Context processing in older adults: Evidence for a theory relating cognitive control to neurobiology in healthy aging

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ABSTRACT

The present study examines the hypothesis that healthy older adults suffer from an impairment of cognitive control resulting from disturbances in the processing of context. This cognitive impairment is postulated to be directly related to dysfunction of the DA system in PFC that occurs during aging. A connectionist computational model is described which postulates specific mechanisms for the role of DA and PFC in regulating control over cognition. The behavioral predictions of the model are tested in a large sample of older and young adults performing variants of a simple cognitive control task - an AX version of the Continuous Performance Test (AX-CPT). The results suggest that older adults exhibit both performance decrements and, counter-intuitively, performance improvements that are in agreement with predictions made from the model. Taken together, the findings provide support for a theory of cognitive aging which postulates a direct relationship between specific aspects of cognitive impairment (context processing) and underlying neurobiological disturbances (PFC and DA system dysfunction).
INTRODUCTION

There are a number of cognitive and biological changes that appear to occur during healthy aging. At the cognitive level, these changes include declines in functions such as episodic and working memory, attention, and inhibition (e.g., Hasher & Zacks, 1988; Moscovitch & Winocur, 1995; Salthouse, 1990; Spieler, Balota, & Faust, 1996). At the biological level, disturbances have been noted in neuroanatomic structures such as the prefrontal cortex (PFC; e.g., Grady et al., 1998; Raz et al., 1997; Salat, Kaye, & Janowsky, in submission; Shaw et al., 1984), and in neurochemical systems, such as the dopamine (DA) system (e.g., Arnsten, Cai, Steere, & Goldman, 1995; Suhara et al., 1991; Volkow et al., 1998). However, despite accumulating evidence about these biological and cognitive changes, there is still little understanding of whether or how they are associated. In particular, we only have a rudimentary understanding of how cognitive disturbances might arise from biological abnormalities, and almost no understanding of how disturbances in different neurobiological systems might interact. The use of formal modeling approaches provides an essential tool in understanding the linkage between neurobiological disturbance and cognitive decline in healthy aging. In our previous work, we have utilized connectionist computational models of cognition to test specific hypotheses regarding the role of DA and PFC in cognitive control (Braver, Barch, & Cohen, 1999b; Braver & Cohen, in press; Cohen, Braver, & O'Reilly, 1996). By cognitive control, we refer to the ability to use internally represented information as a means of guiding and regulating thoughts or behaviors. We have suggested that a central component of cognitive control is the ability to properly maintain and update internal representations of task-relevant context information, and that this ability is critical for attention, inhibition and working memory functions.

In the present study, we show how this modeling approach can also be used to generate novel hypotheses and empirically testable predictions regarding both the cognitive and neurobiological changes that occur in healthy aging. Specifically, we describe a computational model of cognitive control, and discuss how it provides an explicit account of: 1) normal cognitive
control function and; 2) the breakdown in cognitive control that ensues from dysfunction of the DA system in dorsolateral (DL)-PFC. We discuss specific predictions that arise out of the model regarding behavioral performance in healthy older adults. We then present a study that empirically tests these predictions in a large sample of healthy young and older adults performing a simple task of cognitive control – an "AX" version of the Continuous Performance Test (AX-CPT). Before turning to the model, we first review evidence concerning cognitive and neurobiological changes that occur in healthy aging, focusing on cognitive control function, and the PFC and DA systems. We suggest that a wide range of age-related impairments in cognitive control may, in fact, be due to a single fundamental deficit in the ability to properly represent and maintain task-relevant context. We further suggest that these cognitive declines might be due to disturbances in the functional interactions between the PFC and DA systems, which serve as the neural mechanisms underlying context representation and maintenance.

**Cognitive Impairments in Healthy Aging** A large literature on cognitive function in healthy aging suggests that older adults display deficits in multiple different cognitive domains: episodic memory, working memory, inhibition, attention, and "executive" function. Deficits in episodic memory are among the most prominent cognitive deficits found in studies of healthy aging (Craik, 1977; Moscovitch & Winocur, 1992). The episodic memory tasks which appear to show the most severe age-related declines are those involving free recall (Craik & Jennings, 1992), temporal order memory (Parkin, Walter, & Hunkin, 1995), source memory (Spencer & Raz, 1995), and release from proactive inhibition (Dobbs, Aubrey, & Rule, 1989). Interestingly, many researchers have suggested that these types of memory tasks all involve the integration of the outputs of long-term memory with relevant contextual information or strategic cues (Moscovitch & Winocur, 1995; Perfect, 1997). Within working memory, age-related deficits have been consistently observed in both span tasks that involve "on-line" storage and manipulation of information (Craik, Morris, & Gick, 1990; Salthouse, 1990; Verhaeghen & Salthouse, 1997), and in tasks that require active maintenance and monitoring of previous responses, such as the Self Ordered Pointing Test (SOPT; Daigneault & Braun, 1993). Older adults also appear to have difficulty in tasks that
involve suppressing the influence of irrelevant information, or in inhibiting unwanted responses (Hasher & Zacks, 1988; Zacks & Hasher, 1997). For example, healthy older adults display deficits on a number of tasks which are thought to measure inhibitory function, including negative priming (Hasher, Stoltzfus, Zacks, & Rypma, 1991; McDowd & Oseas-Kreger, 1991; Stoltzfus, Hasher, Zacks, Ulivi, & Goldstein, 1993; Tipper, 1991) and stop-signal paradigms (Kramer, Humphrey, Larish, Logan, & Strayer, 1994; May & Hasher, 1998). A fourth cognitive domain which appears to be vulnerable to the effects of aging is in attentional control. In particular, age-related declines are observed in both selective attention tasks, such as the Stroop (Brink & McDowd, 1999; Panek, Rush, & Slade, 1984; Spieler et al., 1996; West & Baylis, 1998; West & Bell, 1997) and in sustained attention or vigilance tasks (Filley & Cullum, 1994; Parasuraman, Nestor, & Greenwood, 1989). Finally, older adults commonly display deficits on tasks designed to measure "executive" function such as the Wisconsin Card Sorting task (WCST; Fristoe, Salthouse, & Woodard, 1997; Kramer et al., 1994; Parkin & Lawrence, 1994), and dual-task paradigms (Brouwer, Waterink, Van Wolffelaar, & Rothengatter, 1991; Jensen & Goldstein, 1991; Korteling, 1993). These tasks require that attentional resources be frequently shifted or divided, or that strategies be flexibly changed as situational demands dictate.

With such a range of tasks from putatively different cognitive domains showing age-related deficits, one interpretation is that healthy aging involves multiple different types of cognitive dysfunction. However, it is also possible that one or more common underlying mechanisms may lead to deficits in multiple tasks domains. For example, Salthouse has proposed a processing speed theory, arguing that a decrease in the speed with which many processing operations can be executed leads to age-related declines in a wide variety of cognitive domains (Salthouse, 1996). The advantage of such a unifying theory is that it is parsimonious, providing a common framework in which to integrate the diversity of findings on cognitive changes in healthy aging. However, the specific mechanisms underlying a potential change in processing speed in healthy aging are unclear, and the relationship of processing speed changes to underlying neurobiological factors has not yet been specified.
Another prominent class of theories which make closer contact with neurobiology, are the so-called "PFC theories of aging" (Moscovitch & Winocur, 1995; Perfect, 1997; West, 1996). The primary idea of these theories is that the pattern of cognitive deficit observed in healthy older adults is remarkably similar to the neuropsychological profile found in patients with known lesions to PFC. Thus normal aging is associated with a decline in PFC function. However, this class of theory does not specify what particular cognitive mechanisms are subserved by PFC, nor how disturbances to these cognitive mechanisms might translate into the pattern of performance impairments found in older adults.

In our work, we have suggested that there is a common element to many of the tasks which appear to be dependent upon the integrity of PFC. Namely, that they require the internal representation and maintenance of context information in the service of exerting control over behavior. For example, in the Stroop task, the context provided by the task instructions must be actively represented and maintained to bias attentional allocation and response selection towards the ink color dimension of a visually presented word. Consequently, we suggest that at least a subset of age-related deficits in working memory, inhibition, attention, and executive function reflect the disturbance of a single underlying processing mechanism that is central to cognitive control. We further suggest that this processing mechanism is housed within the DL-PFC and is regulated by the DA system. In the following sections we argue that disturbances in DL-PFC and the DA system occur in healthy aging, and that these disturbances result in the particular pattern of cognitive impairments observed in older adults.

PFC and DA Disturbances in Healthy Aging Healthy older adults typically show deficits on neuropsychological tests sensitive to PFC damage (Moscovitch & Winocur, 1995; Perfect, 1997; West, 1996). Studies using brain imaging and neuroanatomical techniques have provided more direct support for this hypothesis. For example, studies of gross brain volume have found that although there is a general reduction in brain volume appearing after age 60, the degree of reduction appears to be greatest, and appear earliest, in the frontal cortex (Haug & Eggers, 1991; Salat, Kaye, & Janowsky, 1999). A recent MRI study focusing on cortical gray matter showed that
gray matter in PFC was significantly more affected by aging than other cortical regions (Raz et al., 1997). More detailed analyses have attributed these losses of volume to either neuronal shrinkage or reduction in synaptic density (Huttenlocher, 1979; Peters, Morrison, Rosene, & Hyman, 1998a; Peters et al., 1996; Scheibel, Lindsay, Tomiyasu, & Scheibel, 1975). Moreover, one recent study has shown that in primates, specific synaptic loss within dorsolateral regions of PFC correlates with age-related cognitive impairment (Peters, Sethares, & Moss, 1998b). Studies of resting-state regional cerebral blood flow (rCBF) in aging have also implicated frontal disturbances. rCBF measurements have consistently documented the presence of hypofrontality in older adults (Gur, Gur, Orbist, Skolnick, & Reivich, 1987). More impressively, in a 4-year longitudinal study of healthy older adults, the only significant reduction in rCBF was found in PFC (Shaw et al., 1984).

Functional neuroimaging studies involving cognitive activation may have the most sensitivity for detecting disturbances of PFC function in healthy aging, and relating these to cognitive deficits. Although cognitive neuroimaging studies of healthy aging are still in their infancy, the most prominent age-related changes observed involve abnormal activation of PFC. For example, reduced prefrontal activation has been observed in memory and attentional tasks (Grady et al., 1998; Madden et al., 1997; Schacter, Savage, Alpert, Rauch, & Albert, 1996). Most recently, a study of working memory and aging found that older adults showed abnormal activity in DL-PFC related to the active maintenance of information over increasing delays (Grady et al., 1998).

Normal aging is also associated with changes in neurotransmitter function, in a variety of systems, including dopaminergic, cholinergic, serotonergic, and adrenergic systems (Martin & Rubin, 1997). In non-human primate studies, a common finding is that age-related decreases in neurotransmitter concentration are most pronounced for DA in PFC (Goldman-Rakic & Brown, 1981). These age-mediated reductions in DA transmission in PFC have also been related to changes in cognitive performance. For example, Arnsten has found in a number of studies that pharmacological agents enhancing DA system function can improve working memory performance in aged monkeys (Arnsten, 1993; Arnsten, Cai, Murphy, & Goldman-Rakic, 1994; Arnsten et al., 1995). In humans, disturbances to DA function have also been observed in healthy aging. In a
postmortem study, a significant correlation was found between age and DA receptor concentration (de Keyser, De Backer, Vauquelin, & Ebinger, 1990). More direct evidence for DA disturbances in PFC was observed in an in vivo study using positron emission tomography (PET). In this study, prefrontal DA receptor binding potential was decreased by 39% in older adults (Suhara et al., 1991). A more recent PET study has linked age-related DA decreases directly with cognitive decline in tests sensitive to PFC function (i.e., WCST, Stroop) (Volkow et al., 1998).

The Functional Roles of DA and DL-PFC

Taken together, these findings are consistent with the primary hypothesis of this proposal: that healthy aging is associated with disturbances in DA and DL-PFC function. These findings, however, beg the question of what are the specific roles of DA and DL-PFC in cognition? The PFC is widely thought to play a central role in the control of thought and behavior. The findings are so well-accepted that in the clinical literature, the term 'frontal syndrome' refers to a particular impairment in which the normal control over social and sexual behavior is dysregulated (Hecaen & Albert, 1978; Stuss & Benson, 1986). Neuropsychological studies have demonstrated that patients with PFC lesions show impairments on tasks involving cognitive control, such as the Stroop, WCST, and SOPT. Neurophysiological work with behaving primates has enabled the development and testing of specific hypotheses regarding PFC function. In these studies, it has been found that neurons within PFC exhibit sustained, stimulus-specific activity during the delay periods of simple tasks requiring the active maintenance of task-relevant information, such as in the delayed response paradigm (Fuster, 1989; Goldman-Rakic, 1987).

In the last decade, neuroimaging studies have provided a means by which to directly examine PFC activity in healthy humans. Numerous studies have now replicated the findings from the animal and neuropsychological literature, by demonstrating activity in PFC during a wide range of cognitive control tasks (Baker et al., 1996; Carter, Mintun, & Cohen, 1995; D'Esposito et al., 1995; Prabhakaran, Smith, Desmond, Glover, & Gabrieli, 1997), especially tasks involving working memory (Cohen et al., 1994; Grasby et al., 1993; Jonides et al., 1993; Petrides, Alivisatos, Evans, & Meyer, 1993). More recently, neuroimaging research has also confirmed that DL-PFC is...
specifically involved in active maintenance functions, by demonstrating sustained activity in this region during the maintenance period of working memory tasks (Cohen et al., 1997; Courtney, Ungerleider, Keil, & Haxby, 1997).

A number of studies have also examined the functional role of DA projections to DL-PFC, although these have been less frequent. Neurophysiological evidence suggests that DA appears to alter the responsivity of target neurons to both excitatory and inhibitory afferents (Chiodo & Berger, 1986; Penit-Soria, Audinat, & Crepel, 1987). It has been clearly demonstrated that DA has significant effects on neuronal activity in DL-PFC of behaving primates (Sawaguchi, Matsumara, & Kubota, 1990a). Moreover, when DA activity is pharmacologically manipulated, either locally or systemically, changes have been noted both in delay-related PFC activity and in behavioral performance on tasks requiring active maintenance (Brozoski, Brown, Rosvold, & Goldman, 1979; Sawaguchi & Goldman-Rakic, 1994; Sawaguchi & Goldman-Rakic, 1991; Sawaguchi, Matsumura, & Kubota, 1990b). These findings with regard to behavioral performance have also been observed in humans, with DA antagonists impairing performance on cognitive control tasks (Luciana, Collins, & Depue, 1995; Magliozzi, Mungas, Laubly, & Blunden, 1989), while DA agonists lead to behavioral improvements (Luciana, Depue, Arbisi, & Leon, 1992; Servan-Schreiber, Carter, Bruno, & Cohen, 1998). Thus, the literature to date is fully consistent with the hypotheses that DL-PFC subserves active maintenance functions in cognitive control, and that the DA projection to DL-PFC serves to modulate this process.

A Computational Model of PFC and DA Function in Cognitive Control Despite strong recent interest in DA and DL-PFC, both in the normal and aging literatures, there has been limited explicit theorizing regarding the mechanisms by which they subserve aspects of cognitive processing. There is a consensus that DL-PFC plays a role in the active maintenance of task-relevant information, but the mechanisms by which this occurs have not been specified, and there has been virtually no theorizing about the function of DA, other than to assume that it supports the memory functions of DL-PFC. To develop more explicit theories of DA and DL-PFC function, we have drawn upon computational modeling as a tool for specifying the mechanisms by which these
systems may influence cognition. We have constructed our models within the parallel distributed processing (PDP), or "neural network" framework, allowing us to quantitatively simulate human performance in cognitive tasks using principles of processing that are similar to those believed to apply in the brain (McClelland, 1993; Rumelhart & McClelland, 1986). Thus, information is represented as graded patterns of activity over populations of simple units, processing takes place as the flow of activity from one set of units to another, and learning occurs through the modification of the connection strengths between these. From one perspective, such models are highly simplified, capturing brain-style computation, without necessarily committing to the details of any particular neural system or sub-system. However, with appropriate refinement, such models offer the opportunity to build bridges between our understanding of the low-level properties of neural systems, and their participation in higher level (system) behavior. Along these lines, we have begun to refine our models of cognitive performance, taking account of increasingly detailed information at the neurobiological level.

Our modeling work, has led us to propose three related hypotheses (Braver et al., 1999b; Braver & Cohen, in press) : 1) A central function of DL-PFC is the representation and maintenance of context information (information necessary to select task-appropriate responses); 2) DA activity regulates this function, by gating the access of information into DL-PFC, so that only task-relevant context will be actively maintained; and 3) a reduction of DA effects in DL-PFC results in both weaker representation of context information (since access is partially blocked) and greater decay of maintained context information over time (since information is more susceptible to the interfering effects of noise and task-irrelevant inputs). Each of these hypotheses has begun to receive empirical support in behavioral and neuroimaging studies of normal function. For example, detailed aspects of behavioral data from healthy adults performing a simple cognitive control task (the AX-CPT, described below) under a number of different conditions can be fully captured by the model (Braver et al., 1999b). Additionally, performance of the AX-CPT results in a pattern of DL-PFC activity dynamics that was directly predicted by the model (Barch et al., 1997; Braver et al., 1999b).
One important insight that has emerged from this work is that three cognitive functions that are often treated as independent — attention (selection and support of task-relevant information for processing), active memory (on-line maintenance of such information), and inhibition (suppression of task-irrelevant information) — can all be understood in terms of a single mechanism responsible for the processing of context, operating under different task conditions. When a task involves competing, task-irrelevant processes (as in the Stroop task), it is often assumed that a dedicated inhibitory function is responsible for suppressing, or overriding these irrelevant processes. However, in our models, there is no dedicated mechanism for inhibition. Rather, context representations accomplish the same effect by providing top-down support for task-relevant processes, allowing these to compete effectively against irrelevant ones. In contrast, when a task involves a delay between a cue and a later contingent response, it is usually assumed that a memory function is involved. Once again, there is no dedicated mechanism for this function in our models. Rather, the mechanism used to represent context information is used to maintain task relevant information against the interfering, and cumulative effects of noise over time. Thus, both for tasks that tap "inhibition" and for those that tap "memory," the same mechanism is involved; it is simply a matter of the behavioral conditions under which it operates (i.e., the source of interference) that lead us to label it as having an "inhibitory" or a "memory" function. Furthermore, under both types of conditions, context representations serve an attentional function, by selecting task-relevant information for processing over other potentially competing sources of information. In all circumstances, the same mechanism is involved. When applied to the study of cognitive aging, this perspective suggests that many age-related cognitive deficits can be explained in terms of a disturbance in a single underlying mechanism. In the next section, we describe specific predictions of the model regarding cognitive disturbances hypothesized to occur in older adults during performance of a simple cognitive control task, the AX-CPT.

Model Predictions Regarding Cognitive Control Impairments in Healthy Aging We have conducted an extensive examination of our model's ability to adequately capture essential features of cognitive control within the context of a simple but informative paradigm, known as the AX-CPT
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(Cohen, Barch, Carter, & Servan-Schreiber, 1999; Servan-Schreiber, Cohen, & Steingard, 1996). A primary feature of the AX-CPT is that it enables selective examination of context representation and maintenance. In this task, sequences of letters are presented one at a time, as a series of cue-probe pairs (see Figure 1). The object of the task is to make a target response to an X (probe) but only when it follows an A (cue), and a nontarget response in all other cases. Performance in this task relies on the representation and maintenance of context information, insofar as the correct response to X depends upon the cue stimulus (A or not-A). In our model, PFC is specialized for representing and maintaining the context provided by the cue stimulus. The DA system regulates the access of this context information to DL-PFC, thus providing flexible updating plus interference protection. Context processing function in the AX-CPT can also be examined through specific behavioral performance measures in different task conditions.

In the AX-CPT, target (AX) trials occur with high frequency (70%). This results in two effects: 1) a prepotent tendency to make a target response to an X probe; and 2) a strong expectancy to make a target response following an A cue. Consequently, context information must be used to override or inhibit the tendency to false alarm on BX trials, in which the X probe is preceded by a non-A cue ("B" refers to any non-A cue). In contrast, the context information creates the tendency to false alarm on AY trials, in which an A cue is followed by a non-X probe ("Y" refers to any non-X probe). Consequently, the integrity of context processing can be examined not only through performance on AX target trials, but also through an examination of performance on nontarget trials. In particular, the integrity of context representation can be selectively examined through the relationship of AY to BX performance. If context representations are intact, AY performance should be worse than BX performance (in terms of both errors and RT). Conversely, if context representations are impaired, BX performance should be worse than AY performance. Performance on AX target trials should also be poorer if context processing is impaired, since determination of targets is dependent upon the context provided by the cue. However, a third type of nontarget trial, BY, provides a useful internal control, since in this condition the influence of context on performance should be relatively small (given that both the cue and the probe always
map to a nontarget response). The integrity of context maintenance can also be selectively examined in this paradigm in terms of the effect of the cue-probe delay duration on AY and BX performance. If context maintenance is intact, BX performance should remain constant or improve at long delays, while AY performance should remain constant or worsen with delay. Conversely, if context maintenance is impaired, BX performance should worsen with delay, while AY performance should improve. Thus, the AX-CPT paradigm produces a set of specific and clearly interpretable behavioral performance measures.

We have conducted a systematic series of simulation studies examining performance of the model on the AX-CPT task (Braver et al., 1999b; Braver & Cohen, 1999). First, we have shown that the model can capture detailed aspects of baseline performance data collected in a normative study of over 200 young adults. In particular, the model captures the relationship between AY and BX performance (i.e., AY > BX, for both errors and RT), and the interaction of these effects with cue-probe delay duration (i.e., AY performance worsens with delay, while BX performance slightly improves). Second, we have also examined the effect simulating a reduction of DA activity in DL-PFC on the behavior of the model. Under these conditions, there is a dramatic, but specific effect on both activation dynamics and AX-CPT performance. In particular, when DA activity is disrupted in DL-PFC, DL-PFC will only be weakly activated by the presentation of context information, and this activation will decay over the delay period. These disturbances in DL-PFC activity dynamics are reflected in performance deficits in the model associated with both context representation and maintenance. For example, there are more BX than AY errors, and this effect becomes amplified with delay (see Figure 2). A similar pattern is predicted for RTs (i.e., more slowing of BX than AY RT, and an amplification of this effect with delay). In contrast, BY performance is similar in the intact and disturbed model. These effects of simulating DA disturbances in DL-PFC represent explicit predictions of the model regarding changes in both brain activation patterns and behavior. Inasmuch as we have argued that DA disturbances in DL-PFC are present in healthy aging, our
model can be used as a tool for hypothesis generation regarding the pattern of brain activation and behavior expected in this population during AX-CPT performance.

We attempted to experimentally test these predictions of the model in a large behavioral study of healthy young and older adults performing the AX-CPT. Three different conditions were performed which differentially examined the effect of context representation and task difficulty on performance. In the baseline condition, participants performed the baseline AX-CPT using a long delay between cue and probe (~5s). In the interference condition, participants performed the AX-CPT with irrelevant distractor stimuli presented during the cue-probe delay interval. This manipulation was expected to increase the difficulty of context representation and maintenance. In the degraded condition, participants performed the AX-CPT with stimuli perceptually degraded, such that they were harder to identify. This manipulation was expected to increase task difficulty in a manner that should not affect context representation and maintenance.

METHODS

This study was conducted as part of a Phase I clinical trial examining the cognitive enhancing effects of an experimental psychoactive agent. The clinical trial was sponsored by the Pharmacia & Upjohn company, and was conducted as a multi-institute study involving the University of Pittsburgh Medical School, Stanford University Medical School, Oregon Health Sciences University, University of Michigan, and University of California, Davis. All data presented in the current study were collected as part of a pre-drug baseline testing session.

Participants

Participants in this study were 175 young adults (age range 18-39), and 81 older adults (age range 65-85). No females were studied in the young adult group because of concerns regarding the effects of the experimental drug on females of child-bearing age. Approximately equal numbers of men and women were included in the older adult group. Participants were recruited through advertisements from the communities surrounding each participating institute.
Informed consent was obtained in accordance with the institutional review board, and a cash payment was given in return for participation.

Inclusion criteria for all participants included: a) normal or corrected normal (20/30) vision; b) Mini Mental State Exam over 27 (Folstein, Folstein, & McHugh, 1975); c) Vocabulary subtest standardized score of 8 or higher on the Wechsler Adult Intelligence Scale - Revised (WAIS-R) IQ test; and d) at least 5 years of formal education. In addition, participants were excluded for: a) non-English native language; b) positive urine screen for any Schedule I substances; c) a lifetime history of psychiatric disorders and/or substance dependence or any substance use disorder within six months of testing (DSM-IV criteria); e) evidence of dementia (DSM-IV criteria); f) history or evidence of any neurologic disorder of head trauma or other sensory, motor, or medical problems that could affect cognition or performance. Focused contrasts indicated that the young and older participants did not differ on education level. The older adults scored slightly, but significantly, lower on the WAIS-R Vocabulary test. The demographic characteristics of both participant groups are shown in Table 1.

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Insert Table 1 about here

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Tasks and Apparatus

Participants performed three conditions of the AX-CPT: baseline, interference, and degraded. In all three conditions, sequences of letters were visually presented one at a time in a continuous fashion on a computer display (see Figure 1). Subjects were instructed to make a positive response on target trials and a negative response otherwise. Target trials were defined as a cue-probe sequence, in which the letter A appeared as the cue, and the letter X appeared as the probe. The remaining letters of the alphabet served as invalid cues and non-target probes, with the exception of the letters K and Y, which were excluded due to their similarity in appearance to the letter X. Letter sequences were presented in pseudorandom order, such that target (A-X) trials occurred with 70% frequency, and non-target trials occurred with 30% frequency. Non-targets were divided evenly (10% each) among the following trial types: "B-X" trials, in which an invalid
cue (i.e., non-A) preceded the target; "A-Y" trials, in which a valid cue was followed by a non-target probe (i.e., non-X); and "B-Y" trials, in which an invalid cue was followed by a non-target probe.

Stimuli were presented centrally, for a duration of 300 msec, in 24 point uppercase Helvetica font. A delay of 4900 msec occurred between the presentation of cue and probe stimuli. The intertrial interval was 1000 msec. To increase task difficulty, subjects were instructed to respond to both cue and probe stimuli, pressing one button for targets and another button for nontargets (cues were always considered nontargets). Responses were recorded on a specially constructed button box connected to the computer which recorded response choice and reaction time with 1 millisecond accuracy. For right handed individuals, responses were made with the middle (non-target, middle button) and index (target, right button) fingers of the right hand. For left handed individuals, responses were made with the middle (non-target, middle button) and index (target, right button) fingers of the left hand. Subjects were allowed a total of 1300 msec from stimulus onset in which to respond. Responses which were slower than this limit were not recorded, and elicited feedback (a "bloop" sound) as a prompt to increase speed. The tasks were run on Apple Macintosh computers, using PsyScope software for stimulus presentation and data collection (Cohen, MacWhinney, Flatt, & Provost, 1993).

The baseline condition of the AX-CPT occurred exactly as described above. The degraded and interference conditions were identical to the baseline conditions except in the following respects (see Figure 3). In the degraded condition, visual degradation was introduced by randomly removing (at each presentation) 85% of the pixels which make up each of the letters in the stimulus set. This level of degradation was determined through pilot study to produce approximately 75% accuracy in naming single letters. In the interference condition, distractor letters appearing in a different color (white) were presented in addition to the cue and probe letters. Participants were required to respond to the distractors to ensure encoding (by pressing the non-target button), but were instructed to otherwise ignore them when monitoring for targets. During the delay period of every interference trial, three distractors were presented in sequence, each with a duration of 300 msec, and an interstimulus interval of 1000 msec.
Procedure

Participants were tested in a single testing session. Conditions were run in blocks of 30 trials, with a short rest break provided between each block. Five blocks of each condition (baseline, interference, degraded) were performed, yielding 150 trials total per condition. Participants performed all five blocks of one condition before moving on to the next condition. Condition order was counterbalanced across participants. Prior to performance of the first block of each condition, standardized instructions describing the task appeared on the computer, and the experimenter answered any remaining questions regarding the instructions. Participants were asked to respond as quickly as possible to each stimulus, while maintaining accuracy. One full block of trials were then performed as practice prior to administration of the experimental trials for that condition. This ensured that subjects understood the instructions and were performing the task appropriately.

Data analysis

Data were analyzed using error rates (misses and false alarms), signal detection indices (d') and RTs as the dependent measures of interest. RTs were examined for correct responses only, unless otherwise noted. For each of the three conditions, analyses of non-target error rates and RTs were conducted with repeated measures ANOVAs with group (young, old) as a between-subject factor and trial type (AY, BX, BY) as a within subjects factor. Analyses of target trial error rates and RTs were conducted using paired t-tests. Target (i.e., AX) and non-target trials were analyzed separately because of their different response requirements and their different frequencies of occurrence (i.e., 70% for AX trials, 10% for each of the non-target trials). For the signal detection measures, a correction factor was applied in cases of a perfect hit rate (1.0) or false-alarm rate (0.0), to allow an unbiased estimation of d' (Nuechterlein, 1991). In addition to the traditional computation of d' (i.e., using hits and all false alarms), d' was also computed using just BX false alarms. This additional measure, hereafter referred to as d'-context, has been used in previous AX-
CPT studies to provide a more specific index of sensitivity to context (Cohen et al., 1999; Servan-Schreiber et al., 1996).

RESULTS

Baseline Condition

We first examined AX-CPT performance in the baseline condition. Non-target effects were examined through an analysis of trial type (AY, BX, BY). As expected, there was a highly significant main effect of trial type on both false alarm rates (F(2,508)=42.1, p<.001) and reaction times (F(2,508) = 122.5, p<.001). This reflected the fact that, compared to the BY control trials, performance was significantly worse for both the BX (F(1,254) = 63.98, p<.001; RT: F(1,254)=140.96, p<.001) and AY (errors: F(1,254) = 99.7, p<.001; RT: F(1,254)=426.3, p<.001) trial-types, in which context competes with response biases. The main effect of age on error rates was not significant, either for nontarget (F(1,254) = 0.07, p >.10) or target trials (t(254) = 1.29, p>.1). However, there were highly significant main effects of age on reaction times both for nontarget (F(1,254) = 25. 0, p<.001) and target trials (t(254)= 3.11, p=.002). This finding is consistent with the wealth of literature suggesting a generalized pattern of age-related slowing in reaction time (Birren, Riegel, & Morrison, 1962; Cerella, 1985; Myerson & Hale, 1993; Salthouse, 1996).

The pattern of age-related slowing in reaction time was found to strongly interact with nontarget trial type (F(2,508)=41.8, p<.001; see Figure 4b). This interaction was further examined through two planned contrast tests in which responses on BX and AY trials were compared to BY trials, since BY trials provide a measure of response speed that is uninfluenced by context effects. The first contrast revealed that age-related slowing was significantly increased in BX trials relative to BY trials (F(1,254)= 22.6, p<.001). The second contrast revealed that age-related slowing was significantly reduced in AY trials relative to BY trials (F(1,254)=42.7, p<.001). Both of these results were in the directions predicted by the model. Moreover, a simple effects test on AY reaction times suggested that in this condition, there was no evidence of age-related slowing.
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(F(1,254)=0.45, p>.1), with older adults actually showing numerically faster responses. There was also a trend towards an age x trial type interaction for nontarget errors (F(2,508) = 2.45, p=.09; see Figure 4a). This trend was in the predicted direction, with older adults showing numerically greater BX errors and numerically fewer AY errors than young adults (see Figure 1a). Moreover, the more focused measure of context sensitivity provided by the d'-context measure (AX hits vs. BX false alarms) did reveal significantly reduced sensitivity in older adults (t(254)=2.38, p<.05; see Figure 7).

Interference and Degraded Conditions

We next examined effects of task difficulty by analyzing data from the interference and degraded conditions. The first analysis collapsed across age to investigate the main effect of condition on error rates. Error rates were significantly greater for both the interference (target errors: t(255)=9.8, p<.001; nontarget errors: F(1,254)=230.1, p<.001) and degraded (target errors: t(255)=13.8, p<.001; nontarget errors: F(1,254)=29.4; p<.001) conditions relative to baseline. These results validate that both condition manipulations acted to increase general task difficulty.

The second analysis examined whether these effects of condition interacted with age. A direct comparison of the interference and baseline conditions revealed a number of significant effects. Most importantly, there was a significant age x condition x trial type interaction for nontarget errors (F(2,508)=4.5, p=.01). Contrasts revealed that under interference, older adults made significantly more BX errors than young adults (F(1,254)=5.38, p<.05). However, there were no significant differences in BY errors (F(1,254)=1.83, p>.10). Most strikingly, older adults made significantly fewer errors than young adults on AY trials in the interference condition (F(1,254)=3.86, p=.05). Significant age x condition interactions were also observed for target errors (F(1,254)=9.7, p=.002) and d'-context (F(1,254)=5.8, p<.05; see Figure 7). Under interference, older adults made more target errors than young adults (F(1,254) = 12.14, p<.001),
and showed a greater reduction in context sensitivity. The analysis of reaction times also revealed a significant age x condition interaction for target trials (F(1,254) = 23.7, p<.001), with age-related slowing on target trials being greater under interference. The age x condition x trial type interaction on nontarget reaction time was not significant (F(2,508) = 2.0, p>.1). However, when considering the interference condition alone, the age x trial type interaction remained highly significant (F(2,508) = 39.5, p<.001), with greater age-related slowing in BX trials (F(1,254)= 8.7, p<.01) and reduced age-related slowing in AY trials  (F(1,254)=41.0, p<.001), when compared against the BY trial reference. Thus, for all effects, the predicted age-related changes in AX-CPT performance were accentuated or remained constant under interference. The accuracy and reaction time data for the interference condition are shown in Figure 5.

The direct comparison of the degraded condition to baseline revealed a different pattern than the interference comparison. The age x condition x trial type interaction for non-target errors was not significant (F(2,508)=0.03, p>.1), nor was the age x condition interaction for d'-context (F(1,254)=0.53, p>.1; see Figure 7). There was a significant age x condition effect for target errors (F(1,254)=5.9, p<.001), reflecting increased age differences in target errors in the degraded condition. However, the effect of the degraded condition on target errors was no different from that observed under interference, as evidenced by a non-significant age x condition interaction when directly comparing the degraded and interference conditions F(1,254)=0.2, p>.1). Finally, for reaction times, neither the age x condition effect for target trials (F(1,254)=1.0, p>.1), nor the age x condition x trial type effect for nontarget trials (F(2,508)=2.0, p>.1) was statistically significant. The accuracy and reaction time data for the degraded condition are shown in Figure 6.

Regression Analyses

Across all three conditions of the AX-CPT it appeared that older adults showed a pattern of generalized response slowing. However, in the face of this pattern, it appeared as if response
slowing was significantly increased on BX trials and significantly reduced on AY trials, as predicted by the model. To further test the relationship between age and response time slowing as a function of non-target trial type, we conducted hierarchical multiple regression analyses. Thus, for each task condition (baseline, interference, degraded) we examined whether response time on each of the three nontarget trial types (AY, BX, BY) served as a significant predictor of age, treated here as a continuous rather than a group variable. Responses on BY trials were entered on the first step of the analysis. As expected, across all three conditions, BY response times were positively associated with age, indicating a generalized pattern of slowing (baseline: beta=0.35; t=5.9; p<.001; interference: beta=0.46, t=8.2, p<.001; degraded: beta=0.37, t=6.3, p<.001). Response times for AY and BX trials were added together on the second step. Both trial types were found to be significant predictor variables for all three conditions. Thus, BX response times were found to have an additional positive relationship to age, even after variance due to generalized slowing on BY trials had already been accounted for (baseline: beta=0.28, t=2.8, p<.01; interference: beta=0.32, t=3.3, p=.001; degraded: beta=0.37, t=4.0, p<.001). Most strikingly, across all three conditions, AY response times were found to be negatively associated with age (baseline: beta=-0.22, t=-3.6; p<.001; interference: beta=-0.14; t=-2.2; p<.05; degraded: beta=-0.31, t=-5.1, p<.001). Thus, after accounting for generalized slowing, older age is associated with faster AY responses.

As noted under Methods, there were significant, although modest reduction in WAIS-R vocabulary scores among older adults. In order, to determine whether this difference may have accounted for the age relationships with reaction times described above, we conducted an additional series of hierarchical regressions, in which we forced both WAIS-R vocabulary scores and BY reaction times to enter the equation on the first step. We then conducted the regressions as before, with BX and AY reaction times added on the second step. For all three conditions (baseline, degraded, interference) the pattern of correlations was unchanged. Moreover, WAIS-R scores were not significantly associated with age in any of the conditions.

**DISCUSSION**
The present study was designed to test specific predictions regarding cognitive control function in healthy aging. These predictions were derived from a connectionist computational model which postulates that the representation and maintenance of context is a key mechanism of cognitive control, and which suggests that this mechanism is linked to the functional interactions between specific neural systems, the DL-PFC and DA system. Based on evidence regarding disturbances to these systems in healthy aging, we predicted that older adults should demonstrate evidence of a specific impairment in context processing. The model was used to generate explicit predictions regarding the consequences of this impairment on a simple task requiring cognitive control -- the AX-CPT. The results of the study clearly supported these predictions of the model. Older adults showed impaired performance on BX and AX trials, thus demonstrating a reduced sensitivity to context. More importantly, the results also supported a highly counterintuitive prediction of the model: that context processing deficits would cause older adults to show improved performance relative to young adults on AY trials. In particular, on AY trials older adults tended to make fewer errors and have equally fast responses. Moreover, regression analyses demonstrated that, after accounting for baseline response speed (through BY responses), age was negatively associated with AY reaction time, such that older age is associated with quicker AY responses. This latter finding is especially compelling, given that the relationship between age and reaction time is almost always found to be positive in the cognitive aging literature (Cerella, 1985; Myerson & Hale, 1993; Salthouse, 1996). Finally, the results demonstrated the specificity of the prediction. The interference condition, which increased the demand on context processing, produced an accentuation of the age-related differences in AX-CPT performance. In contrast, the degraded condition, which equally increased task difficulty but did not impact context processing, had virtually no effect on age-related performance changes.

Thus, taken together, the results of this study are consistent with the idea that older adults show deficits in cognitive control due to an impaired context processing mechanism. In the following section, we discuss limitations of the current study and future directions which could increase the generality of our conclusions. In the final section, we discuss the relationship of our
model to other prominent theories of cognitive aging and to our hypotheses regarding cognitive and neurophysiological deficits in other populations (i.e., schizophrenia).

Study Limitations and Future Directions

Representation vs. Maintenance of Context. A primary component of our theory is that older adults will show deficits related to both the representation of context and the maintenance of this information over time. A primary test that we have used in previous studies to examine the integrity of context maintenance is a manipulation of the cue-probe delay duration. Thus, in the AX-CPT subjects perform the task under both short and long delay conditions. In order to equate the two conditions on other factors, such as time-on-task, the intertrial interval (ITI) is counterbalanced with delay, so that short delay trials have a long ITI and long delay trials have a short ITI. A pattern of context processing impairment that amplifies with delay is thought to be diagnostic of deficits in context maintenance. For example, in the AX-CPT, a context maintenance deficit would be reflected in BX performance worsening with delay, while AY performance improves. In the current study, only the long delay condition was included, which limited our ability to make inferences regarding age-related changes in the maintenance of context. In particular, if healthy aging is associated with a deficit in context maintenance, then age-related differences in performance should be reduced under short delay conditions. On the other hand, if healthy aging is only associated with a deficit in context representation but not context maintenance, then the age differences in AX-CPT performance should not interact with delay. Thus, one direction for future research is to examine the role that delay plays in older adults' performance of the AX-CPT.

Context Processing Across Cognitive Domains. A second component of our theory is that the representation and maintenance of context is a central element of cognitive control, and is present in multiple cognitive processes, including attention, working memory, and inhibition. Thus, if healthy aging is associated with a decline in context processing capabilities, then behavioral performance should decline across a wide variety of task domains that involve these cognitive processes. We conceptualize the AX-CPT as a task paradigm which involves all three of these
cognitive processes. Thus, the results of the study are fully consistent with the model. However, an important limitation of the study is that it does not establish whether the context processing impairment generalizes across task domains. Our theory makes two specific and testable predictions. First, it predicts that the performance of older adults on the AX-CPT will be significantly correlated with their performance on standard tests of attention, working memory and inhibition (e.g., Stroop, SOPT, reading span, stop-signal, etc.). Second, it predicts that these correlations will be greatest if the tasks are explicitly designed or modified to challenge context processing. In particular, tasks in which the context information changes rapidly and must be maintained over a delay period are most likely to place the strongest demands on context processing mechanisms. For example, a "high context demand" variant of the standard Stroop task would be one in which the task instructions (i.e., word reading vs. color naming) change on a trial-to-trial basis (e.g., Allport, Styles, & Hsieh, 1994) and which these task instructions are temporally separated from stimulus presentation by a relatively long delay (i.e., 5-10 sec). We suggest that age-related differences in performance on such a variant of the Stroop would increase relative to standard Stroop conditions, and that these changes in performance would be related to the age-related differences observed in AX-CPT performance under high context demand conditions. Thus, a second important direction for future research will be to explicitly test whether context processing deficits underlie age-related performance deficits in multiple task domains.

**DL-PFC and DA Function.** A third component of our theory is that the decline in context processing capabilities that occur with healthy aging are a direct consequence of disturbances in DA function in DL-PFC. As discussed in the Introduction, there is a substantial literature documenting disturbances in both PFC and DA function in healthy older adults. However, this literature has not previously addressed whether these disturbances are related to age-related context processing impairments. Although the current results are consistent with the idea that the age-related impairments observed in the AX-CPT are related to DL-PFC and DA dysfunction, this aspect of the theory was not directly tested. In our previous work, we have used functional neuroimaging methods to show that young adults activated the DL-PFC during AX-CPT performance, and that
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this activation is specifically related to the duration over which context information must be maintained (Barch et al., 1997; Braver et al., 1999b). Thus, a first prediction of the theory is that older adults will show abnormal activation of this brain region during AX-CPT performance. Moreover, the theory predicts that older adults will not show the delay-related increases in DL-PFC activity that have been in young adults. A second prediction of the theory is that older adults will show abnormal activation of the DA system during AX-CPT performance. Although it is harder to directly test predictions regarding the role of DA function in DL-PFC on AX-CPT performance, it should be possible to increase systemic levels of DA through pharmacologic challenges (e.g., administration of DA precursors such as L-DOPA or DA agonists such as apomorphine). The theory suggests that increasing DA levels in older adults should serve to reduce age-related differences in context processing capabilities. Thus, a third direction for future research will be to more directly test the causal relationship between DL-PFC and DA function and context processing in healthy aging.

Relationship to Other Theories of Cognitive Aging

Working Memory Theory. A common hypothesis regarding cognitive deficits in healthy older adults is that they represent a fundamental deficit in working memory capacity (Craik et al., 1990; Light & Anderson, 1985; Salthouse, 1990). Thus, two common predictions of this hypothesis are that: 1) age-related deficits in cognitive performance will increase as the working memory demands of the task are increased; and 2) age-related variance in cognitive performance can be attributed to age-related variance in working memory capacity. Our theory makes a related claim, by suggesting that older adults are impaired in the ability to retain context information in active memory. Thus, as discussed above, we predict that age-related differences in task performance will increase as the demands on the active maintenance of context are increased. We also predict that context processing capabilities may be an important predictor of age differences across a wide variety of task domains. An important question then arises as to whether the representation and maintenance of context information is merely identical to, or just another way of referring to working memory function. We would like to suggest that the representation and
maintenance of context is an important component of working memory, but is not identical to working memory as it is commonly conceived and operationalized.

Working memory is commonly defined as the collection of processes responsible for online maintenance and manipulation of information necessary to perform a cognitive task (Baddeley & Hitch, 1994). Often times, tasks that are referred to as working memory tasks, involve short-term maintenance of the identity of previously presented stimuli, such that they can be repeated back accurately (e.g., simple span tasks). In contrast, context maintenance may not involve memory for the identity of stimuli. For example, in the AX-CPT task, it is not necessary to remember the exact identity of the previous cue – only whether it was an A or non-A. Moreover, we suggest that an important component of context representations is that they are used to bias the processing and/or response made to subsequent, possibly ambiguous events. In this way, we view context representations as similar to goal representations in production system models of cognition (e.g., Anderson, 1983). For example, in the AX-CPT the presentation of an A cue might set up a context representation of the form, "press target button if the next item is an X" whereas presentation of a non-A cue might set up a context representation of the form, "press non-target button if the next item is an X". Thus, one important component of our theory is that it differentiates maintenance of context information from short-term memory of identity information. Moreover, it suggests that age differences on such short-term memory tasks, such as the standard digit span task, may not reflect context processing capabilities, or be strongly dependent upon the function of the DL-PFC and DA systems.

It is interesting to note that studies of working memory in older adults have found that age-related deficits on standard span tasks may not be as severe as the deficits observed on working memory tasks that may make heavier demands on context processing (Fisk & Warr, 1996; Humes, Nelson, Pisoni, & Lively, 1993). Moreover, recent work by Engle and colleagues suggests that the construct of short-term memory may be differentiable from the construct of working memory (Engle, Tuholski, Laughlin, & Conway, 1999). Engle et al. (1999) use the term working memory to refer to processes much more similar to the representation and maintenance of context. Finally, it is
important to note that our theory of context processing does not map cleanly onto standard theoretical ideas regarding working memory. In particular, Baddeley's influential model of working memory posits two types of working memory components: domain-specific storage systems and a central executive controller. In many ways, our notion of context representation is similar to Baddeley's formulation of the central executive, in that context representations are similar to goal states, and serve to govern how other representations are used. However, unlike the central executive, which does not subserve storage functions, we suggest that context information is both represented and actively maintained over time within a specific subsystem of the brain, the DL-PFC.

PFC Theory. Another theory that has been widely discussed in the cognitive aging literature is that cognitive deficits in older adults can be seen as reflecting a "frontal syndrome" (Moscovitch & Winocur, 1995; Perfect, 1997; West, 1996). That is older adults are thought to show the pattern of cognitive deficits that are displayed by patients with damage to PFC. Our theory is highly compatible with this account, in that we suggest older adults show cognitive declines that are directly related to PFC dysfunction. However, our theory goes beyond standard frontal accounts of aging, in that we postulate a specific neurobiological disturbance which impacts a particular region of PFC (reduced DA effects in DL-PFC) to produce a particular type of cognitive deficit (context processing impairment). One important issue to be resolved in future research is the extent to which the cognitive processes associated with DL-PFC function can be differentiated from cognitive processes associated with other regions of PFC, and whether older adults show cognitive deficits that appear to be selective to particular PFC sub-regions. For example, there is now a growing literature which has suggested dissociations between DL-PFC function and the function of ventromedial PFC in terms of the cognitive processes associated with each (Bechara, Damasio, Tranel, & Anderson, 1998; Fuster, 1989). Damasio and colleagues have suggested that ventromedial PFC is involved in the use of emotional and autonomic information to bias decision-making processes (Damasio, 1994). It would be interesting to know whether the pattern of cognitive deficits displayed by healthy older adults is selective to the processes we hypothesize to be subserved by DL-PFC, or whether deficits are also displayed in task domains that...
more closely reflect ventromedial PFC function. To our knowledge, such studies have yet to be conducted. Interestingly, however, a recent volumetric neuroimaging study of healthy aging found that gray and white matter volume in ventromedial regions of PFC was selectively spared relative to other PFC regions (Salat et al., in submission).

Inhibition Theory. Another prominent theory of cognitive aging suggests that the primary cognitive deficits observed in older adults are not due to reduced working memory function, but rather an impairment in inhibition. In particular, this theory, originally proposed by Hasher and Zacks (Hasher & Zacks, 1988), suggests that older adults cannot suppress unwanted behaviors or inhibit irrelevant information from entering working memory. This theory has been influential, in that it accounts for a large amount of data, and provides an explanation of why older adults appear to have minimal deficits in some memory tasks (Zacks & Hasher, 1997). The theory we put forth here is consistent with the suggestion of age-related inhibitory deficits, in that we suggest that a major function of context representations is to provide a mechanism by which task-relevant information can effectively compete with, and suppress task-irrelevant information and responses. Indeed, the present study lends support for the idea of inhibitory deficits in older adults, in that older adults show increased errors and reaction time interference on BX trials. Performance on BX trials is critically dependent upon the ability to inhibit the strong bias to make a target response to the probe stimulus. However, an important difference between our theory and other inhibition theories is that we suggest that context processing is a central mechanism that underlies both working memory and inhibitory function. Thus, in the present study, we postulate that the same impairment in context processing underlies performance on both BX and AX trials. On BX trials context serves an inhibitory function, while on AX trials context serves a memory function, serving to facilitate effective processing. Consequently, we suggest that a more sensitive means of gauging context processing capability is by examining the relationship between AX and BX performance through the d'-context measure. A strong claim of our theory is that inhibitory deficits in healthy older adults will be greatest under task conditions in which successful inhibition
is dependent upon actively maintaining context information over a delay period. This claim remains to be tested.

Processing Speed Theory. Probably the most influential class of theories regarding cognitive aging are processing speed theories, which suggest that age-related cognitive deficits are related to a global decline in the speed with which information is processed in the nervous system (Cerella, 1985; Myerson, Hale, Wagstaff, Poon, & Smith, 1990; Salthouse, 1996). Proponents of this theory point to the fact that much of the age-related variance in cognitive task performance can be accounted for by variance in measures of simple processing speed. A strong form of this theory suggests that a reduction of processing speed in older adults is the primary mechanism underlying all age-related cognitive deficits. Moreover, a primary prediction of the theory is that reaction times in older adults can be expressed as a linear function of young adults reaction times in which the slope is greater than one (Brinley, 1965; Cerella, 1985; Myerson & Hale, 1993). In other words, the longer a task takes for young adults, the greater the expected age difference in reaction time. This prediction falls out of the idea that processing speed is globally slower in older adults across all processing stages and systems. Thus, the more processing a task requires, the greater the amount of slowing to be expected.

The present results are certainly consistent with the idea that older adults show a global slowing in cognitive processing speed. Across all three task conditions, a highly reliable main effect of age was found in reaction times. However, we also observed highly significant age x trial type interactions across all three conditions. These interactions were in part due to the fact that age-related slowing was significantly less on AY trials than it was on BY trials, which may provide a baseline measure of response speed. This finding is significant in that of all four trial types, young adults showed the slowest reaction times on AY trials. As just discussed, a primary prediction of processing speed theories is that the condition which generates the slowest reaction times should be the one which produces the greatest age differences. Yet in the AX-CPT the opposite pattern occurs. Furthermore, we found that AY reaction times across all three conditions showed a negative correlation with age, once the age-relationship of BX and BY responses times were
factored out. To our knowledge, this is the first time that such a negative relationship between age and reaction time has been observed. Moreover, it is unclear how such a relationship could be explained by processing speed theories. Thus, our findings do not directly conflict with processing speed theories, in that they support the idea that processing speed slows with healthy aging. However, the results suggest that processing speed theory is not sufficient to account for all of the age differences observed in AX-CPT performance. We suggest that in addition to generalized slowing, healthy older adults suffer from a specific decline in the ability to represent and maintain context.

DA Theory. Li and colleagues have recently put forth a theory which links at least some cognitive deficits in aging to declines in DA function (Li & Lindenberger, in press). This theory is similar to ours in that it explores the function of DA in cognition through the use of computational simulations. In addition to accounting for impaired cognitive performance through changes in DA function, the theory also suggests that reduced DA activity can account for the findings of increased inter- and intra-individual variability in the performance of older adults. The theory of Li et al. (in press) is highly compatible to our ideas regarding the role of DA in context processing, in that it suggests DA activity serves to modulate the flow of information processing by regulating the sensitivity of units to external input. There is a difference in emphasis between our theory and that of Li et al (in press), in that we have specifically focused on the functional interaction of DA in DL-PFC as the locus for cognitive deficits in aging. In contrast, Li et al. have focused on the DA system more generally, and do not make any claims about specific DA subsystems or projections.

Cognitive Control, DA, and DL-PFC Function in Healthy Aging vs Schizophrenia.

Based on our computational modeling work, we have hypothesized that healthy older adults suffer from cognitive control impairments resulting from a failure to properly represent and maintain context information. Further, we have suggested that these cognitive impairments are directly related to dysfunction of the DA system in DL-PFC that may occur with healthy aging. However, in prior work, we have also suggested that patients with schizophrenia suffer from similar cognitive control impairments, which we have also hypothesized are related to a dysfunction of the
DA system in DL-PFC (Barch et al., submitted; Barch, Carter, Hachten, & Cohen, in press; Braver, Barch, & Cohen, 1999a; Braver, Cohen, & Servan-Schreiber, 1995; Carter & Barch, in press; Cohen et al., 1999; Cohen et al., 1996).

By postulating similar cognitive control impairments in healthy aging and schizophrenia, we do not mean to equate healthy aging with schizophrenia. Clearly, healthy older adults do not suffer from the range of symptoms experienced by patients with schizophrenia, such as delusions and hallucinations. Although healthy aging and schizophrenia may both involve a dysfunction of the DA system in DL-PFC, three major factors likely contribute to critical differences between the two. First, the DA disturbance in DL-PFC is likely to be more severe in schizophrenia than in healthy aging. Second, the etiological mechanisms leading to a dysfunction of the DA system in DL-PFC are likely to be different in healthy aging than in schizophrenia. Further, we do not claim to have complete theories of either aging or schizophrenia. Both healthy aging and schizophrenia likely involve additional disturbances besides DA deficits in DL-PFC, and these additional disturbances may differ between aging and schizophrenia. For example, schizophrenia may also involve deficits in temporal, limbic and subcortical structures, whereas neurophysiological changes occurring with healthy aging may not involve all of these same structures. All of these factors have important ramifications for understanding the pathophysiology and cognitive underpinnings of both healthy aging and schizophrenia, and are issues that will need to be addressed in future work. Nonetheless, we believe our hypothesis regarding the cognitive and neural mechanisms underlying at least a subset of cognitive deficits present in healthy aging is a useful one that may be able to provide critical insights into the literature on cognitive aging and its neurophysiological underpinnings.

CONCLUSIONS

We have presented a theory of cognitive aging that draws an explicit link between a particular cognitive impairment in context processing, and specific neurobiological disturbances in DA and DL-PFC function. We used computer simulations to derive detailed, and counter-intuitive predictions regarding the effects of impaired context processing on performance in the AX-CPT
paradigm. The results of a large sample behavioral study of healthy young and older adults confirmed the predictions of the model, and thus provide an initial measure of support for the theory. We believe that the account we have put forth provides a promising new direction for the study of cognitive deficits in aging, by suggesting that a single common mechanism may underlie age-related cognitive declines across a variety of task domains, and by linking this mechanism to specific neural substrates. Further research will be needed to test additional predictions of the theory and to establish its generality.
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### Table 1

Demographic Characteristics

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* p < .05
FIGURE CAPTIONS

**Figure 1.** Schematic of AX-CPT paradigm. Single letters are visually displayed as a series of cue-probe pairs. A target is defined as the occurrence of an "X" probe immediately following an "A" cue. There are three types of nontarget trials: BX, AY and BY (where "B" refers to any non-A cue, and "Y" refers to any non-X probe).

**Figure 2.** Simulations of context processing impairments in the AX-CPT. Graphs show simulation data from computational model of the AX-CPT task. Light bars show the performance of the intact model. Dark bars show the effect of simulating a disturbance of DA function in PFC, as is thought to occur in healthy aging. Graphs depict accuracy on AY and BX trials, at both short (right panel) and long delays (left panel).

**Figure 3.** Three task conditions of the AX-CPT. In the interference condition three distractors appearing in a different color (shown here in white) are presented sequentially during the cue-probe delay interval. In the degraded condition, a subset of pixels are randomly removed from each letter. In all conditions, the cue-probe delay is long (~5s) while the intertrial interval is short (1s).

**Figure 4.** Data from baseline condition. Performance in each of the four trial types is shown (AX, AY, BX, BY). Light bars represent the performance of young adults, dark bars represent the performance of older adults. A. Percentage of errors for each trial type. There is a trend for greater BX errors, but fewer AY errors in older adults. B. Reaction times (correct trials only) for each trial type. Older adults show disproportionate slowing on BX trials (relative to BY trials) for older adults, but the comparable response speed on AY trials.

**Figure 4.** Data from interference condition. A. Percentage of errors for each trial type. Older adults make significantly more errors on AX and BX trials, but significantly fewer errors on AY trials. B. Reaction times (correct trials only) for each trial type. Older adults show disproportionate response slowing on BX trials (relative to BY trials), but minimal slowing on AY trials.

**Figure 5.** Data from degraded condition. A. Percentage of errors for each trial type. There are no age differences on AY and BX trials. B. Reaction times (correct trials only) for each trial type.
Figure 6. Context sensitivity across the three task conditions. Data is expressed in d' units. The greatest age difference occurs in the interference condition.