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Neuroscience and Biobehavioral Reviews 26 (2002) 809–817

NEUROSCIENCE AND BIOBEHAVIORAL REVIEWS

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A theory of cognitive control, aging cognition, and neuromodulation

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Received 18 August 2002; accepted 23 August 2002

Abstract

A theory is described which links cognitive changes observed in normal aging to an underlying decline in the function of the dopamine (DA) system projection to prefrontal cortex (PFC). The theory postulates that this neural mechanism is integral to the representation, maintenance and updating of context information, and as such impacts cognitive control across a wide range of cognitive domains, including working memory, attention, and inhibition. Behavioral and brain imaging data in support of the theory are discussed, which demonstrate selective impairments in context processing among healthy older adults associated with abnormal PFC activation. These findings highlight the utility of a computational approach to cognitive aging. Current directions for further refinement and validation of the model are outlined.

Keywords: Computational model; Prefrontal cortex; Dopamine; Aging; Executive control; Working memory; Brain imaging; Functional magnetic resonance imaging

1. Introduction

An important goal in the study of cognitive aging is to discover potentially unifying underlying mechanisms that contribute to the diversity of cognitive changes occurring with advancing age. However, this endeavor is a difficult one because it is not usually clear how to link the observable behavioral phenomena typically found in older adults (e.g. slowing response time, poorer performance on laboratory tasks) with latent psychological and/or neurobiological changes. Computational models provide an important tool that can be used to demonstrate how particular changes in underlying cognitive and/or neurobiological mechanisms can lead to a wide range of observable behavior changes. As such, the application of computational models to the study of cognitive aging has the potential to advance our understanding of the core mechanisms that lead to cognitive changes in healthy aging [24]. In our work, we have focused on the mechanisms of cognitive control, how these mechanisms influence behavior across a wide range of cognitive domains, and how they are supported by specific neural systems (i.e. prefrontal cortex (PFC) and the dopamine system). Recently, we have applied this approach towards understanding age related changes in cognitive control, and how such changes might be linked to age-related changes in PFC and dopamine function. Here we present a brief overview of our theoretical model of cognitive control, how the model accounts for the nature of aging cognition, and the ability of this model to capture specific patterns of behavioral performance among older adults on particular cognitive tasks.

As discussed in the other articles in this issue, a large literature on cognitive function in healthy aging suggests that older adults display deficits in multiple different cognitive domains, including episodic memory, working memory, prospective memory, inhibition, attention, and 'executive' function [3,16,21,28,35,43]. In our own work, we have suggested that there is a common element to all of these cognitive domains. Namely, that they place a heavy load on cognitive control. More specifically, we propose that successful performance in a wide variety of cognitive situations centrally depends upon the internal representation, maintenance, and updating of *context information* in the service of exerting control over thoughts and behavior [10,12,15]. 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

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PII: S0149-7634(02)00067-2

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 (which involves naming the ink color of color-named words, e.g. the word RED in green ink), 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 rather than the word name. 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 which 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 [2], which postulate a strict separation of representations for storage versus control.

We further suggest that context processing and cognitive control are subserved by a specific set of underlying neural mechanisms. In particular, we postulate that representations of context information are housed within the dorsolateral portion of the prefrontal cortex (DL-PFC) and actively maintained there when task demands require such active maintenance [30]. The dopamine (DA) projections to DL-PFC are postulated to regulate the access to such context information, insulating this information from the interfering effects of noise over intervals in which the information must be sustained, while at the same time allowing for the appropriate updating of such context information when needed [10]. These assertions are consistent with the neuroscience literature, in which active maintenance in the service of control is a commonly ascribed function to PFC [18,19,27], and the DA system is widely held to modulate the active maintenance properties of PFC [25,37,44]. In our model, the context processing functions of cognitive control critically depend upon DL-PFC and DA system interactions. As a consequence, the model predicts that individuals and populations with impairments in either or both DL-PFC or the DA system should demonstrate specific patterns of impaired cognitive control related to the processing of context.

A growing literature on the neurobiology of healthy aging suggests the PFC and DA systems are among the most strongly affected by increasing age [1,13,31,32,42]. We have explored the use of our theoretical model of cognitive control as a tool for understanding the nature of aging cognition, and to bridge the gap between neurobiological and cognitive findings of age effects. Next we describe in greater detail our model of cognitive control and how it might be used to simulate the cognitive and neurobiological consequences of healthy aging.

2. Model of cognitive control

Our modeling work utilizes 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 [34]. 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.

Our theory of cognitive control 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 (Fig. 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 their competitors. This biasing action of the context module can produce inhibitory effects on processing, by enhancing the activation of an otherwise weak pathway and enabling it to compete effectively with a more dominant one.

The third critical feature of the context module is the modulatory input which reflects the processing functions associated with DA projections into DL-PFC. In other work, we have provided a detailed description of this processing

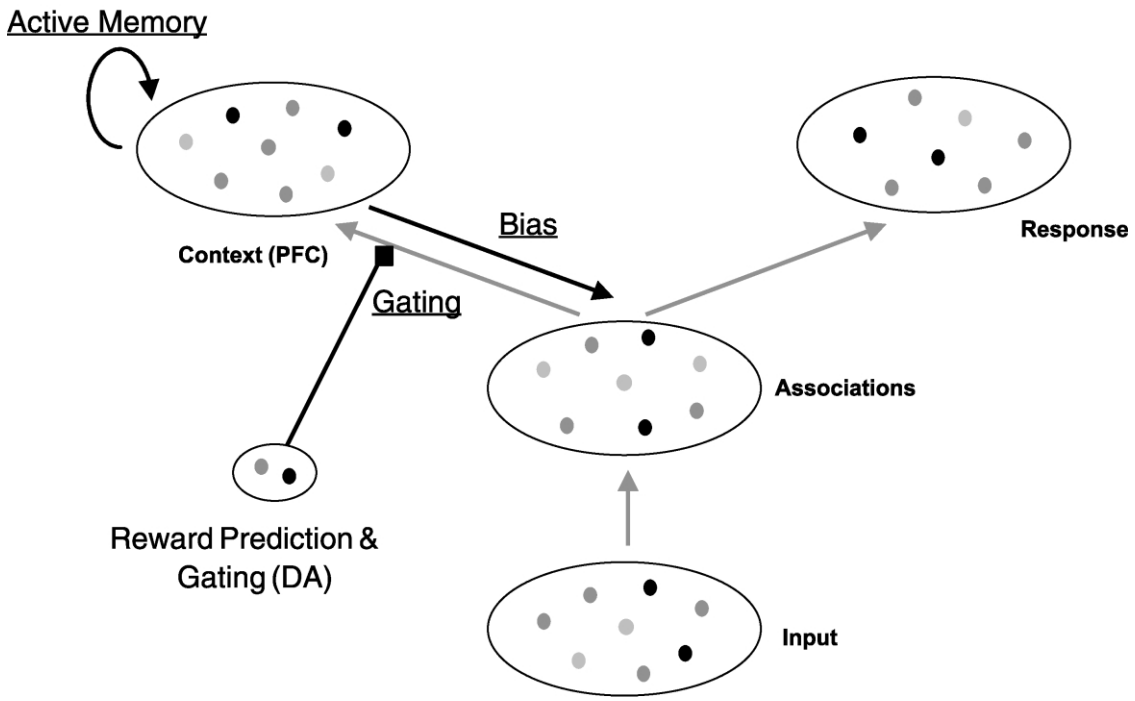

Fig. 1. Diagram of canonical model. Key computational principles of context processing mechanism are shown: (1) active memory through recurrent connections; (2) top-down bias through feedback connections; and (3) regulated access of contextual input through modulatory gating connections.

mechanism [9,10]. 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, which enables selection of task-relevant information as context, due 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. We hypothesize 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 disrupting DA effects in the context module affects the representation of context, by making it less reliable (since access is partially blocked). Moreover,

even when context representations do get activated, disruption of DA effects can also cause the maintenance of those representations may decay more quickly over time (since information is more susceptible to the interfering effects of noise and task-irrelevant inputs) [11]. 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. In our previous work, we have provided empirical support for our hypotheses across a number of studies in healthy young adults, as well as in other populations thought to suffer from cognitive control impairments (e.g. schizophrenia patients) [4,6,8,9,11,12].

A particular focus of this empirical work has been the use of an experimental paradigm we have developed to enable selective measurements of cognitive control and context processing functions. The paradigm, known as the AX-CPT, is a modified version of the classic Continuous Performance Test or CPT [33]. In the AX-CPT, information from a contextual cue is required to drive correct responding to a subsequent ambiguous probe item. Thus, target trials occur when the cue 'A' is followed by the probe 'X'. However, on trials in which the same 'X' probe occurs without being preceded by the 'A' cue, a non-target response must be given. These 'BX' trials provide an index of the integrity of context representation. The demands on context representation are made even stronger by the fact that 'AX' trials occur with high frequency. This leads to a dominant tendency to make a target response to the 'X' probe. This dominant response tendency must be inhibited on BX trials, but such inhibition can only occur if the preceding context (e.g. 'A' or 'non-A' cue) is properly represented. The high frequency of targets also enables the contextual cue to drive expectations about upcoming responses. Thus, the appearance of an 'A' cue can prime or bias attention toward the target response prior to the onset of the probe. When this occurs, but the probe is not an X ('AY' trial), such context-driven expectancies (if robustly maintained) can lead to an increased tendency to false alarm. Manipulations of the delay between the cue and the probe also provide a means for examining not just context representation, but also how well context information can be maintained over time. Impairments in maintenance should lead to greater context processing failures following a long delay than at short delays. It is important to note that context serves opposite functions on BX and AY trials. Specifically, context information improves performance on BX trials, but tends to impair performance on AY trials. The consequence of this pattern is that individuals with intact context processing abilities should show relatively good performance on BX trials, but poor performance no AY trials. In contrast, in individuals with impaired context processing capabilities, AY performance should be relatively good while BX performance should be poor. If context maintenance functions are also disrupted, these patterns should further interact with the delay between the cue and the probe.

We have applied our cognitive control model to the AX-CPT, simulating task performance under both intact and impaired conditions [6,11]. In the model, the contextual cue information is represented and maintained within the module that corresponds to DL-PFC function. The updating of such context information is regulated by the component of the model that corresponds to midbrain DA projections to PFC. The intact model provides a good account of both behavioral performance and DL-PFC activity dynamics in healthy young adults. Behaviorally, the model captures the relationship between AY and BX performance (i.e. AY > BX, for both errors and RT), the interaction of these effects with cue-probe delay duration (i.e. AY performance worsens with delay, while BX performance slightly improves), as well as a number of more subtle effects [6]. At the neurobiological level, we have supported the model by finding that: (1) DL-PFC regions are engaged during AX-CPT performance and shows greater activity in the long delay [4,11]; and (2) DL-PFC activity shows sustained activity of dynamics throughout a long cue-probe delay period (but not an equivalent length inter-trial interval) of the AX-CPT, consistent with a role for this region in active maintenance functions [11]. Finally, we have also provided preliminary support for the role of DA in AX-CPT performance in a pharmacological challenge study [6], in which AX-CPT performance improved under placebo-controlled administration of low-dose D-amphetamine (which acts to stimulate central DA release).

We have also used the cognitive control model of the AX-CPT to simulate performance under impaired conditions (for further information regarding the computational details of these simulations, see Refs. [6,9,11]). As discussed above, we hypothesized that the functioning of the DA projections to DL-PFC serve as a critical underpinning of intact cognitive control. Thus, when this projection is disrupted, one should find impairments in context processing. Consistent with this hypothesis, we have found that reducing context processing functions through simulation of reduced DA effects in DL-PFC produces particular the pattern of impaired behavioral performance described above [6]. For example, when DA projections to DL-PFC are altered, there is more BX than AY errors, and this effect becomes amplified with delay. A similar pattern occurs for RTs (i.e. more slowing of BX than AY RT, and an amplification of this effect with delay). The 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 have begun to empirically test these predictions of the model regarding age-related changes in context processing and cognitive control through studies of behavioral and

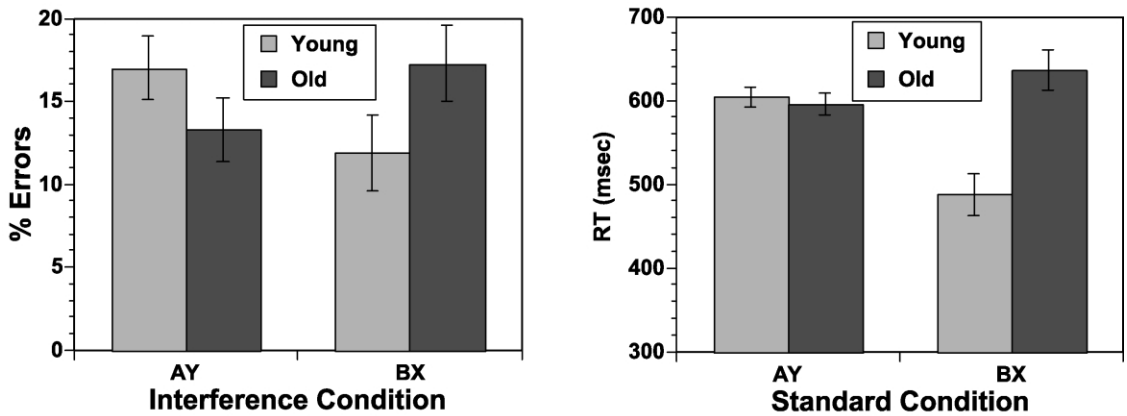

Fig. 2. Aging and AX-CPT Performance. Behavioral data from a study involving 175 younger adults (age range, 18–39; dark bars) and 81 healthy older adults (age range, 65–85; light bars) and. Left panel: error data from interference (high context demand) condition. Older adults show significantly more BX errors, but significantly fewer AY errors ($p < 0.05$). Right panel: reaction time data from standard condition. There is highly significant age-related slowing on BX trials ($p < 0.001$), but no slowing on AY trials ($p > 0.5$).

brain activity differences between younger and adults on experimental tasks. Ideally, such studies would be conducted longitudinally to provide a strong test of our hypotheses. However, given the practical complexities of such studies, we instead have begun our empirical testing using cross-sectional data. In our first behavioral study, we found that the impaired cognitive control model provides a good account of the performance patterns found in healthy older adults on the AX-CPT (Fig. 2; [7]). In this study, 175 young adults (age range, 18–39) and 81 older adults (age range, 65–85) were studied. Healthy older adults produced more BX than AY errors. Moreover, this pattern and the contrast in performance patterns between younger and older adults was amplified under conditions that placed even greater demands on context processing (i.e. an interference version of the task in which distractor stimuli were presented during the cue-probe delay). Specifically, in the interference condition, the older adults produced significantly more BX errors than young adults, but significantly *fewer* AY errors. The most striking behavioral finding was that on AY trials, the reaction times of older adults were equal to or faster than those of healthy young adults (i.e. RT differences were not significant). This pattern was somewhat remarkable given the near ubiquity of reaction time slowing found in older adults performing cognitive tasks. This last counter-intuitive finding (which was predicted by the model) provides a high degree of support for the cognitive control model and its application to healthy aging, since it is unclear how one would predict or explain such a result without the aid of an explicit theoretical model.

3. Current empirical directions

In our current work, we are continuing our exploration of the model and its ability to account for both behavioral performance and brain activity in older adults. Specifically, our initial behavioral study with the AX-CPT in older adults left a number of issues unaddressed. A first issue is that the impaired cognitive control model predicts that older adults will show deficits related to both the representation of context and the maintenance of this information over time. The cognitive control model suggests that relationship between context representation and context maintenance can be examined through the influence of cue-probe delay on AX-CPT performance. If context representation and maintenance are both disturbed, then age-related changes in AX-CPT performance should be greatest following a long delay relative to a short delay. In contrast, if context maintenance is intact, then older adult's performance should not be differentially affected by delay. To examine this issue, we recently completed a behavioral study with the AX-CPT in which we manipulated the delay between the cue and the probe (either 1 or 5 s) [36]. Interestingly, we found a complex pattern of results such that within healthy older adults, performance patterns were influenced by increasing age (Fig. 3). Specifically, we divided the healthy older adults into 'young-old' and 'old-old' groups. Young adults displayed the typical context processing advantage for BX trials (i.e. BX performance significantly better than AY, in both errors and RT). However, in both older adult groups this context processing advantage was absent (i.e. BX performance worse or equal to AY performance in both errors and RT), suggesting a disturbance in context representation. Critically, we found differences between the two older adult groups in terms of context maintenance. In the young-old group context maintenance was intact (e.g. performance was not affected by delay). In contrast, in the old-old group, we found that both context representation and maintenance were disturbed, such that BX performance significantly worsened with delay, while AY performance actually significantly improved. These results suggest that context representation and maintenance may reflect dissociable cognitive control functions that are differentially

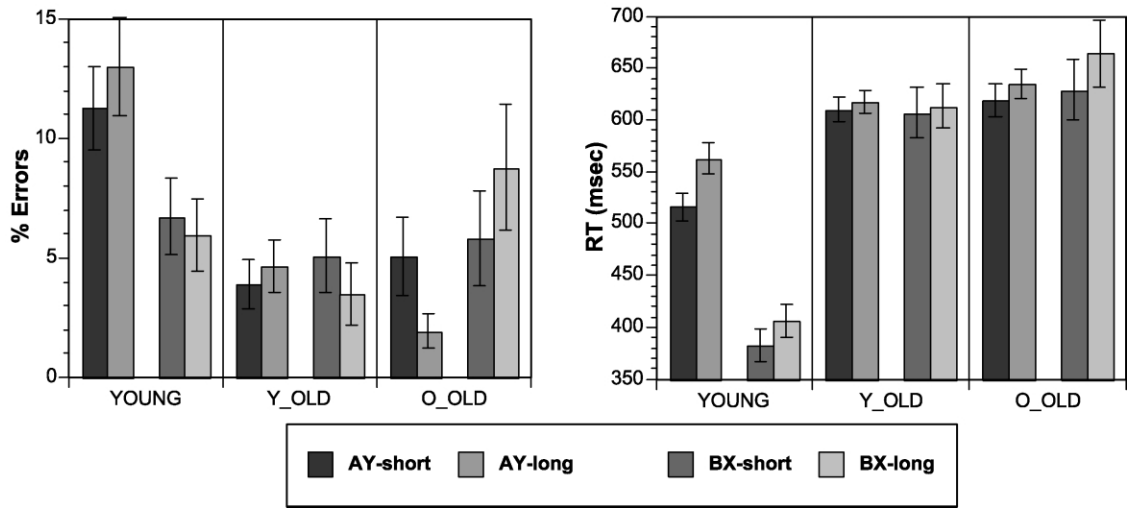

Fig. 3. Effects of age and delay within older adults in the AX-CPT. Behavioral data from a study involving 51 younger adults (YOUNG: left panel; age range, 18–39), 48 'young-old' adults (Y_OLD: middle panel; age range, 66–75; light bars) and 41 'old-old' adults (O_OLD: right panel; age range, 76–92). In young adults, BX performance was significantly better than AY in both errors and reaction time ($p < 0.001$), but this effect was not present in either older adult group. In both older adult groups relative to YOUNG, AY performance was improved (primarily in errors, $p < 0.001$), but BX performance was worsened (primarily in reaction time, $p < 0.001$). The Y-OLD group showed the same delay effect as the YOUNG group (AY performance worsening, but BX improving). However, the O_OLD group showed an inverted delay effect relative to the other two groups, especially in terms of errors (observed as an age x trial type x delay interaction; $< .05$).

vulnerable to increasing age. As such, the findings indicate that some elaboration may be needed to the basic cognitive control model in order to fully specify the nature of dissociability in the neurocomputational mechanisms supporting these two functions. We touch on this point further below.

A second important issue relates to the generality of scope of our model. We have suggested that declines in context processing and cognitive control function in healthy aging will impact performance across a range of cognitive domains and tasks, including attention, working memory, and inhibition. In other words, one question is whether context processing functions play the same role in other cognitive control tasks across different domains that they do in the AX-CPT task. One way in which to address this question empirically is to determine whether performance on the AX-CPT in older adults is correlated with performance on other tasks thought to measure constructs related to cognitive control such as working memory, inhibition and attention. If older adults performance on the AX-CPT is predictive of their performance on other cognitive control tasks, this would suggest that the context processing constructs measured by the AX-CPT generalize across domains. Our initial effort to investigate this issue involved a study comparing AX-CPT performance in older adults to other commonly used tasks of working memory, such as the N-back, reading span and digit span tasks [22]. Consistent with our hypothesis, older adult's AX-CPT performance was significantly correlated with their performance on all three other tasks. The next step will be to examine performance inter-relationships with tasks in other cognitive domains, such as attention, inhibition and episodic memory.

A third critical issue is that the theory of cognitive control predicts that declines in context processing capabilities that occur with healthy aging should be associated with disturbances in DA function in DL-PFC. The behavioral results that we have obtained with the AX-CPT task are consistent with the idea that the age-related impairments in context processing are related to DL-PFC and DA dysfunction. However, to test this hypothesis more directly, we have used functional magnetic resonance imaging (fMRI) to examine the pattern of DL-PFC activity in older adult's while they perform the AX-CPT [5]. In numerous prior studies with healthy young adults, we have found that left DL-PFC activity appears to critically support the representation and maintenance of context, as activity levels in left DL-PFC track with the length of time context information needs to be maintained [4,8,11]. However, consistent with the predictions of the impaired model, healthy older adults do not show this same pattern of left DL-PFC activity (Fig. 4). Older adults show greater left DL-PFC activity than younger adults at the short delay, but showed decreased activity at longer delays, in contrast to the typical increase in DL-PFC activity observed in young adults associated with increased context maintenance demands. Importantly, the failure to show a context related increase in DL-PFC activity occurred in the face of a generalized increase in task-related brain activity among older adults. This suggests that DL-PFC is selectively involved with impaired context processing function during performance of the AX-CPT. However, this initial neuroimaging study used a blocked task design that did not allow us to track the dynamics of brain activity over the course of a trial. Thus it will be important to know whether

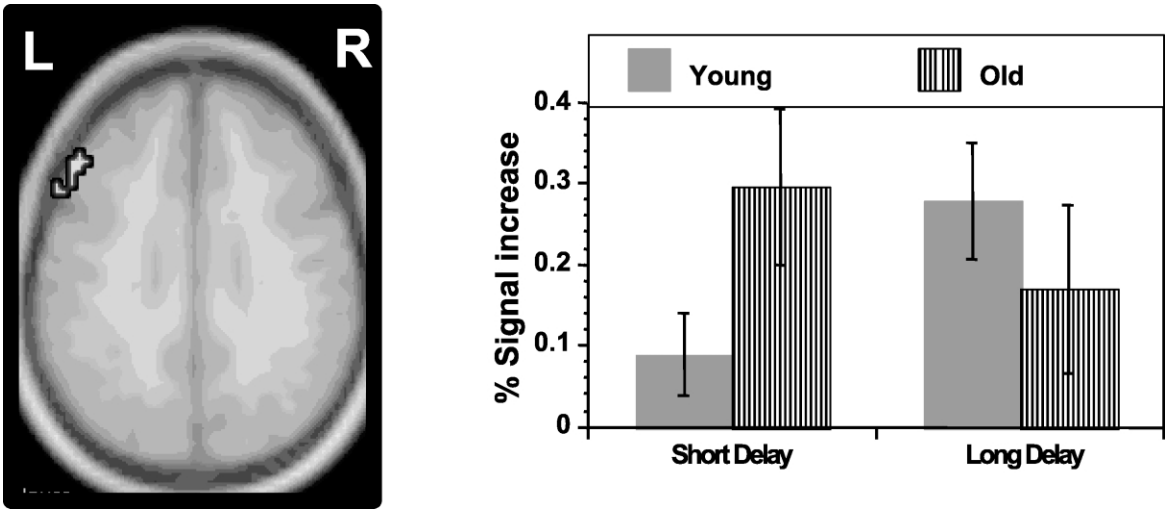

Fig. 4. AX-CPT brain activity. Group data from a fMRI study involving 21 young adults (age range, 18–31) and 20 older adults (age range, 66–83). Left panel: locus of age-related activation changes in left hemisphere DL-PFC (Brodmann's Area 46/9). Functional data of DL-PFC region showing significant age *x* delay interaction ($p < 0.05$, corrected) are superimposed on an anatomical image of an axial brain slice located 33 mm superior to the AC-PC plane. Right panel: DL-PFC activity levels (indicated as percent change in fMRI signal relative to a fixation control condition) in short and long delay AX-CPT conditions for both young and old adults. Activity demonstrates significant delay-related activation increase in young adults ($p < 0.05$), but delay-related decrease in activity in older adults.

the age-related changes in DL-PFC activity during AX-CPT performance reflect changes in maintenance-related activity, but intact initial activation following cue presentation, or whether even the initial activation of DL-PFC is impaired in older adults. Dynamic measures of PFC activity are possible with event-related functional neuroimaging designs, and such studies represent an important next phase of research.

4. Current theoretical directions

In Section 3, we attempted to provide greater specification regarding empirical support for the model of cognitive control and its ability to account for cognitive and neurobiological changes associated with healthy aging. Here we discuss our current thinking with regard to the neurocomputational mechanisms that we believe support cognitive control. The model of cognitive control we have put forward postulates that DA projections to DL-PFC play a crucial role in supporting context processing functions. Further, we have hypothesized that impairments in these DA projections are responsible for age-related changes in context processing. An important question though is the precise mechanisms by which DA may modulate DL-PFC and the cognitive and computational consequences of such modulation.

Growing evidence suggests that the midbrain DA system signals information in terms of both tonic and phasic activity changes, and that these two types of activity dynamics are functionally dissociable [20]. For example, chronic stress appears to impact the level of tonic DA release [26,41], whereas the occurrence of unpredictable, rewarding events triggers phasic changes in DA activity [38]. Recent primate studies have suggested that phasic DA activity may be critically important for operant conditioning, by allowing for learning of conditioned stimuli that predict future reinforcement [39]. Our recent work has suggested that tonic and phasic DA activity might have also different computational effects on cognitive control function. Specifically, we have postulated that phasic bursts of DA activity may trigger the updating of context information in PFC, by signaling the presence of salient (i.e. reward-predictive) information in the environment that should be represented as context [10]. In contrast, the tonic level of DA activity may serve an important role in the active maintenance of context information. Like others, we have suggested that tonic DA activity might alter the responsivity (i.e. gain) of DL-PFC neurons to other inputs [23,40]. If activity is maintained via recirculation among local recurrent connections in DL-PFC, then such tonic DA-mediated gain modulation would serve to enhance the robustness and stability of such maintenance [17]. Thus, DA may support both the updating and maintenance of context information, but via different neural and computational mechanisms [14].

Interestingly, such an account of the role of DA in context representation versus maintenance might also provide an explanation for our recent findings of age-related dissociations in AX-CPT performance within healthy older adult populations. As discussed above, we have recently found evidence that young-old adults are impaired in context representation, but not maintenance, while old-old adults are impaired in both functions. One possible explanation of this data is that the DA system shows a differential time-course of decline in the fidelity of

phasic versus DA activity, with phasic DA responses being most vulnerable to increasing age and tonic DA responses only becoming disturbed with more advanced age. Thus, young-old adults may show impairment in context representation related to altered phasic DA activity, but may not yet show impairments in context maintenance because the tonic DA system is still relatively intact. However, with further advancing age, the old-old adults may show both context representation and maintenance difficulties because both tonic and phasic DA activity are impaired. Of course, this intriguing hypothesis will requires testing and validation via additional simulation studies, which are currently ongoing in our lab.

5. Conclusions

A complete understanding of cognitive aging will require a linkage of neurobiological changes to behavioral ones and an explicit mechanistic account of how these two levels of description inter-relate. There is a growing appreciation for the use of computational modeling approaches to bridge the gap between behavior and biology in the study of cognitive aging [24,29]. Like other investigators using such approaches, we have focused on the DA system and DL-PFC as critical neurobiological loci underlying age-related cognitive declines. Our model suggests that the DA and DL-PFC subserve a very specific cognitive function, that of enabling the representation, maintenance and updating of task-relevant contextual information. We suggest that in normal aging, the changes occurring in the interaction of the DA system with DL-PFC produces a disruption of context processing, that impacts cognitive control functions across a wide range of cognitive domains, including working memory, attention and inhibition. We have begun an exploration of the predictions of the model in a number of recent behavioral and brain imaging studies. These studies have supported the notion of a selective impairment in context processing in healthy older adults, and that this impairment may be tied to abnormal activation of the DL-PFC. Our work to date only provides a first-step towards understanding the role that context processing disturbances, and abnormal DA-PFC interactions play in cognitive aging, and it is likely that many refinements will be needed to the model, some of which we are already beginning to explore. Nevertheless, we firmly believe that a computational approach will provide a strong degree of leverage and explanatory power needed to make sense of the complexity and diversity of cognitive changes that occur with advancing age.

Acknowledgements

This research was supported by NIA AG18138. The authors thank Martha Storandt for guidance and support in conducting behavioral studies, and Beth Keys, Carrie Racine and Ajay Satpute for assistance in data collection and analysis.

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