed. Chapter 6 is concerned with age differences in error processing. On the basis of results from two ERP experiments and simulations using a computational model of error processing, an explanation of age differences in ERN/Ne amplitude is proposed.

The five empirical chapters have each been submitted to or accepted in international psychological journals. They have been inserted in this thesis in their original, submitted or accepted form. In particular to acknowledge the important contributions of several co-authors to each of these articles, I present here a list of references.


