Unifying cognitive aging: From neuromodulation to representation to cognition

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Abstract

An integrative theory relating cognitive aging deficits observed at the behavioral level with age-related deficiency in neuromodulation causing less distinctive cortical representations is tested in a series of neural network simulations. Age-related attenuation of catecholaminergic function is simulated by lowering the mean gain of the processing unit, which subsequently reduces responsivity and raises intra-network activation variability. Age differences in learning rate, asymptotic performance, interference susceptibility, complexity cost, *intra*- and *inter*-individual variability, and ability dedifferentiation can all be modeled. Together, the simulations illustrate catecholamine's role in regulating the fidelity of neural information processing and subsequent effects leading to cognitive aging deficits. © 2000 Elsevier Science B.V. All rights reserved.

Keywords: Cognitive aging; Neuromodulation; Catecholamine; Neural networks; Gain parameter

1. Introduction

Declines in basic mechanisms of cognition pervade the aging process. Behaviorally, age differences in processing speed [26], learning rate [16], asymptotic performance [4,27], interference susceptibility [16,18], complexity cost [17] and *intra*-individual [11] as well as *inter*-individual variability [23] are robust effects. Biologically, brain

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aging involves neuroanatomical and neurochemical changes [29]. Accounts postu-
lated at either the information-processing level assuming age-related reduction in
general processing resources [25] or at the biological level hypothesizing age-related
increase in neural noise [34] were proposed as mediating factors for cognitive deficits
observed behaviorally. However, thus far data and explanations of cognitive aging
deficits have been mostly confined within one (or two) of these levels.

As an attempt to facilitate integration, we offer a cross-level theoretical conjecture
aiming at integrating findings of age-related decrements in catecholaminergic func-
tion, the functional properties of catecholaminergic modulation, catecholaminergic
effects on neural information processing, and various benchmark behavioral manifes-
tations of cognitive aging. Empirical evidences from different levels are reviewed as we
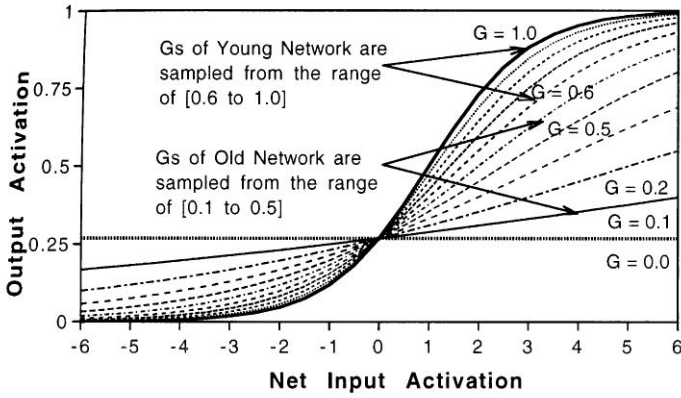unfold and evaluate our conjecture.

2. Aging, neuromodulation and information processing

The relationship between cognitive aging and age-related deficiency in neuro-
modulation has recently become an important topic in aging research. Given their
roles in modulating prefrontal cortical (PFC) cognitive functions [2], age-related
depletion of catecholamine, consisting of dopamine (DA) and its metabolic products,
norepinephrine (NE) and epinephrine, is of specific interest. There is consensus of
age-related decline in dopaminergic function in the striatum, basal ganglia, and
prefrontal cortex (PFC) both in terms of transmitter contents and binding mecha-
nisms [8,12,13,29]. There is also evidence for functional relationships between age-
related deficits in the catecholaminergic system and age-related decrements in differ-
ent aspects of cognitive functioning, such as processing speed [21] and working
memory [28]. In the light of these findings, some researchers have suggested that
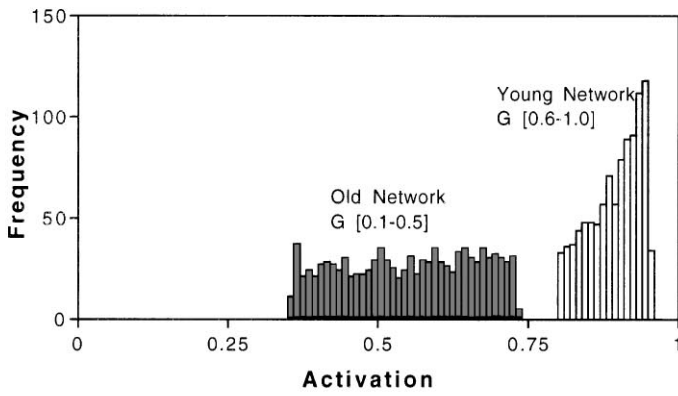catecholamine mechanisms play important roles in cognitive aging [1,12,33].

Fig. 1. (A) The logistic activation function at different values of G. The logistic function is strictly for all
value of $G > 0$ and all values of the bias. Physiological evidence suggesting that the function relating the
strength of an input signal to a neuron's firing rate is sigmoid, with its steepest slope around the baseline
firing rate. In addition, small increments in excitatory signals produce greater changes in firing frequency
than those produced by the same amount of increment in inhibitory signals. These properties can be jointly
captured by the logistic function with a constant negative bias [5,10]. The bias of the logistic function is set
to -1.0. Reducing G flattens the activation function such that a unit becomes less responsive: with
a decrease in G, the effect of the net input is decreased both for excitatory and inhibitory signals.
Subsequently, a G-reduced unit is also less responsive to the difference between signals. For instance, two
different signals, $+1$ and -1, produce a much greater difference in output activation when $G = 1.0$ (0.5 vs.
0.12) than when $G = 0.1$ (0.29 vs. 0.25); and when $G = 0$ (only as an example of the limiting case) the unit's
response remains always at its baseline activation. Given a same range of variability in Gs, reducing mean
G also increases the variability in output activation. (B) Reducing G and the temporal variability of
activation. Reducing mean G (0.8 and 0.3 for the "young" and "old" networks, respectively) increases the
temporal variability of a unit's response to an identical signal (was set to 4.0) across 1000 trials. (C) Internal
activation patterns across five intermediate units of one young (mean $G = 0.8$) and one old (mean $G = 0.3$)
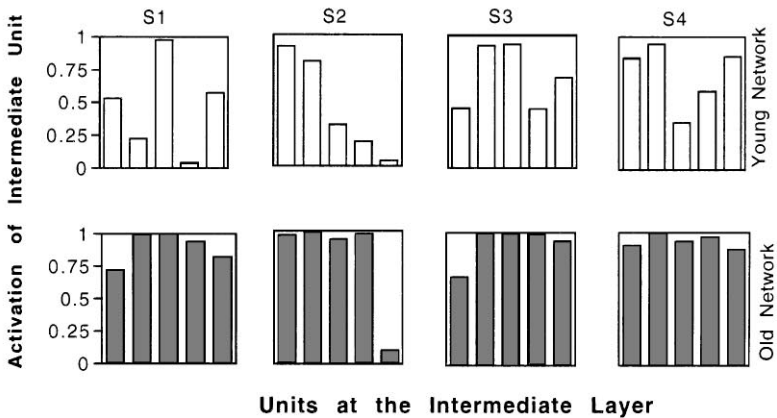network after learning four different stimuli.

A

Gs of Young Network are sampled from the range of [0.6 to 1.0]

Gs of Old Network are sampled from the range of [0.1 to 0.5]

G = 1.0

G = 0.6

G = 0.5

G = 0.2

G = 0.1

G = 0.0

Output Activation

Net Input Activation

B

Frequency

Old Network G [0.1-0.5]

Young Network G [0.6-1.0]

Activation

C

Activation of Intermediate Unit

S1   S2   S3   S4

Young Network

Old Network

Units at the Intermediate Layer

3. Modeling age-related attenuation of catecholaminergic function

Outside of cognitive aging research, there are quite a few formal models of neuromodulation [7]. However, to our knowledge no research effort has yet been devoted to instantiate formal models that link aging-induced changes in catecholaminergic modulation with a broad class of human cognitive aging phenomena at the behavioral level. In this study, we computationally investigate general principles of neural mechanisms linking catecholaminergic modulation of cortical neuron's responsivity to the distinctiveness of cortical representation and cognitive aging deficits.

Catecholamine's modulatory roles are manifold, depending on cortical regions, receptor types, and the techniques and concentrations of drug admission. In some cases, it has been found that catecholamine enhances a neuron's postsynaptic responsivity to afferent signal [6,9]. Or as suggested by more recent evidence, dopaminergic modulation in the PFC can be extremely precise, affecting a specific component of the PFC neuron's excitatory and inhibitory afferent input, without altering the neuron's general excitability [35]. These diversities notwithstanding, a general feature of catecholaminergic modulation can be more abstractly conceptualized as altering the signal-to-noise ratio of cortical information processing [6,9,30]. This general effect can be modeled by adjusting the gain parameter (G) of the logistic activation function of processing units in feedforward backpropagation networks [30].

In previous studies, once the value of G was reduced during the testing phase to simulate attenuated catecholaminergic function underlying cognitive deficits of schizophrenia [30] or increased to simulate the effect of D-amphetamine on mechanisms of selective attention [31], the same G was applied to all units in the presumably affected module(s), and was kept constant across all processing steps. In order to incorporate evidence of random fluctuations in transmitter substance caused by probabilistic transmitter release [14,15], we propose that rather than having a static G, each processing unit's G is to be randomly sampled at each processing step during training and testing. Hence, the activation function of a unit is defined with a stochastic G that varies its value slightly at each processing step:

$$\left\{ \begin{array}{l} A_{i_t} = \dfrac{1}{1 + e^{-(G_{i_t} \times \mathrm{Net}_{i_t} + \mathrm{bias})}} \\[2em] \text{with } G_{i_t} \in [G_{\min}, G_{\max}], \; G_{\min} > 0, \quad \mathrm{Net}_{i_t} = \sum_j W_{ij} I_j \\[1em] \text{where at each } t, \; I_j \text{ is the } j\text{th input, and } W_{ij} \text{ is the connection weight.} \end{array} \right\}$$

Age-related decline in catecholaminergic function can then be simulated by reducing the mean of the uniform distribution from which the Gs of the units are sampled, while keeping the range of G constant.

3.1. Reducing G reduces responsivity, raises variability, and reduces representational distinctiveness

Reducing mean G flattens the logistic activation function, such that a unit's average responsivity is reduced (Fig. 1A). Subsequently, a G-reduced unit becomes less discriminative in responding to differences in inputs. In addition, reducing mean G also increases the variability in the unit's output activation. Fig. 1B shows that for a given unit, reducing mean G increases intra-network temporal variability. Similarly, when the Gs of different units in a network are randomly sampled, then the same magnitude of variability in Gs produces a greater amount of variability in output activation across units when the values of Gs are in a lower range. Conceptually, this implements a larger amount of random spatial variability of activation across units.

Note that a given amount of variability in G, simulating random fluctuations in transmitter substance, causes a greater amount of variability in output activation when the values of G are in a lower range. It is a common view that cognitive aging deficits are associated with age-related increase in neuronal noise [34]; however, mechanisms for age-related increase in neuronal noise are not explicated. The interaction between the mean magnitude of G and the effect of stochastic variation in G on intra-network variability computationally depicts a potential neurochemical mechanism for age-related increase in neural noise: as aging attenuates the efficacy of neuromodulation, random fluctuations in transmitter substances due to probabilistic transmitter release have a greater impact on the overall level of random variability in the aging brain.

Furthermore, reduced responsivity and increased intra-network variability subsequently affect the distinctiveness of the network's internal representations. To illustrate, activation patterns across the intermediate units of one "young" and one "old" network after learning different input–output pairs are plotted in Fig. 1C. Clearly, the activation patterns of the old network in response to different input signals are *less* distinctive than the activation patterns of the young network.

Together these results show that reduced responsivity of the units and increased intra-network variability as caused by mean G reduction subsequently give rise to less differentiated (i.e., more *inter*-dependent) internal stimulus representations at the intermediate layer. One potential biological implication of this property is that as age-related catecholamine deficiency drives down cortical neurons' responsivity and raises the level of random variability during neural information processing, cortical representations elicited by different stimuli become less differentiable.

4. Simulations linking catecholaminergic modulation with behavioral data

The conjectured theoretical path from age-related decrement in catecholaminergic function to higher levels of random variability in the aging brain then to less-distinctive cortical representation and cognitive aging deficits is examined with respect to multiple constraints from benchmark behavioral data. The effects of mean G reduction in accounting for typical cognitive aging phenomena are tested in a series of simulations. To broadly sample different aspects of the behavioral data, the

simulations include age effects on mean performance (see 4.1–4.3), variability in performance, and covariations between performances (see 4.4). All simulations involve two groups of otherwise identical networks that *differed only* in the mean values of their Gs (mean $G_{young\ network} = 0.8$ and mean $G_{old\ network} = 0.3$; for additional implementation details refer to Li and Lindenberger [19] and Li et al. [20].

4.1. Aging, learning rate and asymptotic performance

Memory research shows that the number of trials required for learning paired-associates (i.e., arbitrary word pairs, such as computer–violin) increases dramatically with advancing age [16,22]. Fig. 2A shows that, like the older subjects, the "old"
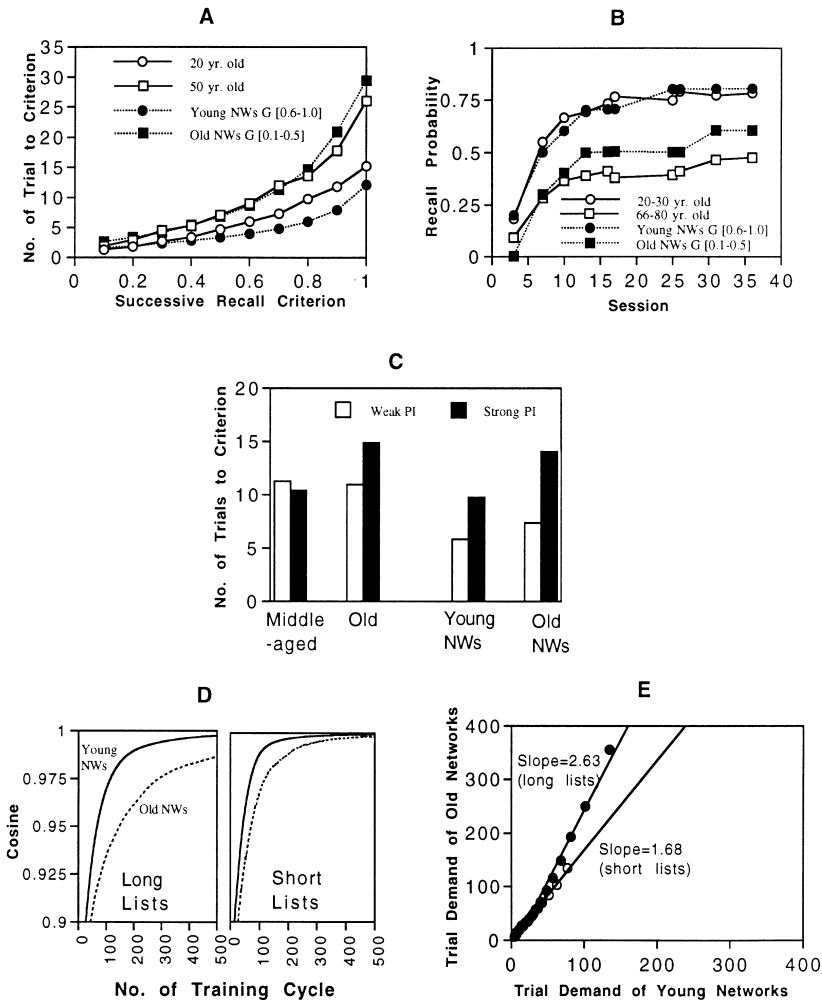
networks (i.e., with reduced mean *G*) require more learning trials than the young networks to reach increasingly strict recall criteria in paired-associate learning.

If old and young adults differ only in their learning rates, then one could expect that given enough practice old adults would eventually perform at the level of young adults. Alas, ample data of aging and practice effects on skill acquisition show that negative age differences often persist at asymptotes [4,27]. Fig. 2B shows that negative age differences in learning paired-associates at asymptotes can also be accounted for by reducing mean *G*.

4.2. Aging and interference susceptibility

Another prominent cognitive aging deficit is older adults' increasing susceptibility to interference [16,18]. For instance, it is usually found that older adults are more susceptible to proactive interference (PI, the negative effect of previous learning on subsequent learning) than young people. Fig. 2C shows that while the number of trials required to learn the second list to criterion does not differ for middle-aged participants under conditions of weak and strong PI, old adults need many more trials to learn the second list when PI is strong. This age by degree of PI interaction can be simulated by the networks with high and low *G*s. In line with the empirical data, the "old" networks show a disproportional increase in the number of trials required for learning the second list in the strong PI condition. This effect of mean *G* reduction on increasing interference susceptibility has also been demonstrated in a multidimensional perceptual categorization paradigm [19].

4.3. Aging and complexity cost

One other robust empirical regularity of cognitive aging is the so-called age by complexity (or difficulty) effect, referring to the global pattern of an increase in the

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Fig. 2. (A) Negative age effect on learning paired-associates in human subjects [22] and simulations. In neural network simulations it is often that one simulation cycle does not correspond exactly to one trial of human learning. For the results reported here, exchange factor is 17 (one human learning trial = 17 network cycles). The successive recall criterion is from 10% to 100% correct for humans, and retrieved cosine from 0.90 to 0.99 for the networks. (B) Negative age difference in asymptotic performance in human subjects [4] and simulations. In this simulation, the exchange factor is 25 (one human session = 25 network cycles, in the empirical study a session involves multiple learning trials). (C) Age-related increase in susceptibility to PI in dual-list paired-associate learning in human subjects [18] and simulations. Exchange factor used here was the same as Fig. 3A. The number of trials given for the first-list learning manipulated the extent of PI. For the weak and strong PI conditions, the networks were trained on the first list for 50 and 200 trials, respectively. The criterion of the second-list learning was an average of cosine = 0.95 across different study lists. (D) Differences between the young and old learning curves as a function of task difficulty. Learning functions of young and old networks differed more in the more difficult condition (implemented here with longer list length; list length for the short and long lists were 3 and 8, respectively). (E) Young–old functions based on the learning curves presented in panel D. The slope of the young–old function is steeper in the condition with long lists, indicating a larger difference between the two groups of networks when learning the more difficult task.
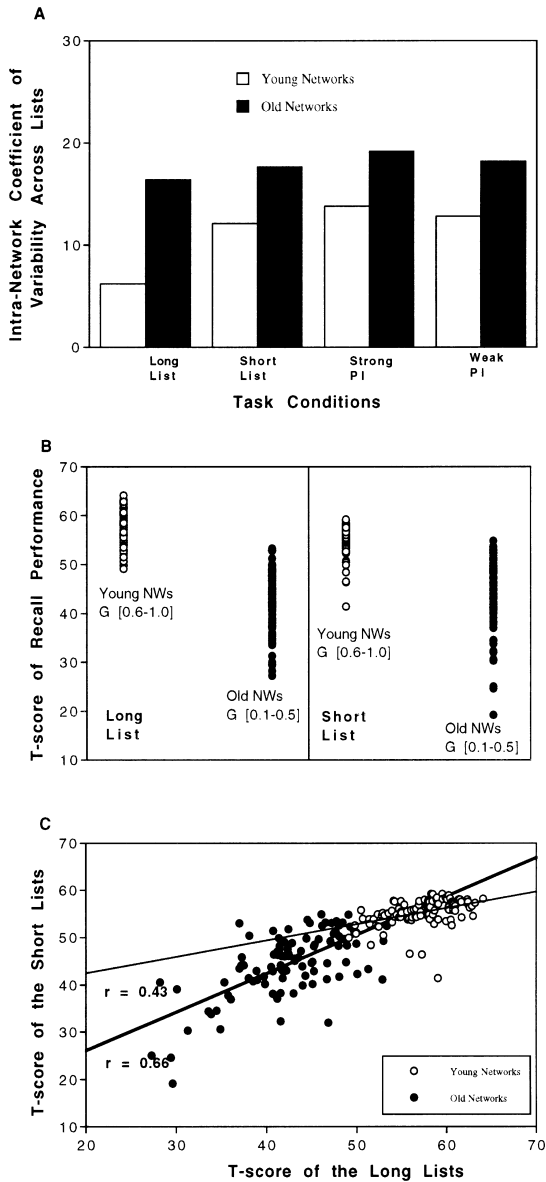
Fig. 3. (A) The effect of mean G reduction on intra-network variability in performance level across different study lists in four conditions. (B) The effect of mean G reduction on inter-network variability. Across conditions of list length, reducing mean G not only reduces mean-level performance, but also increases *inter*-network variability. (In the condition with short lists, the SD of "young" and "old" networks are 2.85 and 5.62, respectively. In the condition with long lists, the SD of "young" and "old" networks are 3.51 and 6.92, respectively). (C) The effect of mean G reduction on covariation of performances. Reducing mean G increases the correlation between performances with short and long lists. The correlation is stronger for the "old" than for the "young" networks ($r = 0.66$ vs. $r = 0.43$, the difference between these two correlations is statistically significant, $z = 2.4$).

magnitude of negative age differences with increasing processing demands or task difficulty [17]. To test whether mean G reduction can account for this broad class of phenomena, we simulated this effect within the paired-associate learning paradigm, using list length to manipulate task difficulty.

In line with the age by complexity effect, the difference between "young" and "old" networks is larger in the condition of long lists (Fig. 2D). A more detailed approach to examine the effect is to regress the number of trials required by the "old" networks to reach a range of successive performance criteria on the number of trials required by the "young" networks to reach the same criteria for both conditions. If indeed there is an age by complexity interaction, then the slope of the regression line for the more difficult condition should be steeper than that of the easier condition [17]. The corresponding young network–old network functions shown in Fig. 2E confirm the expectation.

4.4. Aging, *performance variability and covariation of performance*

In addition to negative age changes in levels, behavioral data also point to age-related increments in both *intra*-individual [11] and *inter*-individual [23] variability. To examine the effect of mean G reduction on *intra*-network variability, the coefficient of variability was computed for each network based on its levels of performance across different study lists in the four conditions of paired-associates learning described earlier. Averages based on the coefficients of variability of each network were then computed for the "young" and "old" networks. Across all conditions, the average intra-network variability was larger in old networks (Fig. 3A).

With respect to examining the effect of mean G reduction on *inter*-network variability, two groups of 100 networks were given the same amount of training to learn long and short lists of paired-associates. Performances of all networks were standardized across groups to a T-score metric (mean = 50, SD = 10). Results presented in Fig. 3B show that across conditions of list length mean G reduction not only lowers the mean levels of performance, but also raises *inter*-network variability.

Many psychometric studies conducted since the 1920s found that the correlation coefficients among subscales of intelligence tests are usually larger in older samples [3,24]. To examine the effect of mean G reduction on the patterns of covariation, T-scores of 100 networks after learning the long lists were correlated for each of the two network groups with their own performances after learning the short lists. Fig. 3C shows that the correlation between the performances with long and short lists is stronger in "old" than in "young" networks (see [19] for additional related demonstrations with respect to other experimental paradigms).

5. Conclusions

We have demonstrated with a series of simulations that a single-parameter manipulation (i.e., reducing the mean G of the activation function) accounts for a wide range of cognitive aging phenomena. Regarding unifying data and theories across levels, the

simulations explicate a theoretical path from attenuated catecholaminergic function to reduced neural responsivity and increased neural noise to less distinct cortical representation in the aging brain to behavioral manifestations of age-related cognitive deficits. The interaction between the magnitude of G and its random variability on intra-network variability illustrates a potential neurochemical mechanism for age-related increase of neural noise involving the joint effect of age-related decline in neuromodulation and probabilistic transmitter release. Further theoretical efforts should aim at specifying formalisms that address the relationships between the distinctiveness of cortical representations and fundamental aspects of cognition, such as working memory capacity and processing speed. Regarding the integration of behavioral data across measurement dimensions, the demonstration that mean G reduction simultaneously accounts for age-related differences in mean, variability, and covariation instantiates Spearman's [32] classical notion of general brain energy being the joint cause for individual differences in these three aspects of behavior, and the differentiation–dedifferentiation hypothesis of ability structure across the lifespan emerging from this notion [3,24].

In this study, we focus on catecholaminergic modulation because of converging evidence with respect to its functional properties and age-related changes. However, the computational formalisms demonstrated here should generalize to other transmitter substances as long as they exhibit similar functional properties and age gradients.

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