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Context Processing in Older Adults: Evidence for a Theory Relating Cognitive Control to Neurobiology in Healthy Aging

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A theory of cognitive aging is presented in which healthy older adults are hypothesized to suffer from disturbances in the processing of context that impair cognitive control function across multiple domains, including attention, inhibition, and working memory. These cognitive disturbances are postulated to be directly related to age-related decline in the function of the dopamine (DA) system in the prefrontal cortex (PFC). A connectionist computational model is described that implements specific mechanisms for the role of DA and PFC in context processing. The behavioral predictions of the model were tested in a large sample of older ($N = 81$) and young ($N = 175$) adults performing variants of a simple cognitive control task that placed differential demands on context processing. Older adults exhibited both performance decrements and, counterintuitively, performance improvements that are in close agreement with model predictions.

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, 1999; Shaw et al., 1984) and in neurochemical systems such as the dopamine (DA) system (e.g., Arnsten, Cai, Steere, & Goldman-Rakic, 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, researchers have only a rudimentary understanding of how cognitive disturbances in aging 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 used connectionist computational models to test specific hypotheses regarding the role of DA and PFC in cognition (Braver, Barch, & Cohen, 1999b; Braver & Cohen, 2000, 2001; Cohen, Braver, & O'Reilly, 1996). Specifically, we have argued that interactions between these neural systems subserve the representation, maintenance, and updating of context information. Furthermore, we have argued that these context processing capabilities are critically involved in the ability to exert control over thoughts and actions and thus contribute centrally to multiple cognitive domains, including attention, inhibition, and working memory.

In the present study, we describe how our modeling approach can also be used in the study of healthy aging to generate novel

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hypotheses and empirically testable predictions regarding age-related cognitive and neurobiological changes and the relationship between them. Specifically, we describe a computational model of context processing and discuss how it provides an explicit account of (a) normal cognitive control function and (b) 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 in which we empirically tested these predictions in a large sample of healthy young and older adults performing a simple cognitive control task. 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 on 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, maintain, and update 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 that 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 both in 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 Task (SOPT; Daigneault & Braun, 1993). Older adults also appear to have difficulty in tasks that involve suppressing the influence of irrelevant information or inhibiting unwanted responses (Hasher & Zacks, 1988; Zacks & Hasher, 1997). For example, healthy older adults display deficits on a number of tasks that 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 that appears to be vulnerable to the effects of aging is attentional control. In particular, age-related declines are observed in both selective attention tasks, such as the Stroop Color and Word Test (Stroop, 1935; see also 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 Test (WCST; Grant & Berg, 1948; see also 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 disturbances to multiple different types of cognitive processes. However, it is also possible that one or more common underlying mechanisms may lead to deficits in multiple tasks domains. For example, Salthouse (1996) 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. 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 that make closer contact with neurobiology comprises 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 that appear to be dependent on the integrity of PFC—namely, that they require the internal representation, maintenance, and updating of context information in the service of exerting control over thoughts and behavior (Braver & Cohen, 2000; Braver et al., 1999b; Cohen et al., 1996). We define *context* as any task-relevant information that is internally represented in such a form that it can bias processing in the pathways responsible for task performance. Goal representations are one form of such information, which have their influence on planning and overt behavior. However, we use the more general term *context* to include representations that may have their effect earlier in the processing stream, on interpretive or attentional processes. 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 toward the ink color dimension of a visually presented word. Thus, context representations may include a specific prior stimulus or the result of processing a sequence of stimuli, as well as task instructions or a particular intended action. Representations of context are particularly important for situations in which there is strong competition

for response selection. These situations may arise when the appropriate response is one that is relatively infrequent or when the inappropriate response is dominant and must be inhibited (such as the word name in the Stroop task). Because context representations are maintained on-line, in an active state, they are continually accessible and available to influence processing. Consequently, context can be thought of as one component of working memory. Specifically, context can be viewed as the subset of representations within working memory that govern how other representations are used. In this manner, context representations simultaneously subserve both mnemonic and control functions. This aspect of the model differentiates it from standard models of working memory (e.g., Baddeley, 1986, 1993), which postulate a strict separation of representations for storage versus control.

We suggest that impairments of context processing play a central role in understanding the cognitive declines that occur during healthy aging. Specifically, we suggest that at least a subset of age-related deficits in working memory, inhibition, attention, and executive function reflect the disturbance of this common underlying mechanism. We further suggest that this context 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 shown 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 et al., 1999). A recent magnetic resonance imaging 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 et al., 1996; Peters, Morrison, Rosene, & Hyman, 1998; Scheibel, Lindsay, Tomiyasu, & Scheibel, 1975). Moreover, one recent study showed that in primates, specific synaptic loss within dorsolateral regions of PFC correlates with age-related cognitive impairment (Peters, Sethares, & Moss, 1998). Studies of resting-state regional cerebral blood flow (rCBF) in aging have also implicated frontal disturbances. The presence of hypofrontality in older adults has consistently been documented by rCBF measurements (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 (Cabeza, 2001; Grady, 2000). 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 showed that older adults had 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, serotinergic, and adrenergic systems (Martin & Rubin, 1997). In nonhuman 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., the WCST and the Stroop test; Volkow et al., 1998).

The Functional Roles of DA and DL-PFC

Taken together, these findings are consistent with the primary hypothesis of the present study: 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 past 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 tasks involving a cognitive control component (Cabeza & Nyberg, 2000), especially tasks involving working memory (D'Esposito et al., 1998; Smith & Jonides, 1999). 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 (Braver & Cohen, 2001; 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, including effects on delay-related activity during active memory (Sawaguchi, Matsumara, & Kubota, 1990a, 1990b). Moreover, pharmacological blockades of DA receptors in circumscribed regions of primate DL-PFC have been found to produce reversible deficits in performance on active memory tasks (Sawaguchi & Goldman-Rakic, 1991). These findings of DA effects on behavioral performance have also been observed in humans, with DA antagonists impairing performance on cognitive control tasks (Luciana, Collins, & Depue, 1998; Magliozzi, Mungas, Laubly, & Blunden, 1989), whereas 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 related to 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 general and aging literatures, there has been limited explicit theorizing regarding the mechanisms by which they subserve aspects of cognitive processing. There is consensus that DL-PFC plays a role in the active maintenance of task-relevant information, but there is not yet agreement regarding the mechanisms by which active maintenance occurs (Durstewitz, Seamans, & Sejnowski, 2000), and there has been very little theorizing about the function of DA (but see Li, Lindenberger, & Frensch, 2000), other than to assume it supports the memory functions of DL-PFC. To develop more explicit theories of DA and DL-PFC function, we drew on computational modeling as a tool for specifying the mechanisms by which these systems may influence cognition. Our modeling work makes use of the parallel distributed processing, 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 units. From one perspective, such models are highly simplified, capturing brain-style computation without necessarily committing to the details of any particular neural system or subsystem. 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.

The theory of cognitive control put forward here can be schematized in the form of a simple canonical model, in which a context module serves as an indirect pathway that modulates processing in a direct stimulus–response pathway (see Figure 1). This context processing module represents the functions of DL-PFC. There are three critical features of this module that provide it with the capacity for control over processing. The first is that there is strong recurrent connectivity within the context layer, which allows for the active maintenance of information. Thus, input to the context layer can be sustained through activity recirculation along mutually excitatory connections, even when the external source of input is no longer present. The second critical feature of the context pathway is its feedback connection to the direct pathway. This provides a means for activity within the context module to provide an additional source of input, which can modulate the flow of processing within the direct pathway. In particular, feedback from the context layer serves to bias the local competition for representation that exists within each module, favoring one activation pathway or set of representations over competitors. This biasing action of the context module can produce inhibitory effects on processing by allowing a weak pathway to inhibit the more dominant one.

The third critical feature of the context module is the modulatory input that reflects the processing functions associated with DA projections into DL-PFC. In other work, we have provided a detailed description of this processing mechanism (Braver, Barch, & Cohen, 1999a; Braver & Cohen, 1999, 2000). Briefly, this connection serves to regulate the access of incoming afferents into the context module. Specifically, we hypothesize that the connection serves as a gating mechanism. When the gate is opened, as is hypothesized to occur when there is phasic DA activity, incoming information can gain access to the context layer, thus updating the current state of context representation. Conversely, when the gate is closed, access to the context module is restricted, thus protecting context representations from the interfering effects of noise, or other irrelevant inputs. We have hypothesized that the timing of gating signals is learned through a reward prediction learning mechanism associated with the midbrain DA system (Schultz, Dayan, & Montague, 1997), which enables selection of task-

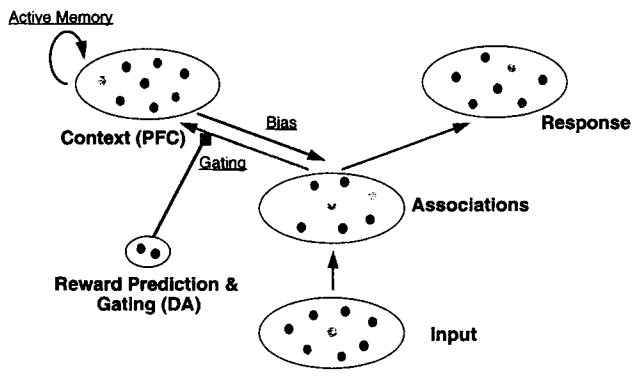

Figure 1. Diagram of canonical model. Key computational principles of context processing mechanism are shown: active memory through recurrent connections, top-down bias through feedback connections, and regulated access of contextual input through modulatory gating connections. PFC = prefrontal cortex; DA = dopamine.

relevant information as context, because of the association of that information with the potential for future reinforcement.

An important insight that has emerged from our work is that the context processing functions of our model demonstrate how a single underlying mechanism, operating under different task conditions, might subserve 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). 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 model, 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 working memory function is involved. Once again, there is no dedicated mechanism for this function in our model. 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 "working 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 "working 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. Thus, in all circumstances, the same context processing mechanism is involved. Here we put forth the hypothesis that in healthy aging this context processing mechanism is impaired. Consequently, we suggest disturbances in context processing may form a common basis for many of the age-related deficits observed across multiple cognitive domains, including attention, inhibition, and working memory.

Our simulation work with this model of cognitive control has also suggested that a neurobiological locus for such a context processing impairment may be found when the DA projections to DL-PFC are disrupted. Specifically, we have found that in the model when DA effects are reduced in the context module, the representation of context becomes less reliable (because access is partially blocked) and that even when context representations do get activated, the maintenance of those representations decays more quickly over time (because information is more susceptible to the interfering effects of noise and task-irrelevant inputs). These disturbances in DL-PFC activity dynamics result in a pattern of behavioral impairment that is reflected both in terms of context representation and maintenance. The behavioral impairments of the model can be clearly seen in the performance of cognitive control tasks that heavily rely on these context processing functions. A task that we have used to demonstrate these effects is a simple but informative paradigm—a version of the classic Continuous Performance Test (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956), known as the *AX*-CPT (Cohen, Barch, Carter, & Servan-Schreiber, 1999; Servan-Schreiber, Cohen, & Steingard, 1996). In the next section, we describe specific predictions of the model regarding cognitive disturbances hypothesized to occur in older adults during performance of the *AX*-CPT.

Model Predictions Regarding Cognitive Control Impairments in Healthy Aging

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 2). The object of the task is to make a target response to an *X* (the probe), but only when it follows an *A* (the cue), and a nontarget response in all other cases (hence the name *AX*). Performance in this task relies on the representation and maintenance of context information, insofar as the correct response to *X* depends on 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.

Of importance, the *AX*-CPT also provides behavioral measures for examining different aspects of context processing. In particular, within the *AX*-CPT, context information serves inhibitory, attentional, and working memory functions. In the task, target (*AX*) trials occur with high frequency (70%). This induces two types of biases in participants. The first is a bias to make a target response to the occurrence of an *X* probe. On those trials in which a target response should not be made to the *X* probe (i.e., *BX* trials, where *B* refers to any non-*A* cue), context information must be used in an inhibitory fashion to override the tendency to false alarm. The second bias that occurs in the *AX*-CPT is an expectancy to make a target response following the occurrence of an *A* cue. In this case, the context provided by the cue serves a predictive function that directs attention to a particular response (i.e., attention to action; A. Allport, 1989; Norman & Shallice, 1986). On those trials in which the cue is an invalid predictor of the response (i.e., *AY* trials, where *Y* refers to any non-*X* probe), this attentional function of context creates the tendency to false alarm. This type of cue validity effect is similar to others that have been well studied in the attentional literature (e.g., Posner, 1980). Thus, the integrity of context processing can be examined not only through performance

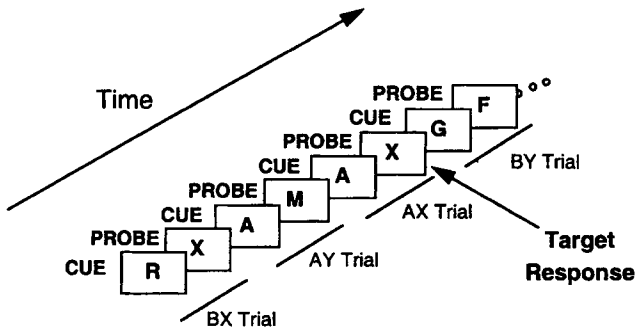

Figure 2. Schematic of *AX*–Continuous Performance Test paradigm. Single letters are visually displayed as a series of cue–probe pairs. A target is defined as the occurrence of an *X* probe immediately following an *A* cue. There are three types of nontarget trials: *BX*, *AY*, and *BY* (where *B* refers to any non-*A* cue, and *Y* refers to any non-*X* probe).

on *AX* target trials but also through an examination of performance on nontarget trials.

A key element of our theory is that both attentional and inhibitory functions in the *AX*-CPT should be subserved by a single underlying mechanism—the internal representation of context information within DL-PFC. This assumption can be tested by examining the relationship of *AY* to *BX* performance. Note that on *BX* trials, the internal representation of context should improve performance, by inhibiting an inappropriate response bias. However, on *AY* trials, representation of context should impair performance, by creating an inappropriate expectancy bias. Thus, if context representations are intact, *AY* performance should be worse than *BX* performance in terms of both errors and reaction time (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, because determination of targets is dependent on the context provided by the cue. However, *AX* performance should not be as impaired as *BX* performance, because on *AX* trials, the response bias works in participants' favor, by increasing the tendency to make the correct target response. Finally, a third type of nontarget trial, *BY*, provides a useful internal control, because 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 *AX*-CPT paradigm also provides a means for examining the mnemonic role of context information through the cue–probe delay duration. Specifically, under conditions in which there is a long cue–probe delay (e.g., 5–10 s), context information must be actively maintained within working memory. Our theory suggests that context information is both represented and actively maintained within DL-PFC. Thus, the same context processing mechanism that subserves inhibitory and attentional functions also subserves working memory functions. Consequently, a strong prediction of the theory is that the effect of delay will interact with performance on *AY* and *BX* trials. If context maintenance is intact, then the strength of context representations should either hold constant or increase with delay (i.e., if it takes some period of time for context representations to reach full activation strength). Consequently, *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, then context representations should lose strength over time. This should lead to a worsening of *BX* performance with a delay, but an improvement in *AY* performance.

We have found that reducing context processing functions through simulation of reduced DA effects in DL-PFC produces just these effects on the behavior of the model. For example, there are more *BX* than *AY* errors, and this effect becomes amplified with delay (see Figure 3). A similar pattern occurs 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. Before turning to our tests of these model predictions in a study of healthy older adults, we describe some of the direct support for the model we have obtained in young adults.

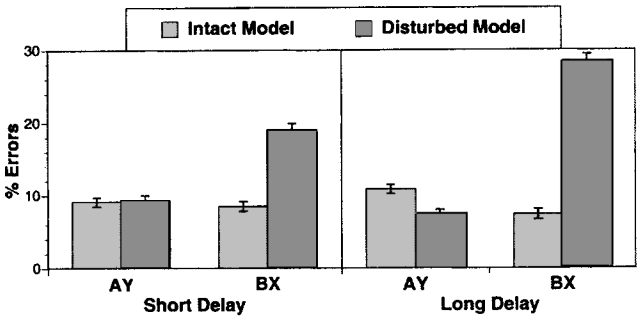

Figure 3. Simulations of context processing impairment in the *AX*-Continuous Performance Test (*AX*-CPT). Simulation data from the computational model of the *AX*-CPT task are shown. Disturbed model data show the effect of simulating a disturbance of dopamine function in prefrontal cortex as is thought to occur in healthy aging. Accuracy is depicted on *AY* and *BX* trials at both short and long delays. Error bars depict standard errors of the means.

Empirical Support for the Model

We have conducted an extensive series of studies validating the behavioral and neurobiological predictions of the model in terms of normal context processing function (Braver et al., 1999b). At the behavioral level, we have shown in a normative study of more than 200 young adults that the model accounts for detailed aspects of performance data from the *AX*-CPT. 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, whereas *BX* performance slightly improves). Moreover, the model also captures subtler effects in the data, such as the relationship between accuracy and response speed, as well as the effects of different task manipulations on behavior (i.e., interference and degraded *AX*-CPT conditions, described below). At the neurobiological level, we have provided direct support for our hypotheses regarding the role of DL-PFC during *AX*-CPT performance. Specifically, we have found that (a) DL-PFC regions are engaged during *AX*-CPT performance and show greater activity with long delay (Barch et al., 1997) and (b) DL-PFC activity shows a sustained pattern of dynamics throughout a long cue–probe delay period (but not an equivalent length intertrial interval) of the *AX*-CPT, consistent with a role for this region in active maintenance functions (Braver & Cohen, 2001). Finally, we have also provided preliminary support for the role of DA in *AX*-CPT performance in a pharmacological challenge study (Braver, 1997), in which *AX*-CPT performance improved under placebo-controlled administration of low-dose *d*-amphetamine (which acts to stimulate central DA release).

Overview of the Current Study

The current study extended our examination of the model by applying it to the domain of healthy aging. We tested model

predictions regarding the behavioral effects of context processing deficits in aging in a large sample of healthy older and younger adults performing the *AX*-CPT. Participants in the study performed the *AX*-CPT task in three different conditions that 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 (~5 s). We predicted that in this condition, the performance of older adults would correspond to the distinct behavioral signature identified in the model as indicative of a selective deficit in context processing—namely, poorer performance on *AX* and *BX* trials, unchanged performance on *BY* trials, but improved performance on *AY* trials. In two additional task conditions, we tested the specificity of our predictions by manipulating task difficulty in ways that were expected to differentially impact context processing demands. 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 task difficulty by increasing the demand on context representation and maintenance. In the degraded condition, participants performed the *AX*-CPT with stimuli that were perceptually degraded by noise, such that they were harder to identify. This manipulation was expected to increase task difficulty by increasing perceptual demands but, critically, was not expected to affect context representation and maintenance. Our prediction was that age-related changes in performance would become amplified in the interference condition but should remain constant in the degraded condition. This prediction reflects the hypothesis that age-related changes in performance on the *AX*-CPT would be specifically related to the demands on context processing and not on other factors that may affect task difficulty.

Method

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 predrug 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 women were studied in the young adult group because of concerns regarding the effects of the experimental drug on women 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 Examination over 27 (Folstein, Folstein, & McHugh, 1975), (c) Vocabulary subtest standardized score of 8 or higher on the Wechsler Adult Intelligence Scale—Revised (WAIS–R; Wechsler, 1981) 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 substance; (c) lifetime history of psychiatric disorders or substance dependence or any substance use disorder within 6 months of testing (on the basis of *Diagnostic and Statistical Manual of Mental Disorders* [4th ed.; *DSM–IV*; American Psychiatric Association, 1994] criteria); (e) evidence of dementia (on the basis of using *DSM–IV* criteria); (f) history or evidence of any neurologic disorder or 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.

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 2). Participants 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 nontarget probes, with the exception of the letters *K* and *Y*, which were excluded because of their similarity in appearance to the letter *X*. Letter sequences were presented in pseudorandom order, such that target (*AX*) trials occurred with 70% frequency and nontarget trials occurred with 30% frequency. Nontargets were divided evenly (10% each) among the following trial types: *BX* trials, in which an invalid cue (i.e., non-*A*) preceded the target; *AY* trials, in which a valid cue was followed by a nontarget probe (i.e., non-*X*); and *BY* trials, in which an invalid cue was followed by a nontarget probe.

Stimuli were presented centrally, red on a black background, for a duration of 300 ms in 24-point uppercase Helvetica font. A delay of 4,900 ms occurred between the presentation of cue and probe stimuli. The intertrial interval was 1,000 ms. To increase task difficulty, we instructed participants 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 that recorded response choice and reaction time with 1-ms accuracy. For right-handed individuals, responses were made with the middle (nontarget, middle button) and index (target, left button) fingers of the right hand. For left-handed individuals, responses were made with the middle (nontarget, middle button) and index (target, right button) fingers of the left hand. Participants were allowed a total of 1,300 ms from stimulus onset in which to respond. Responses that were slower than this limit were not recorded and elicited feedback (a "bloop" sound) as a prompt to increase speed. The tasks were conducted on Apple Macintosh computers, using PsyScope software (Cohen, MacWhinney, Flatt, & Provost, 1993) for stimulus presentation and data collection.

The baseline condition of the *AX*-CPT occurred exactly as described above. The degraded and interference conditions were identical to the baseline condition except in the following respects (see Figure 4). In the

Table 1
Demographic Characteristics

	Young adults			Older adults		
Characteristic	*M*	*SD*	%	*M*	*SD*	%
Age (years)	24.6	5.5		72.0	5.1	
Sex (male)			100			44
Education (years)	15.6	2.3		15.4	3.1	
WAIS–R Vocabulary score	14.8	2.9		13.9*	3.0	

Note. WAIS–R = Wechsler Adult Intelligence Scale—Revised.
* $p < .05$.

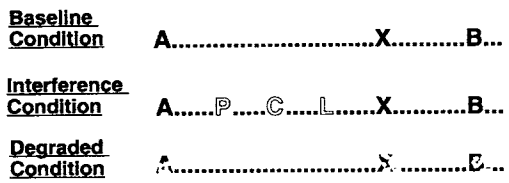

Figure 4. Three task conditions of the *AX*–Continuous Performance Test. In the interference condition three distractors appearing in a different color (shown here in white) were presented sequentially during the cue–probe delay interval. In the degraded condition, a subset of pixels was randomly removed from each letter. In all conditions, the cue–probe delay was long (~5 s) whereas the intertrial interval was short (1 s).

degraded condition, visual degradation was introduced by randomly removing (at each presentation) 85% of the pixels that 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 (which could be any letter but *A, K, X,* or *Y*) 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 nontarget 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 ms and an interstimulus interval of 1,000 ms.

Procedure

Participants were tested in a single session. Conditions consisted of 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 was then performed as practice prior to administration of the experimental trials for that condition. This ensured that participants understood the instructions and were performing the task appropriately.

Data Analysis

Data were analyzed in each condition 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. For each of the three conditions, analyses of nontarget error rates and RTs were conducted with repeated measures analyses of variance with group (young, old) as a between-subjects 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 nontarget trials were analyzed separately because of their different response requirements (target button vs. nontarget button press) and their different frequencies of occurrence (i.e., 70% for *AX* trials, 10% for each of the nontarget 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). This correction factor (hit rate = $2^{-(1/N)}$; false alarm = $1 - 2^{-(1/N)}$, where N = the number of target or nontarget trials) allows an unbiased estimation of d' in such cases (Nuechterlein, 1991). Instead of the traditional computation of d' (i.e., using hits and all false alarms), d' was computed using just *BX* false alarms. This measure, hereinafter 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 (see Figure 5). 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 RTs 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 RT (Birren, Riegel, & Morrison, 1962; Cerella, 1985; Myerson & Hale, 1993; Salthouse, 1996). Nontarget effects were examined more specifically through an analysis including nontarget trial type (*AY, BX, BY*) as

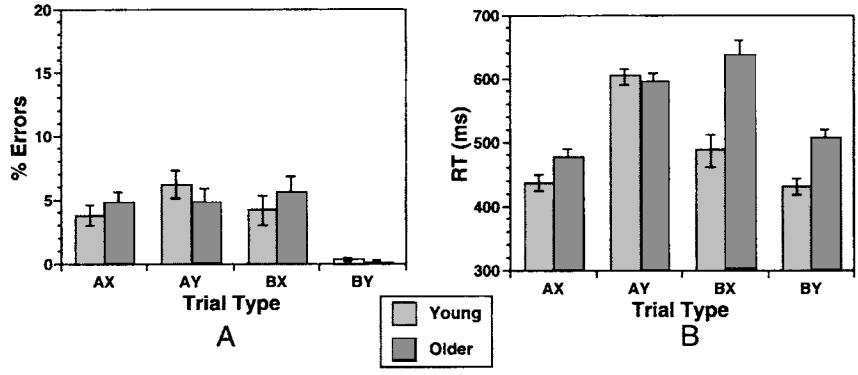

Figure 5. Data from the baseline condition. Performance in each of the four trial types is shown (*AX, AY, BX, BY*). Error bars depict standard errors of the means. A: Percentage of errors for each trial type. There was a trend for greater BX errors but fewer *AY* errors in older adults. B: Reaction times (RTs; correct trials only) for each trial type. Older adults showed disproportionate slowing on *BX* trials (relative to *BY* trials) but comparable response speed on *AY* trials.

an additional factor. As expected, there was a highly significant main effect of this factor on both false alarm rates, $F(2, 508) = 42.1, p < .001$, and RTs, $F(2, 508) = 122.5, p < .001$. This reflected the fact that, compared with the *BY* control trials, performance was significantly worse for both the *BX* and *AY* trial types, in which context competes with response and expectancy biases: *BX* errors, $F(1, 254) = 63.98, p < .001$; *BX* RT, $F(1, 254) = 140.96, p < .001$; *AY* errors, $F(1, 254) = 99.7, p < .001$; *AY* RT, $F(1, 254) = 426.3, p < .001$.

The effect of nontarget trial type was found to strongly interact with the pattern of age-related slowing in RT, $F(2, 508) = 41.8$, $p < .001$ (see Figure 5B). This interaction was further examined through two planned contrast tests in which responses on *BX* and *AY* trials were compared with *BY* trials, because *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 showing was significantly reduced in *AY* trials relative to *BY* trials, $F(1, 254) = 42.7, p < .001$. Moreover, a simple effects test on *AY* RTs suggested that in this condition, there was no evidence of age-related slowing, $t(254) = -0.45, p > .1$, with older adults actually showing numerically faster responses. Both of these results were in the directions predicted by the model. It is also worth noting that age-related slowing on *BX* trials appeared to be quite a bit greater than that observed on *AX* trials (although we did not evaluate this finding statistically). This is also consistent with the model, in that *AX* responses were aided by the induced response bias, whereas *BX* responses must counteract this bias. There was also a trend towards an Age × Trial Type interaction for nontarget errors, $F(2, 508) = 2.45, p = .09$ (see Figure 5A). 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 5A). Moreover, the more focused measure of context sensitivity provided by the *d'*-context measure (i.e., *AX* hits vs. *BX* false alarms) did reveal significantly reduced sensitivity in older adults, $t(254) = 2.38, p < .05$ (see Figure 6).

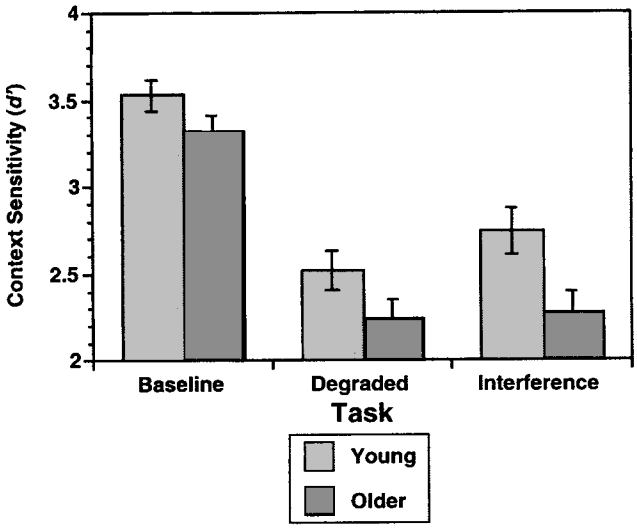

Figure 6. Context sensitivity across the three task conditions. Data are expressed in *d'* units. The greatest age difference occurred in the interference condition. Error bars represent standard errors of the means.

Interference and Degraded Conditions

We next tested the prediction that the interference and degradation manipulations increased the difficulty of the task. Because task difficulty is somewhat difficult to define unambiguously, we adopted the operational criterion that an increase in task difficulty should result in an increase in error rates. We tested effects of task difficulty in the interference and degraded conditions by collapsing across age to examine the main effect of condition on error rates. Error rates were significantly greater for both the interference and degraded conditions relative to baseline: interference target errors, $t(255) = 9.8, p < .001$; interference nontarget errors, $F(1, 254) = 230.1, p < .001$; degraded target errors, $t(255) = 13.8, p < .001$; degraded nontarget errors, $F(1, 254) = 29.4, p < .001$. These results are consistent with our hypothesis 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 (see Figure 7). Most important, there was a significant Age × Condition × 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 × 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 6). 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 RTs also revealed a significant Age × 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 × Condition × Trial Type interaction on nontarget RT was not significant, $F(2, 508) = 2.0, p > .1$. However, when considering the interference condition alone, we found that the Age × 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 direct comparison of the degraded condition to baseline revealed a different pattern than the interference comparison (see Figure 8). The Age × Condition × Trial Type interaction for nontarget errors was not significant, $F(2, 508) = 0.03, p > .1$, nor was the Age × Condition interaction for d' context, $F(1, 254) = 0.53, p > .1$ (see Figure 6). There was a significant Age × Condition effect for target errors, $F(1, 254) = 5.9, p < .001$, reflecting increased age differences in target errors in the degraded

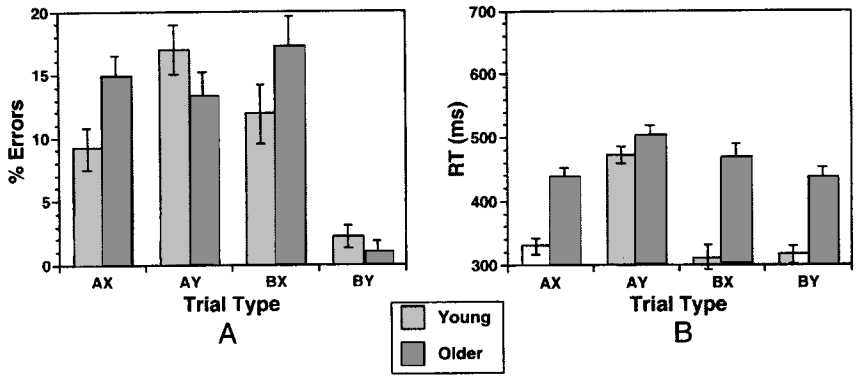

Figure 7. Data from the interference condition. Error bars depict standard errors of the means. A: Percentage of errors for each trial type. Older adults made significantly more errors than did young adults on *AX* and *BX* trials but significantly fewer errors on *AY* trials. B: Reaction times (RTs; correct trials only) for each trial type. Older adults showed disproportionate response slowing on *BX* trials (relative to *BY* trials) but minimal slowing on *AY* trials.

condition. However, the effect of the degraded condition on target errors was no different from that observed under interference as evidenced by a nonsignificant Age × Condition interaction when directly comparing the degraded and interference conditions, $F(1, 254) = 0.2$, $p > .1$. Finally, for RTs, neither the Age × Condition effect for target trials, $F(1, 254) = 1.0$, $p > .1$, nor the Age × Condition × Trial Type effect for nontarget trials, $F(2, 508) = 2.0$, $p > .1$, was statistically significant.

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 nontarget 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, $\beta = .35$, $t(254) = 5.9$, $p < .001$; interference, $\beta = .46$, $t(254) = 8.2$, $p < .001$; degraded, $\beta = .37$, $t(254) = 6.3$, $p < .001$. 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, $\beta = .29$, $t(252) = 2.8$, $p < .01$; interference, $\beta = .32$, $t(252) = 3.3$, $p = .001$; degraded, $\beta = .37$, $t(252) = 4.0$, $p < .001$. Most strikingly, across all three conditions, *AY* response times were found to be negatively associated with age: baseline, $\beta = -.23$, $t(252) = -3.6$, $p < .001$; interference, $\beta = -.14$, $t(252) = -2.2$, $p < .05$; degraded, $\beta = -.31$, $t(252) = -5.1$, $p < .001$. Thus, after

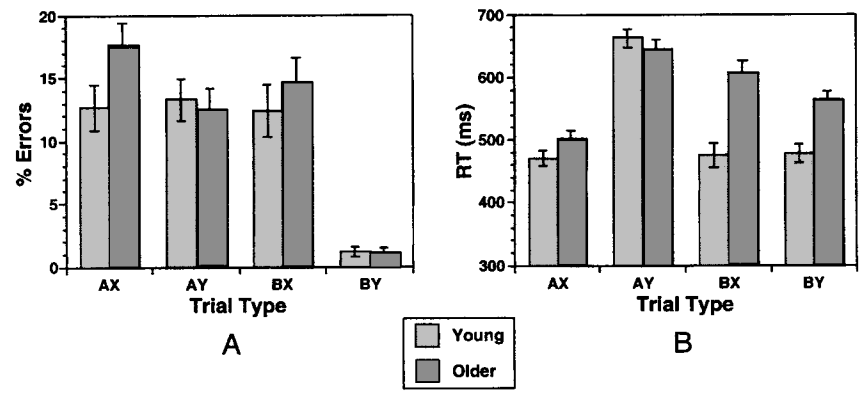

Figure 8. Data from the degraded condition. Error bars represent standard errors of the means. A: Percentage of errors for each trial type. There were no age differences on *AY* and *BX* trials. B: Reaction times (RTs; correct trials only) for each trial type.

accounting for generalized slowing, older age is associated with faster *AY* responses.

As noted in the Method section, there were significant, although modest, reductions in WAIS–R Vocabulary scores among older adults. In order to determine whether this difference may have accounted for the age relationships with RTs described above, we conducted an additional series of hierarchical regressions, in which we forced both WAIS–R Vocabulary scores and *BY* RTs to enter the equation on the first step. We then conducted the regressions as before, with *BX* and *AY* RTs 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 postulating that the representation and maintenance of context is a key mechanism of cognitive control and suggesting that this mechanism is linked to the functional interactions between specific neural systems, the DL-PFC and DA systems. On the basis of 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 support these predictions of the model. Older adults showed impaired performance on *BX* and *AX* trials, thus demonstrating a reduced sensitivity to context. More important, the results also support 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* RT, such that older age is associated with quicker *AY* responses. This latter finding is especially compelling given that the relationship between age and RT has almost always been found to be positive in the cognitive aging literature (Cerella, 1985; Myerson & Hale, 1993; Salthouse, 1996). Finally, the results demonstrate 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 (in terms of the increase in error rates) but did not differentially 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 that could increase the generality of our conclusions. In the final sections, 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

The current study provides an important measure of initial support for our hypothesis that healthy aging is associated with a specific decline in context processing. This hypothesis was formulated based on an explicit computational model that suggests that context processing forms a central element of cognitive control function and, as such, plays an important role in the domains of inhibition, attention, and working memory. Furthermore, the model suggests that context processing impairments can arise as a consequence of dysfunctional interactions between the DA system and DL-PFC. As such, the current results are also consistent with our suggestion that age-related deficits across a number of cognitive domains involving attention, inhibition, and working memory may in fact be due to a common underlying disturbance in context processing and that the neurobiological substrate of this disturbance is dysfunction of the DA system in DL-PFC. However, it must be noted that a limitation of the current study is that it did not directly test these latter two claims of our hypothesis. Moreover, even in terms of the behavioral effects of context processing impairments, the current study did not address whether the full extent of *AX*-CPT context processing deficits predicted by the model is present in older adults. These limitations of the study are discussed in further detail below, along with specific directions for future research.

Representation versus 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 (Braver, Cohen, & Barch, in press). Thus, in the *AX*-CPT participants perform the task under both short- and long-delay conditions. A pattern of context processing impairment that becomes amplified 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, whereas *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 associated only with a deficit in context representation, 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. In particular, we suggest that these different processes reflect the operation of a common mechanism operating under different conditions. 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 that involves all three of these cognitive

processes (working memory, inhibition, attention). For example, performance on *AY* trials reflects the effect of context processing under attentional conditions, whereas *BX* performance reflects context effects under inhibitory conditions. The fact that older adults show performance changes in both conditions is fully consistent with this aspect of the model. However, an important limitation of the study is that it did not establish whether the context processing impairment generalizes across different 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., D. A. Allport, Styles, & Hsieh, 1994) and in which these task instructions are temporally separated from stimulus presentation by a relatively long delay (i.e., 5–10 s). 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 occurs with healthy aging is a direct consequence of disturbances in DA function in DL-PFC. As discussed above, 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 specifically associated with 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 this activation is specifically related to the duration over which context information must be maintained (Barch et al., 1997; Braver & Cohen, 2001). 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 seen in young adults.[1] 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 that DA function in DL-PFC plays in *AX*-CPT performance, it should be possible to increase systemic levels of DA through pharmacological challenges (e.g., administration of DA precursors such as L-dopa or DA agonists such as bromocriptine). The theory suggests that increasing DA levels in older adults should serve to reduce age-related differences in context processing capabilities. However, this prediction is complicated by the possibility that older adults have reduced DA receptor concentration in PFC (e.g., Suhara et al., 1991), which may prevent such pharmacological manipulations of DA levels from being fully utilized there. Nevertheless, 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 standard predictions of this hypothesis are that (a) age-related deficits in cognitive performance will increase as the working memory demands of the task are increased and (b) 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/maintenance of context information is merely identical to or just another way of referring to working memory function. We suggest that the representation/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 on-line maintenance and manipulation of information necessary to perform a cognitive task (Baddeley & Hitch, 1994). Oftentimes, 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 not an *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 *nontarget* button if the next item is an *X*." Thus, one important

[1] We recently obtained preliminary confirmation of this second prediction in an fMRI study of the *AX*-CPT involving healthy older adults (Barch, Braver, Racine, & Satpute, 2001). As predicted by the theory, results indicated that older adults failed to show a delay-related increase in DL-PFC activity during *AX*-CPT performance.

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 on 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) used 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 (1986) 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 (1993) 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 is 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 that 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 subregions. For example, there is now a growing literature that has suggested dissociations between DL-PFC function and the functions of both ventrolateral and ventromedial PFC in terms of the cognitive processes associated with each region.

In particular, on the basis of recent neuroimaging evidence, several investigators have suggested a dissociation between dorsolateral and ventrolateral PFC regions, corresponding to a distinction between maintenance and manipulation in working memory (D'Esposito et al., 1998; Owen, 1997). We have focused on DL-PFC as the locus of context representation and maintenance because of our own neuroimaging results showing that this region shows evidence of sustained activity over delays in the *AX*-CPT and other related working memory tasks (Barch et al., 1997; Braver & Cohen, 2001; Cohen et al., 1997). However, it is possible that maintenance and/or manipulation of context information within working memory may involve both dorsolateral and ventrolateral PFC and that both regions may show age-related disturbances. Likewise, Damasio and colleagues have suggested a dissociation between ventromedial and dorsolateral PFC, hypothesizing that ventromedial PFC regions are involved in the use of emotional and autonomic information to bias decision-making processes (Bechara, Damasio, Tranel, & Anderson, 1998; 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 showed that gray and white matter volume in ventromedial regions of PFC was selectively spared relative to other PFC regions (Salat, Kaye, & Janowsky, in press).

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 to an impairment in inhibition. In particular, this theory, originally proposed by Hasher and 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 to the idea of inhibitory deficits in older adults in that older adults show increased errors and RT interference on *BX* trials. Performance on *BX* trials is critically dependent on 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, whereas 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 to examine 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 on 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

have pointed 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 RTs in older adults can be expressed as a linear function of young adults' RTs 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 RT. 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 RTs. However, we also observed highly significant Age × 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 the baseline *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 RTs on *AY* trials. As just discussed, a primary prediction of processing speed theories is that the condition that generates the slowest RTs should be the one that produces the greatest age differences. Yet in the *AX*-CPT the opposite pattern occurs. Furthermore, we found that *AY* RTs across all three conditions showed a negative correlation with age, once the age relationships 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 RT 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 recently put forth a theory that links at least some cognitive deficits in aging to declines in DA function (Li & Lindenberger, 1999; Li, Lindenberger, & Frensch, 2000; Li, & Lindenberger, & Sikstrom, 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 intraindividual variability in the performance of older adults. The theory of Li and colleagues is highly compatible with 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 and colleagues, 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 and colleagues (Li & Lindenberger, 1999; Li et al., 2000, in press) have focused on the DA system more generally and did not make any claims about specific DA subsystems or projections.

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

On the basis of 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, Carter, et al., 2001; Barch, Carter, Hachten, & Cohen, 1999; Braver, Cohen, & Servan-Schreiber, 1995; Braver et al., 1999a; Carter & Barch, 2000; Cohen et al., 1996, 1999).

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. Third, 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 further research is needed to address these issues. 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 neurobiological 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 counterintuitive—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 is needed to test additional predictions of the theory and to establish its generality.

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