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Cross-Level Unification: A Computational Exploration of the Link Between Deterioration of Neurotransmitter Systems and Dedifferentiation of Cognitive Abilities in Old Age

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Introduction

In the last few decades, much research progress has been made in neuroscience and in many subfields of psychology such as cognition and development. Alas, while empirical data and theories have been accumulating within each of these disciplines rapidly, overarching theoretical orientations which aim at integrating subsets of these fields are scarce, the importance of cross-domain or cross-level unification as revealed in the history of science notwithstanding. Take psychology as a particular example of disunity either in terms of methodologies, domains of research, or levels of analysis: Not only is there a lack of integration, to the contrary, strong bifurcations exist between the experimental and psychometric traditions (Cronbach, 1957, 1975), between the studies of child development and adult development/aging (Baltes, Staudinger, & Lindenberger, in press), and between the behavioral and biological studies of cognition (Churchland & Sejnowski, 1988). Segregation, either within a given discipline or between disciplines, is not optimal. According to Leibniz (1690/1951, p. 73), who thought that scientific inquiry can be viewed as "an ocean that is continuous everywhere without a break or division" (cf. Gigerenzer, 1991), interdisciplinary exchange and integration are not only desirable but also necessary for science to progress.

Unification via Interdisciplinary Coevolution: The Example of Cognitive Neuroscience

Although it has not been stressed until recently, a positive change towards a zeitgeist of interdisciplinary integration among subfields of psychology and

neuroscience is slowly emerging. Many philosophers of science, neuroscientists and psychologists now assert that unification of theories and findings at different levels and in different domains is a *process* within which the *coevolution* of theories in related fields can take place by ways of cross-level hypothesis generation and testing (e. g., Baltes et al., in press; Bechtel, 1988; Churchland, 1988; Churchland & Sejnowski, 1988; Llinas & Churchland, 1996; Newell, 1990; Plude, Enns, & Brodeur, 1994; Posner, 1992; Royce, 1987; Schacter, 1992; Schneider, 1993; Shepard, 1987; Staats, 1991).

In the last decade of the 20th century, an example of a fruitful convergence of research from formerly isolated fields is the emergence of cognitive neuroscience. With the goal of uniting computational cognitive science, experimental cognitive psychology and neuroscience, researchers endorsing the cognitive neuroscience orientation have been working towards bridging the gap between the descriptions of information processing and the specifications of brain functioning. Indeed, the various studies reported in this volume exemplify some attempts at more integrated views of memory functioning by drawing together data and theories from different levels.

Given that the agenda of cognitive neuroscience is to unify empirical regularities *and* theories of cognition at the behavioral, information processing, and biological levels, the research strategy has been to simultaneously collect, via neural imaging techniques, experimental data concerning both behavioral manifestations and neuronal properties of cognitive systems. In addition to such experimental endeavors, theoretical efforts have also been devoted to the construction of two types of neural models serving different but complementary purposes: (a) specific models which aim at capturing the dynamics and anatomy of particular neural circuitry (e. g., Houk, Davis, & Beiser, 1995; Berns & Sejnowski, 1998), and (b) general models which try to capture global principles of neural information processing, such as signal coding, transmission, and storage, that might overall apply in many different cortical networks (cf. Churchland & Sejnowski, 1988; e. g., McClelland, McNaughton, & O'Reilly, 1995; Servan-Schreiber, Printz, & Cohen, 1990).

Research Goal and Organization

The computational investigations described in this chapter belong to the class of general models. Specifically, we explore, via connectionist simulations, a potential theoretical path from the deterioration of neural information processing to the dedifferentiation of cognitive abilities that is empirically observed in old people. To investigate this theoretical link, we looked at two sets of mechanisms. The first set concerns the effect of neurotransmitters, in particularly catecholamines, on the signal-to-noise ratio of neural transmission

and the subsequent effect on the level of random variability within the central nervous system (CNS). The second set has to do with the relations between these biological processes and age-related increase in intraindividual and interindividual variability and the concomitant dedifferentiation of ability structure in old age.

The organization of this chapter is as follows: We first describe experimental results regarding age-related dedifferentiation in old people's ability profiles and age-related increase in interindividual and intraindividual variability. A few general conceptual accounts for these empirical findings will be presented, along with a short description of an attempt to formally integrate these two sets of findings and explanations at a purely descriptive level. We then present empirical findings on aging-induced deterioration of neurotransmitter systems (Morrison & Hof, 1997) and the increase in CNS variability at the biological level. In trying to bridge the gap between the empirical phenomena observed at these two levels, we propose a computational approach which varies the responsivity of the processing units and the internal variability of connectionist networks by manipulating the gain parameter of the sigmoid activation function. Specifically, the aging-induced depletion of catecholamines (Gabrieli, 1995) is simulated by reducing the value of the gain parameter. After describing the foundations upon which our computational model is based, we then report two sets of simulations, each involving three groups of networks that *differ only* in the means of the uniform distributions from which values of gain parameters were sampled. Then, we examine the effect of this gain parameter manipulation on the intercorrelations between the networks' performances in two task domains (i. e., episodic memory and categorization learning). At the end, we discuss limitations of the present formalization and its implications for the study of lifespan cognitive development.

Dedifferentiation of Ability Structure, Variability, and Some Conceptual Accounts

In this section, we review two separate sets of empirical findings at the behavioral level that are taken from cognitive aging research and psychometric studies of intelligence. To our knowledge, these two sets of findings have been rarely reviewed together, implying that they are formerly thought to be independent. The first set of findings concerns the tendency towards dedifferentiation of intellectual abilities from early adulthood to old age. The second set of results pertains to age-related increase in both interindividual and intraindividual variability in old age. General explanations previously proposed to account for these findings are also discussed.

Dedifferentiation of Ability Structure

At the behavioral level, one of the most replicable and important psychometric findings about mental abilities is the positive manifold (i. e., patterns of positive correlations) between tests of different cognitive abilities. As early as the turn of this century, Spearman (1904) identified a factor of general intelligence *(g)* to indicate the degree of the positive manifold. Patterns of positive manifold, as represented in factor analytical models, are usually taken as descriptive indicators for the organizations of the mental abilities measured by intelligence tests. Given that a central issue in developmental and especially lifespan research concerns changes in the organization of behaviors, the notion that development may modulate the degree of differentiation or lack of differentiation (i. e., dedifferentiation) among mental abilities has been of great interest to researchers of intelligence and its development (e. g., Burt, 1954; Garrett, 1946; Reinert, 1970; Spearman, 1927). To date, results from cross-sectional psychometric studies of intelligence show a general trend from a lack of differentiation to differentiation to dedifferentiation across the lifespan, as the ontogeny of cognition proceeds, respectively, from childhood to adulthood and finally into old age. Empirical findings regarding the differentiation of cognitive abilities in child development will be discussed later. Here we first focus on data pertaining to cognitive aging. Empirical evidence for age-related dedifferentiation of cognitive abilities has been found both with respect to intercorrelations among tasks that are within the same domain of functioning (i. e., intrasystemic relations) and across different domains of functioning (i. e., intersystemic relations).

Dedifferentiation of Intrasystemic Relations in Psychometric Studies

As people age, the statistical structural patterns involving different types of mental abilities become less differentiable. For instance, using Wechsler's normative data, Balinsky (1941) found that differentiation increased from early adolescence to adulthood and then reversed in later adulthood. Similarly, Lienert and Crott (1964) tested adolescents (age 10–12 years), young adults (age 18–20 years), and older adults (age 45–60 years) on 14 ability tests, and found that the percentage of variance in the first centroid factor was 45, 41 and 47. Baltes, Cornelius, Spiro, Nesselroade and Willis (1980) found that the factor structure of fluid and crystallized intelligence was less differentiable in old people (60 to 89 years old). Likewise, Hayslip and Sterns (1979) found that intercorrelations among tests of fluid and crystallized intelligence were higher for older than for younger adults. Cunningham (1980) compared the ability structures of several adult age groups, and found that although similar factor

loading patterns could be obtained for adults of increasing age, there was an age-related increase in the magnitude of factor covariance. Besides the results from cross-sectional studies, in a longitudinal study, McHugh and Owens (1954) found that the first unrotated principal component accounted for 53% of the variance in the Army Alpha Test when the participants were at the age of 19, and increased to 63.4% when the participants reached the age of 50. In addition to these earlier findings, two very recent studies provide new support for age-related dedifferentiation in intrasystemic relationships. For instance, Baltes and Lindenberger (1997) found that the strengths of intercorrelations between five intellectual abilities were stronger (median $r = 0.71$ vs. median $r = 0.37$) for old people (age 70–103 years) than for young people (age 25–69 years). Babcock, Laguna, and Roesch (1997) examined the factor structure of processing speed (involving a total of nine speed measures) in young (age 18–24 years) and old (age 55–80 years) people. Their results showed that although the number of factors and factor loadings were invariant across the two age groups, the interfactor correlations, the variance-covariance matrices, and the unique variances differed between the groups, all indicating a greater degree of dedifferentiation in the old group.

Dedifferentiation of Intersystemic Relations in Cognitive Aging Research

Besides the age-related strengthening of intrasystemic relationships that is evident in psychometric studies, recent cognitive aging research has also identified an intersystemic relationship between cognitive and simple sensory and sensorimotor functioning. For instance, Granick, Kleban, and Weiss (1976) reported high correlations between auditory threshold at various frequencies and scores of the verbal ($r = 0.44$) and digit symbol ($r = 0.36$) subtests of the Wechsler's Intelligence Scale. Baltes and Lindenberger (1997) and Lindenberger and Baltes (1994) found that the relationship between the performance measures of sensory (i. e., auditory and visual acuity) and sensorimotor (i. e., balance and gait) functioning and those of cognitive functioning (including tests of processing speed, memory, reasoning, practical knowledge and verbal fluency) was of such magnitude that, for the age range from 70 to 100 years, practically all age differences (91%) in cognitive functioning, which corresponds to about 40% of the total interindividual differences in cognitive functioning, were associated with and therefore can be predicted by relatively simple sensory and sensorimotor measures. Likewise, Salthouse, Hancock, Meinz, and Hambrick (1996) showed that, for the age range from 18 to 92 years, visual acuity shared a very large proportion of age-related interindividual differences in measures of working memory, associative learning, and concept identification.

Indications of Dedifferentiation in Neural Information Processing

In addition to the findings of age-related dedifferentiation of old people's ability structure at the behavioral level, recent results from studies of brain imaging also gave initial indications of a parallel trend at the biological level. For instance, Grady and colleagues (Grady et al., 1992, 1994) examined aging-induced changes in object and spatial visual processing at the level of regional cerebral blood flow (rCBF). Their results demonstrated that during object matching, old people showed more activation than young people in the right prefrontal cortex; and during location matching, old people showed more rCBF activation than young people in several areas of prefrontal cortex (i. e., in bilateral inferior parietal cortex and left medial parietal cortex). Based on these results, Grady et al. (1994) suggested that during visual processing the neural circuitry in the occipital visual area is more efficiently used in young people; whereas in old people there is more reliance on other additional cortical networks (in particularly for spatial vision), indicating a stronger degree of interdependence among different processes at the cortical level. Animal models of aging also provide evidence in support of a tendency of age-related functional dedifferentiation at the cortical level. For instance, it was found that the receptive fields of the hind-paw representations in sensorimotor cortex and the cortical areas excited by tactile point-stimulation to be large and highly overlapping in old rates, but relatively small and focused in young rates (Spengler, Godde, & Dinse, 1995).

In summary, the phenomenon of increasing interdependence among different functions and processes in old age is relatively ubiquitous. At the behavioral level, empirical evidence has been found both with respect to intrasystemic and intersystemic relationships; and with different types of performance measures, ranging from standardized intelligence tests and elementary experimental cognitive tasks of memory and processing speed to sensory acuity. At the biological level, empirical supports came both from neural imaging studies on the dynamics of brain metabolism in humans and from animal models of brain aging.

Age-Related Increase in Variability

We now turn to describe the phenomena of age-related increase in both interindividual and intraindividual variability. Although the idea of relating cognitive aging deficits to aging-induced increase in neural noise was first introduced in about four decades ago (e. g., Crossman & Szafran, 1956; Welford, 1965), most studies of cognitive aging, however, focus only on measures of central tendency. Issues on age-related increase in dispersion (i. e., intraindi-

vidual variability) or diversity (i. e., interindividual variability) and the relationship between these two types of variability, on the other hand, have not been emphasized in gerontological research (cf. Nesselroade, 1991a,b). Nevertheless, meta-analyses based on longitudinal and cross-sectional studies which reported measures of variability indicated an age-related increase in variability.

Interindividual Variability

For instance, with respect to cognitive variables, (i. e., memory and other measures of intelligence) 79% of the studies (6 longitudinal and 48 cross-sectional) reviewed by Nelson and Dannefer (1992) reported an increase of variability with age. Similarly, results from Morse's (1993) meta-analysis (only cross-sectional studies were included) showed that interindividual variability in measures of response time (RT), memory, and fluid intelligence increased with age. Hale, Myerson, Smith, and Poon (1988) examined the question exclusively with respect to RT, and found age-related increase in interindividual variability. In addition to these meta-analytical studies, other experimental studies also showed that interindividual variability in episodic memory, measures of fluid intelligence (Christensen, Mackinnon, Jorm, Henderson, Scott, & Korten, 1994), and digit memory span (Rabbitt, 1993) increased with age.

Intraindividual Variability

Besides age-related increase in interindividual variability, there is also evidence for age-related increase in *intra*individual variability. For instance, intertrial variability in RT was also found to increase with age (e. g., Fozard, Thomas, & Waugh, 1976; Salthouse, 1993). In addition to results regarding intraindividual variability of response latency, Li, Aggen, Nesselroade, and Baltes (1998) measured memory and sensorimotor performances in a small sample of community-dwelling elderly (age 64–86 years) in 13 biweekly measurement occasions that spanned across six months. Their results showed a trend in the direction of a positive correlations between age and the magnitude of intraindividual variability in memory ($r = 0.46$, $p < 0.05$, n = 19) and sensorimotor ($r = 0.20$, $p = 0.4$, n = 19) performance.

Indications of Increased CNS Variability

In addition to the aforementioned empirical evidence of age-related increase in intraindividual and interindividual variability in RT and other performance measures at the behavioral level, neurobiological studies have also shown

indications of a trend of increased CNS variability. For instance, Kraiuhin, Gordon, Stanfield, and Meares (1986) examined the relationship between age and auditory P300 latency (a component of event related potentials) via a tone discrimination task in normal adults (age 15 to 89 years). In addition to a significant relationship between age and P300 latency, their results also showed significantly more latency variability in the subsample of older adults (i. e., adults over 45 years). Kugler, Taghavy, and Platt's (1993) review of studies involving the P300 potential analysis of cognitive human brain aging also indicated a trend of age-related increase in P300 variability.

In brief, although the issue of age changes in interindividual and intraindividual variability has been somewhat ignored in gerontological research, the available empirical evidence seems to suggest a trend of age-related increase in both types of variability. Data supporting this tendency have been found both at the behavioral and biological levels.

Conceptual Explanations of Dedifferentiation and Increased Variability

A few explanations for the two sets of empirical phenomena reviewed above have been suggested at the conceptual level. In this section, we present some of these general accounts. Controversies associated with some of these explanations are also discussed.

Common-Cause Hypothesis and Dedifferentiation

Regarding the phenomenon of age-related dedifferentiation of ability structure, one explanation, known as the common-cause hypothesis (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994; Lindenberger, Marsiske, & Baltes, 1998), proposes that normal aging is associated with a general loss of cognitive capacity and plasticity that is in turn caused by aging-induced deterioration of general neurobiological mechanisms which compromise the integrity of the brain across a wide range of areas and functional circuitry. Mechanisms and processes of brain aging are then postulated to constrict the functional cerebral space (Kinsbourne & Hicks, 1978), which could manifest at the behavioral level as the dedifferentiation of ability structure.

Some Controversies Regarding the Explanations of Increased Behavioral-Level Variability

With respect to the phenomenon of increased variability in old age, some researches have proposed that age-related increase in *inter*individual differences might be associated with individual differences in the rates of neuro-

bioogical deterioration that are associated with aging (Birren, Woods & Williams, 1980; Rabbitt, 1981; Welford, 1980). However, within the cognitive aging literature, this view has been specifically questioned with respect to age-related increase in RT variability. It has been demonstrated that the correlation between age and the standard deviation of RT is greatly attenuated or, in some cases, eliminated when the effect of mean RT is statistically controlled* (e. g., Hale et al., 1988; Salthouse, 1993). Therefore, it was argued that a general mechanism of age-related slowing, in and of itself, is sufficient to cause the increase in *inter*individual variability, and that increased variability in RT should be viewed as a consequence, rather than a cause, of age-related slowing. Nonetheless, age-related slowing itself as a phenomenon at the behavioral level still needs to be explained. In addition, interpretations of causality that are based on statistical control rather than direct experimental manipulation should be taken with constraints. Specifically, with respect to the statistical explanatory advantage of mean RT over the variability of RT in predicting age, one should at least note that in addition to the causal relationship interpreted by Hale et al. (1988) and Salthouse (1993), a difference in measurement reliability of these two types of measures, favoring the measure of central tendency, can be one other important factor contributing to the explanatory advantage of mean RT. Besides, central-tendency measures of RT also do not exhibit explanatory advantage when predicting other variables. For instance, some psychometric studies of intelligence have shown that trial-by-trial intraindividual variability in RT consistently correlates more highly with the factor of general intelligence than mean RT, despite the fact that the test-retest reliability of the measures of variability is usually lower than that of mean RT or median RT (e. g., Jensen, 1992; Smith & Stanley, 1987). People who show greater intraindividual variability in their response latencies tend to score lower on IQ tests. In addition, it was demonstrated that although the standard deviation of RT and median RT are highly correlated, they still reflect independent sources of variance that are specific to each of the two variables. Using Spearman's (1904, 1927) formula to compute the true-score correlation between the standard deviation and the median of the RT distribution, Jensen (1992) found that the overall specificity (i. e., variance specific to each of the two variables) is still about 34.4% of the total true-score variance.

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* The generalizability of this finding to performance measures other than response times is not clear. For instance, Li et al. (1998) found that the positive relationship between age and the magnitude of intraindividual variability in memory performance was not affected after controlling for mean-level performance ($r = 0.460$ before controlling for mean performance, and $r = 0.456$ after controlling for mean performance). However, the relationship between age and the magnitude of intraindividual variability in sensorimotor functioning was eliminated after controlling for mean-level performance.

CNS Variability as an Explanation for Intraindividual Response Variability

Psychologists interested in understanding biological correlates of intelligence have proposed that intraindividual variability in RT at the behavioral level could be related to CNS variability, which in turn is one of the biological bases of intelligence. For instance, both Eysenck (1982) and Hendrickson (1982) hypothesized that intraindividual response variability in RT could be caused by random errors, or what might be called "neural noise" in the transmission of neural signal in the CNS. This view parallels the neural-noise hypothesis in the gerontological literature (e. g., Crossman & Szafran, 1956; Welford, 1965, 1981, 1984). Indeed, as Hale et al. (1988) have argued, the finding that RT variability can be predicted, independent of age, from mean RT does lend support to the contention that there is no need to postulate an extra hypothesis of differential rates of brain aging in order to account for age-related increase in *inter*individual variability. However, such a result does not exclude the possibility that *intra*individual response variability could be an indicator of some kinds of base conditions (or more metaphorically put as "hums" of a living system by Nesselroade, 1991a) produced by organic processes taking place within the organism, for example, processes such as metabolic activities within the nervous systems (Fiske & Rice, 1955, pp. 219–220). Specifically, the explanatory advantage of measures of central tendency over measures of variability at the behavioral level does not preclude the possibility that at the level of the CNS, biochemical mechanisms which increase the level of random variability either in neural coding or neural transmission might be one of the causes for age-related slowing and other aspects of cognitive aging, such as the dedifferentiation of ability structure.

Linking Dedifferentiation to Increased Variability within Computational Frameworks

Up to this point, we have reviewed two sets of empirical findings, namely, age-related dedifferentiation of ability structure and age-related increase in variability at both the behavioral and biological levels. We have also presented some conceptual explanations that have been proposed to account for these results. As revealed in the preceding review, there is an apparent lack of integration both at the level of data and at the level of theory. Just as the empirical phenomena themselves have been investigated independently, explanations of these data have also been advanced separately. In addition, the theoretical notions been offered so far have remained at the conceptual level. Therefore, the main purpose of our study is to explore a joint platform for the explanation

of age changes in level, variability, and covariance within formal computational frameworks. The central issues is how aging-induced changes in the fidelity of neural transmission may bring about, at the behavioral level, not only decrements in performance level (as seen in longer reaction times and less accurate performance) and increments in variability, but also increments in the degree of covariation between different dimensions and domains of cognitive performance. In other words, is it possible, or even necessary, that generalized decrements in the efficacy of neural transmission simultaneously affect all three aspects of behavior? To our knowledge, no prior theoretical work has been done to formally address this question. However, a few general ideas hinting at potential relationships among subsets of these phenomena have been suggested in some researchers' earlier writings.

For instance, Cerella (1990) suggested a possible relationship between neural connectivity and mean response latency. Specifically, Cerella proposed that aging disrupts the connectivity between neurons, and that the loss in connectivity extends the length of the pathway through which a signal travels, because a signal must step around broken links in its path. Longer pathways lead then to longer response latencies.

In trying to link Reed and Jensen's (1991) finding of a positive correlation ($r = 0.26, p < 0.002, n = 200$) between IQ and brain nerve conduction velocity (NCV) measured in the visual tract with Eysenck's (1982) and Hendrickson's (1982) views on the biological basis of intraindividual variability in RT, Jensen (1992) speculated that:

> "... it is a reasonable hypothesis that the correlation between nerve conduction velocity (NCV) in the visual tract and IQ is the indirect result of similarity of NCV throughout different regions of the brain, including the higher association centers involved in complex reasoning. However, the fact that there are three synapses in the visual tract, at each of which there could be some probability of a momentary 'error' in transmission, means that the Reed-Jensen finding could also possibly support Eysenck's theory that the average latency of the neural response registered at the visual cortex results, not from NCV per se, but from the accumulation of delays due to random errors in transmission, the errors presumably occurring at the synapses." (p. 871)

The "error" in the above quotation was taken by these researchers to represent the lack of fidelity of neural information processing (i. e., the probability that a given message encoded in a series of pulse trains will arrive at its destination in the identical form in which it was encoded, Eysenck, 1982, p. 9). From the quote, it is clear that Jensen (1992) along with Eysenck (1982) and

Hendrickson (1982) suggested a relationship between the fidelity of neural transmission and response latency. However, the issues of how errors in neural transmission might affect variability observed both at the biological and behavioral level and what might be the relationship between variability level and intercorrelations among different cognitive functioning were not clearly specified. Similarly, although the neural-noise hypothesis of cognitive aging (Crossman & Szafran, 1956; Welford, 1965) suggests that age-related behavioral slowing and other deficits are likely to be associated with the increased noise level in neural transmission, the potential relationships between variabilities at the biological and behavioral levels and the patterns of covariations are not specified or discussed.

Following these lines of reasoning, we further speculate that less accurate information transmissions would lead to a higher level of random variability in the total information content within the system. Furthermore, increased random variability in the CNS might in turn play a role in age-related changes in variability at the behavioral level and in patterns of intercorrelations between different cognitive processes. We have formally instantiated these two sets of conceptual notions at the descriptive and implementation levels. Before presenting these formalisms, we discuss a set of recent empirical findings which, in part, inspired our theorizing about the relationships between CNS variability, behavioral variability, and dedifferentiation.

Intraindividual Variability and Intersystemic Relationship: Initial Empirical Indications

In terms of initial empirical findings that are at least related, if not directly parallel, to the above theoretical speculations, Li et al. (1998) recently investigated the link between memory and sensorimotor functioning within a sample of old adults via intraindividual response variability at the behavioral level. They found suggestive trends of positive correlations between the strength of a given individual's intersystemic link between memory and sensorimotor functioning and the magnitude of his or her own intraindividual variability in memory ($r = 0.29$; $p = 0.22$, $n = 19$) and sensorimotor ($r = 0.22$, $p = 0.37$, $n = 19$) performance. Li et al. (1998) argued that such a relation between the magnitude of intraindividual variability in different domains of functioning and the strength of the intersystemic link between these functions should not be trivialized as a mere statistical artifact.

Indeed, the topic of range restriction is commonly discussed within the context of interindividual difference research, and it has to do with restricted selectivity in sampling. If the range of a sample is selectively restricted, the intercorrelations among a set of variables that one observes in this given

sample might *underestimate* the true correlations in the population (e. g., Lawley, 1943; Pearson, 1903). Hence, within the context of interindividual differences, a finding of a relationship between the magnitude of interindividual variability in different performance measures and the strength of the intercorrelations among these variables might not have *substantive* value and should be interpreted cautiously, because such a relation, if not merely reflects the extent of range selectivity in the sample, is at least confounded by it.

A relationship between the magnitude of *intraindividual* response variability and the strength of intersystemic link is, however, conceptually different. While sampling variability usually bears no direct relevance to many of the theoretical constructs of cognitive functioning at the individual's level, intraindividual variability and its biological basis, on the other hand, have been central to many theories of intelligence (e. g., Eysenck, 1982; Hendrickson, 1982; Jensen, 1982), cognitive development (e. g., Siegler, 1994; Siegler & Ellis, 1996; van der Maas & Molenaar, 1992) and cognitive aging (Hanno & Hoyer, 1994; Li, Lindenberger, & Frensch, 1996; Welford, 1965, 1981, 1984). Hence, it may be of some interest to researchers in these areas, if one could more formally examine the link between age-related increase in intraindividual variability, dedifferentiation of ability structure, and aging-induced degeneration in neurotransmitter systems within computational models.

A Formalization at the Descriptive Level

As an initial attempt, Li and Lindenberger (1998) carried out Monte Carlo simulations to quantitatively examine this issue at a purely descriptive level. In these simulations, it was assumed that the brain state (i. e., total neural information content within the brain at a given moment) could be represented by a random state vector (cf. Anderson, 1983). Elements of the brain state vector were sampled from a normal distribution with a given mean and standard deviation. Two mathematical functions, A and B, represented two different processes; but both functions utilized the information content in the common brain state vector. In other words, the distributional properties of the information in the brain state vector were shared by both processes, but were transformed differently, depending on the specific function types. Furthermore, it was assumed that these two processes were not perfectly reliable, hence each of them was associated with some processing noise. The processing noise of each function was assumed to be independent.

The simulation results showed that across three different pairings of the A and B function types (i. e., logistic and polynomial, linear and linear, and exponential and power) and three different levels of processing noise, the magnitude of the correlation between the outcomes of the two functions increased

as the level of variability in the random vector representing brain state increased. Admittedly, this model oversimplifies many issues. However, it does allow one to start exploring mathematical or statistical principles addressing the issue of correlations between dependent random variables (e. g., Zimmerman, 1976) as potential mathematical formalisms that could describe and support the relationship(s) between CNS variability and the intercorrelations among different cognitive functions.

If one adopts the cognitive neuroscience orientation, it is then not satisfying to only address these phenomena descriptively. At the empirical level, one question still needs to be answered is what kinds of aging-induced neurobiological changes are likely to increase random variability in the CNS, presumably, via affecting the fidelity of neural information processing? At the level of formal modeling, one question awaiting answers is what other types of formalism can "implement" an increase in within system variability in ways that capture, at least, some functional properties of the related biological mechanisms, as opposed to a formalism, such as that of Li and Lindenberger (1998), which treats variability as a primitive in the formulation and only describes the phenomena? Therefore, with the simulations reported in this chapter we attempted to extend the descriptive results from Li and Lindenberger's (1998) Monte Carlo simulations to one type of processing model within which variability does not have to be treated as a primitive that is to be manipulated directly; rather it is the derivative of other mechanisms which mimic functional aspects of neural information processing. We now turn to present some empirical findings on aging-induced degeneration in neurotransmitter systems, and describe the computational foundations for the simulations to be presented.

Aging-Induced Deterioration of Catecholaminergic System

Epinephrine, norepinephrine, and dopamine belong to a family of neurotransmitters called catecholamines. To date, some biochemical evidence has accumulated, suggesting the role of catecholamines as neuromodulators of information processing in the brain. This is to say that catecholamines themselves do not directly change the firing rate of a neuron; however, the release of catecholamines enhances the responsivity of a neuron to other incoming afferent signals. This effect has been interpreted as the modulation of the neuron's signal-to-noise ratio (e. g., Clark, Geffen, & Geffen, 1987; DeFrance, Sikes, & Chronister, 1985; Mamelak & Hobson, 1989; Servan-Schreiber et al., 1990; Spitzer, 1997; Yang & Mogenson, 1990). In the course of normal aging, the concentration of catecholamines in the striatum and basal ganglia decreases by 7% or 8% during each decade of life (e. g., see Gabrieli, 1995; Morgan & May, 1990; Rogers & Bloom, 1985, for reviews). By extension, because of

the decline in its catecholamine concentration, the aging brain might be a noisier (or with a higher level of random variability) information processing system, as suggested by the neural-noise hypothesis.

Afew potential links between catecholamines and age-related behavioral variations in rats, non-human primates, and humans, have been documented. For instance, in training young and old rats to perform escape-and-avoidance tasks, Spirduso and colleagues found that the density of dopamine receptors was associated with response speed and its variance: the higher the density, the faster and less variable the RT (MacRae, Spirduso, & Wilcox, 1988; Spirduso, Mayfield, Grant, & Schallert, 1989). Similarly, Schultz, Studer, Romo et al.'s (1989) results showed that depletions of nigrostriatal dopamine neurons in monkeys not only increased motor reaction time and movement time, it also increased RT variability. With respect to memory performance, in a delayed-response task designed to test short-term memory capacity in non-human primates, Arnsten and Goldman-Rakic (1985) showed that memory deficits of aged monkeys, who suffered from 50% dopamine depletion in their prefrontal cortex, can be alleviated by catecholaminergic agonists. Similar associations have also been found between degeneration of the dopaminergic system and working-memory deficits (Sawaguchi & Goldman-Rakic, 1991), as well as attentional impairment (Corwin, Kanter, Watson, Heilman, Valenstein, & Hashimoto, 1986; Rothman, 1996). In humans, Kischka, Kammer, Maier, and Weisbrod et al. (1996) found that the injection of L-dopa, a dopamine agonist, reduced the magnitude of semantic priming marginally and the magnitude of indirect priming significantly. This finding suggests that the increase of semantic priming effects in old age (see Laver & Burke, 1993 for review) could be related to compromised dopaminergic mechanisms.

Formalization at the Implementation Level: Modeling the Effects of Catecholamines

A Unit's Responsivity

Within the framework of connectionist modeling, Servan-Schreiber et al. (1990) demonstrated that the modulatory effects of catecholamines (i. e., the sharpening of a neuron's signal-to-noise ratio) can be simulated by the gain parameter of the logistic activation. Equation 1 defines the activation function,

$$Output\ Activation_i = \frac{1}{1+e^{-(gain \times netinput_i + bias)}} \quad (1)$$

Figure 1 shows that reducing the value of the gain parameter (simulating attenuated efficacy of the catecholaminergic system) flattens the activation pro-

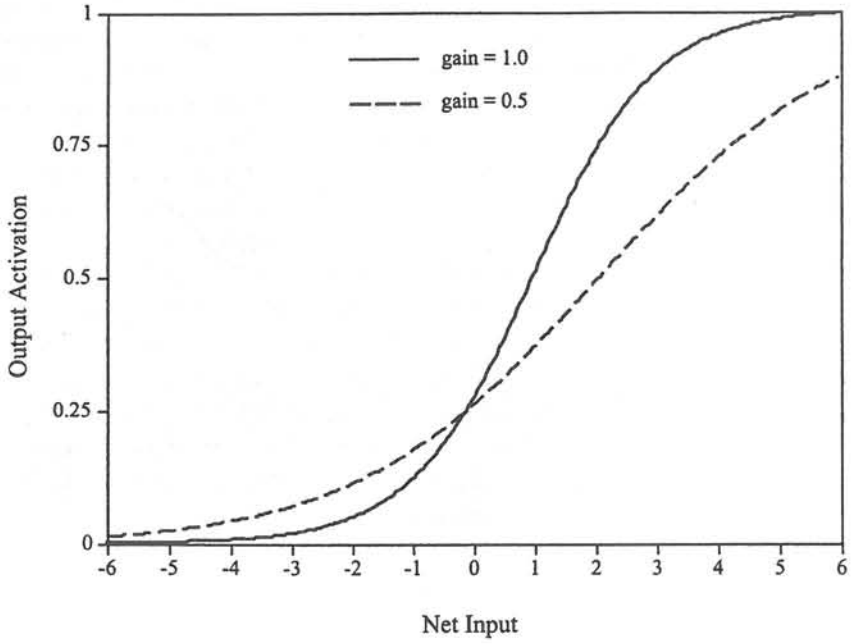

Figure 1. Gain parameter's effect on responsivity: sigmoid activation function of units in back-propagation network for two values of gain (the bias parameter of the activation function was set to –1.0).

file, hence the unit become less sensitive to changes in incoming afferent signals. Under conditions of static gain (i. e., gain parameters of all units are fixed at a given value and remain the same across all processing steps), the manipulation proposed by Servan-Schreiber et al. (1990) captures, however, only one aspect of the modulatory effects of the catecholamines, namely, the fine tuning of a neuron's responsivity.

Intra-Network Variability

Li et al. (1996) demonstrated a second property of the gain parameter when it is assumed to be stochastic (i. e., values of the gain parameters of units in a network were sampled from a uniform distribution at each processing step). When stochastic gains are used to simulate fluctuations in the concentration of transmitter substances (e. g., Kempf, Mandel, Oliverio, & Pulisi-Allegra, 1982; Manshardt & Wurtman, 1968; Reis, Weinbren, & Corvelli, 1968), the variability in a given unit's output activation in response to an input signal across different processing steps is systematically related to the mean of the

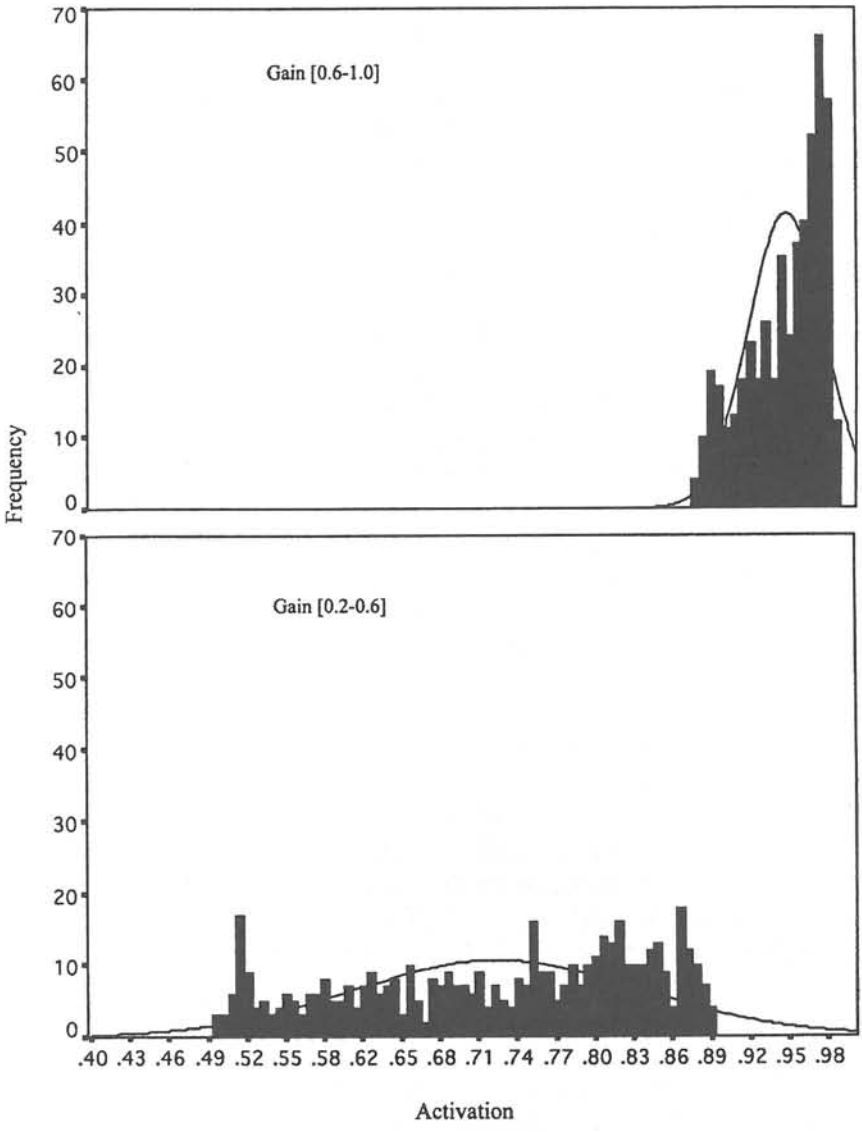

Figure 2. Gain parameter's effect on internal variability: distributions of output activations with respect to a fixed Gaussian signal with small noise across 500 trials for two ranges of gain parameter values.

gain parameters. For example, a comparison of the top and bottom panels in Figure 2 shows that the amount of variability in a unit's output activation with respect to a given Gaussian input signal that is embedded in a small amount of background noise is much greater when values of the gain parameters are sampled from the distribution with a smaller mean, although the ranges of variability in the gain parameter values are identical for both distributions (i. e., 0.4). Thus, networks with a smaller mean value for the gain parameters undergo a greater level of random intra-network variability across time, even if the same signal (or set of signals) were presented repeatedly. In this way, the stochastic gain manipulation allows us to more directly implement the relation between the reduction in catecholaminergic modulation and the increase in CNS variability in ways that are in line with earlier speculations about the relationship between the fidelity of neural transmission and the trial-by-trial variability in RT (Eysenck, 1982; Hendrickson, 1982). In fact, Li et al. (1996) already showed that a series of benchmark phenomena of cognitive aging deficits, ranging from slowed learning rate, lowered asymptotic performance, task-complexity effects and the increase in interindividual and intraindividual variability can be simulated by combining both properties of the gain parameter (i. e., the regulations of the responsivity of a unit and the level of intra-network variability).

Taken together, there is evidence suggesting that the efficacy of the catecholaminergic system is compromised during the process of aging, and that this might be the biological basis for some of the cognitive deficits observed at the behavioral level. In addition, it has been demonstrated that the modulatory effects of catecholamines (Servan-Schreiber et al., 1990) and their implications for cognitive aging (Li et al., 1996) can be reasonably modeled by the gain parameter. In the following, we use the stochastic gain manipulation to explicate the links between the deterioration in catecholaminergic system, its impact on the signal-to-noise ratio of neural information processing and CNS variability, age-related increase in interindividual variability, and the dedifferentiation of ability structure observed in old age.

Simulations

Two sets of simulations are presented. Both sets of simulations involved the standard back-propagation networks with fully interconnected layers of input, hidden and output units. Three groups of otherwise identical networks that differed only in the mean value of their gain parameters were trained and tested in each simulation. Using the stochastic gain manipulation to simulate the effect of aging-induced deterioration of catecholamine effects, the values

of the gain parameters of the "young networks" were randomly sampled from a uniform distribution within the range [0.6, 1.0], the gain parameters of the "middle networks" were sampled from the range [0.4,0.8], and lastly, the parameters of the "old networks" were sampled from the range [0.2,0.6]. One hundred networks, each started with a different random initial weight configuration, were included for each of the three network groups. It is well-known that the initial weight configuration of a network affects learning (Baldi & Chauvin, 1991; Kolen & Goel, 1991). At the beginning of learning, the initial weight configuration defines a specific starting position in the hyperspace that is jointly defined by values of all the weights and the minimum error point as defined in downhill gradient descent learning. During learning, the network must try to gradually minimize the difference (also called error) between its output activation and the target output activation. Therefore, depending on the starting location defined by the initial weight configuration, a network can have a fast or slow rate in reaching the criterion performance. Hence, interindividual differences in initial learning ability can be simulated by networks that start with different initial weight configurations. An identical set of 100 random seeds was used to define the initial weight configurations for networks in each of the three groups. This controls for the effect of initial weight configuration on learning across groups and ensures that differences observed in the performances across the three groups of networks arise only from the gain parameter manipulation.

Simulation 1: Intercorrelations among Paired-Associate Recall of Different List Length

In empirical studies using the paired-associate learning paradigm (Barnes & Underwood, 1959), participants first learn a list of word pairs, for instance, *computer* and *typewriter*, *automobile* and *airplane*, and etc. to some performance criterion. At test, the participants are expected to recall the second item (or the B item) of the pair (*typewriter* and *airplane* in the example given here), when probed with the first item (*computer* and *automobile* in the above example) of the pair (the A item). In order to simulate paired-associate learning using back-propagation networks, random asymmetric binary (0 1) input and output vectors were used to represent the A and B items. In this simulation, the architecture of the networks involved 14 input, 5 hidden, and 14 output units. The first four input and output units represented context information of a given list, and were kept the same across all items of a given list. The remaining 10 input and output units represented unique item information. On average, all item patterns consisted of an equal number of 1s and 0s. In this simulation, the gain manipulation was applied only to the output units. Three

additional network parameters, learning rate, momentum, and bias were set, respectively, at 0.1, 0.9, and –1.0, for all networks throughout the simulations.

The networks were trained to learn three paired-associate recall tasks that were defined by list length (i. e., 3, 5, and 8 items per list). Five lists were included for each of the three tasks. Lists with different length were constructed such that the shorter lists were nested within the longer lists. More specifically, five-item lists contained five items from the 8-item lists, and the 3-item lists contained 3 items from the five-item lists. This nesting of shorter lists within the longer lists was necessary to ensure that intercorrelations among the three tasks could arise from the shared information content (i. e., the specific input-output mapping) between the lists. Learning in connectionist networks is adaptive. This implies that the network's performance is jointly determined by the network architecture, parameter settings, initial weight configuration, and task requirements defined by the input-output mapping of a given task. In the simulations reported here, all of these aspects were kept constant, with the exception that task requirements differed across task conditions. However, if the tasks do not share some aspects of the input-output mapping, one cannot expect that the rank order of the effects of a set of initial weight configurations (simulating interindividual differences in initial learning ability in a given sample) on learning in one task should relate systematically to the rank order of the same set of initial weight configurations in a completely different task. Therefore, it was important to ensure that different tasks at least share some related input-output mappings.

Recall performance was determined by the similarity between target and actual output activation patterns. Similarity of the two output vectors were defined by the retrieved cosine. Specifically, given two vectors, **a** and **b**, the retrieved cosine is the ratio of the dot product between **a** and **b** to the product of the lengths of the two vectors (Goebel & Lewandowsky, 1991). Retrieved cosine is a preferred measure of vector similarity because it is invariant of the length of the vector and is scaled within the range of 0 to 1, with 0 representing maximum dissimilarity, and 1 representing maximum similarity. Performances of all networks in all three tasks were evaluated after 100 learning trials, at which point the retrieved cosine measure of the "middle networks" in the most difficult task (i. e., the 8-item condition) reached 0.965.

Results

Gain Parameter and Mean-Level Performance

The mean performance of each network group in recalling lists of different list lengths are plotted in Figure 3. Results from analysis of variance (ANOVA)

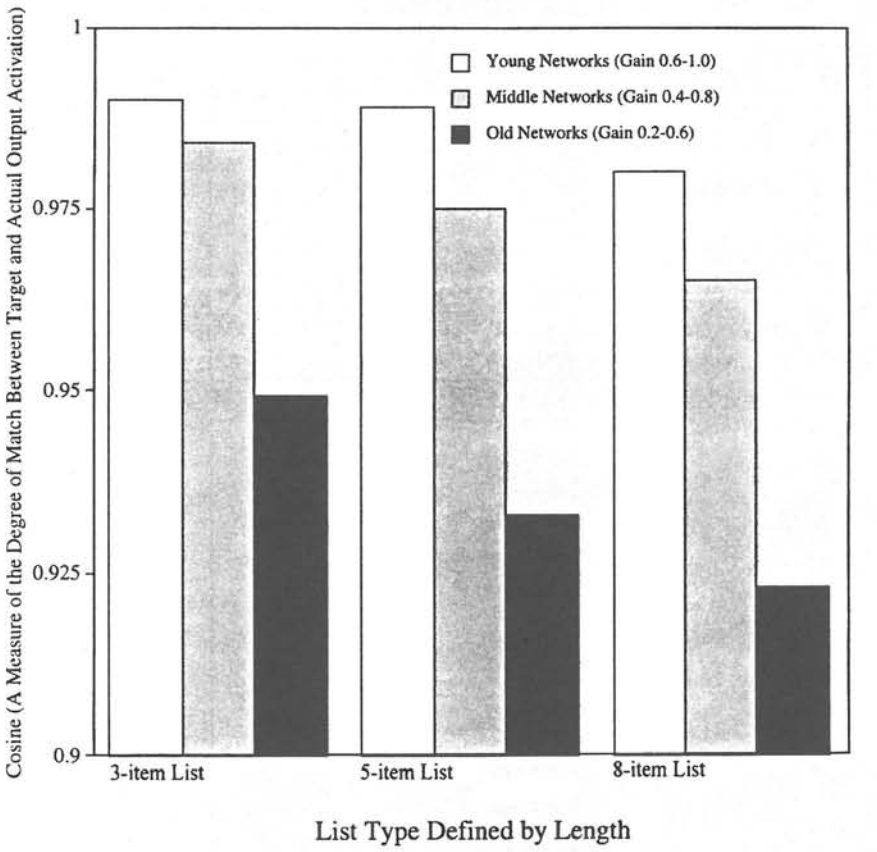

Figure 3. Neural networks' performance in the paired-associate recall task as a function of gain parameter and list length.

of a two-way split-plot factorial design involving three treatment levels and three groups showed that the main effects of list length and group, and the length by group interaction were all significant with p values less than 0.001. For all network groups, performance was best for short lists – a result that is in line with the classical list-length effects found in a wide range of memory performance, ranging from recognition, free recall, to cued recall (e. g., Strong, 1912; Gillund & Shiffrin, 1984). In addition, the "cost" of learning longer lists in comparison to the shorter lists was largest for the old networks and smallest for the young networks, as indicated by the significant interaction between group and list length. This finding is in good agreement with the age by complexity effect that is often reported in the cognitive aging literature (e. g., McDowd & Craik, 1988).

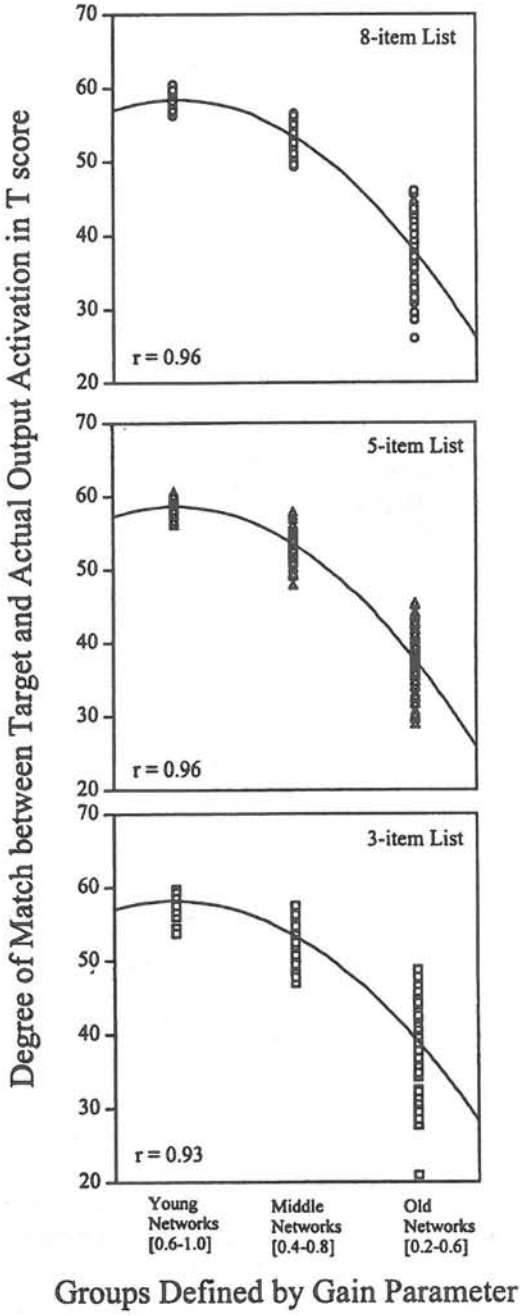

Figure 4. Inter-network variability as a function of the gain parameter in paired-associate recall of three different list lengths.

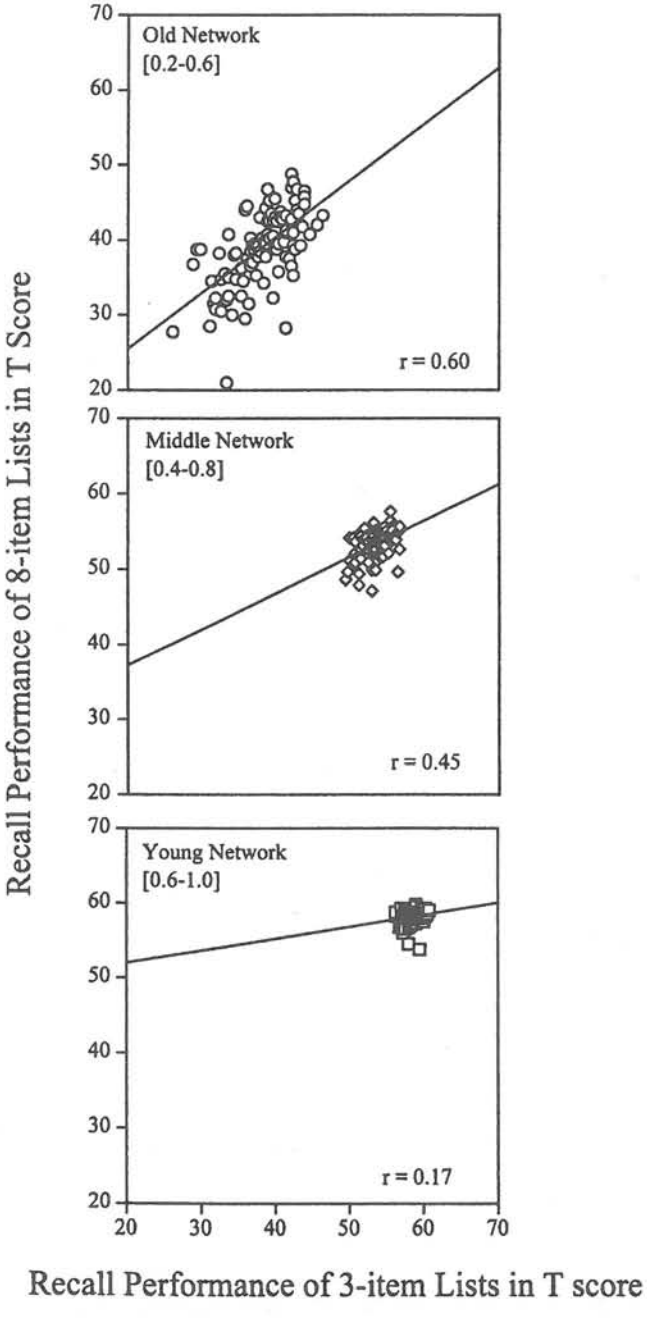

Figure 5. Correlations between performances in long and short lists as a function of the gain parameter.

Gain Parameter and Inter-Network Variability

In line with our theoretical expectations, the gain parameter also affected the magnitude of inter-network variability and the correlational structure involving the three paired-associate tasks. For each list, the retrieved cosine measure was first transformed, across the three network groups, into T scores (mean = 50, SD = 10). Within each list length, the average across five lists was computed based on the T scores. Figure 4 shows that for each of the three list lengths, inter-network variability increases as the mean value of the gain parameter decreases. A similar set of findings involving networks with a different architecture and a different range of gain parameter values was found in Li et al. (1996). Furthermore, the finding of quadratic declining trends as a function of the gain parameter is also in agreement with the empirical data of age-related declines in measures of fluid intelligence (e. g., Baltes & Lindenberger, 1997).

Gain Parameter and Patterns of Intercorrelations

Moreover, the gain parameter also affected the patterns of intercorrelations between the three recall tasks. Figure 5 shows scatter plots of the correlation between the performances of the networks in the 8-item and the 3-item conditions as a function of the gain parameter manipulation. Going from top to bottom in Figure 5, it is clear that the correlation between the performance on these two tasks was weakest in the young networks ($r_{old} = r_{mid} > r_{young}$, $z = 3.63$). Table 1 shows that as the values of the gain parameters decrease (simulating aging-induced deterioration in catecholamine systems), the correlations between the three memory tasks increase. Principal component analyses of the three correlation matrices show that the percentage of variance accounted for by the first principle component increases as the values of the gain parameters decrease, indicating a less differentiated structure in the performance of the old networks.

Table 1. Correlations between performances of lists of different length

Young Networks Gain Parameter [0.6–1.0]			Middle Networks Gain Parameter [0.4–0.8]			Old Networks Gain Parameter [0.2–0.6]		
	8-item	5-item		8-item	5-item		8-item	5-item
5-item	0.31	–	5-item	0.59	–	5-item	0.74	–
3-item	0.17	0.24	3-item	0.45	0.54	3-item	0.60	0.74
Variance accounted for by 1st PC:		49.3%	Variance accounted for by 1st PC:		68.3%	Variance accounted for by 1st PC:		79.6%

Simulation 2: Intercorrelations among Categorization Tasks of Different Discriminability

In this simulation we examined the effect of the gain parameter on the correlational structure of the networks' performances in three categorization tasks with different levels of between-category discriminability. Again, three groups of networks, with 100 networks in each group, were trained to learn 2-choice categorization tasks involving bivariate normal stimuli. A bivariate normal category is defined by normally distributed values (with known mean and variance) on two stimulus dimensions (e. g., Ashby & Gott, 1988; Ashby & Maddox, 1992). In this simulation, each network had two input, hidden, and output units. The gain manipulation was applied to all units in the network. Each of the two input units represented one of the two stimulus dimensions, and each of the two output units represented one of the two response categories. The networks were trained to categorize the stimuli into two categories, A and B, depending on whether Dimension 1 was greater or smaller than Dimension 2. Training exemplars for each of the two categories were sampled from bivariate normal distributions with means of 0.3 and 0.7 for the first and second dimensions of category A, and a reverse set of means, 0.7 and 0.3, for the two dimensions of category B. The networks were trained to learn categorization tasks with three levels of between-category discriminability. Within each condition, the networks were trained on a total of 2000 stimulus exemplars during learning. During testing, 400 testing patterns were constructed by crossing 20 values (ranging from –0.5 to 1.4 with a stepsize of 0.1) for the first input dimension and 20 identical values for the second dimension. The degree of discriminability between categories was manipulated by varying the extent of overlap between categories. This was defined by the standard deviations of the two stimulus dimensions. Figure 6 shows the extent of overlap between the probability density functions of two sets of binomial categories that are defined by less (top panel, $SD = 0.2$) or more spread (bottom panel, $SD = 0.6$) stimulus dimensions. As shown here, when the standard deviation is large (bottom panel), the overlap between categories increases and the between-category discriminability decreases. Three additional parameters, learning rate, momentum, and bias were set, respectively, at 0.1, 0.7, and –1.0, for all networks throughout the simulations

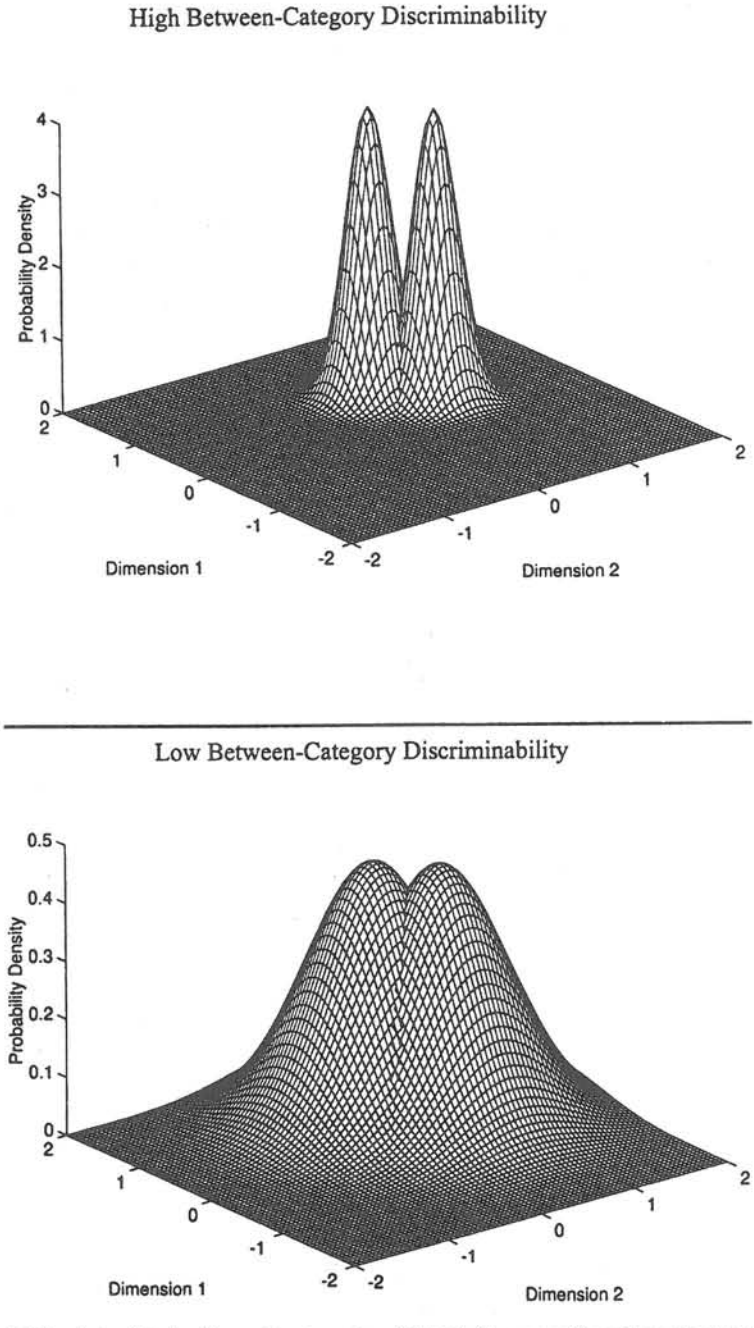

Figure 6. Bivariate distributions of categories with high (top panel) and low (bottom panel) between-category discriminability.

Results

Gain Parameter and Mean-Level Performance

Performance of the three groups of networks as a function of between-category discriminability are plotted in Figure 7. Results from ANOVA using a two-way split-plot factorial design involving three treatment levels and three groups showed that the main effects of discriminability, group, and the interaction between discriminability and group, were all significant, with p values less than 0.001. For all network groups, performance decreased as between-category discriminability decreased. With respect to the main effect of group, young and middle networks performed comparably; however the old networks performed much more poorly in all conditions and was disproportionately worse in the low discriminability condition.

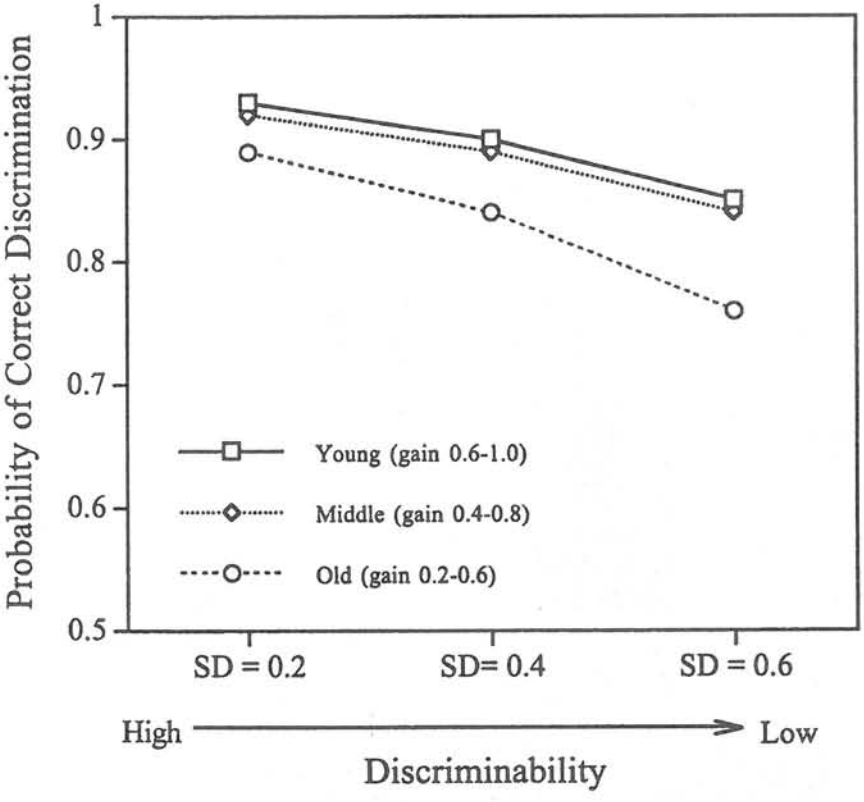

Figure 7. Neural networks' performance in the two-choice categorization task as a function of gain parameter and discriminability (chance performance is 50%).

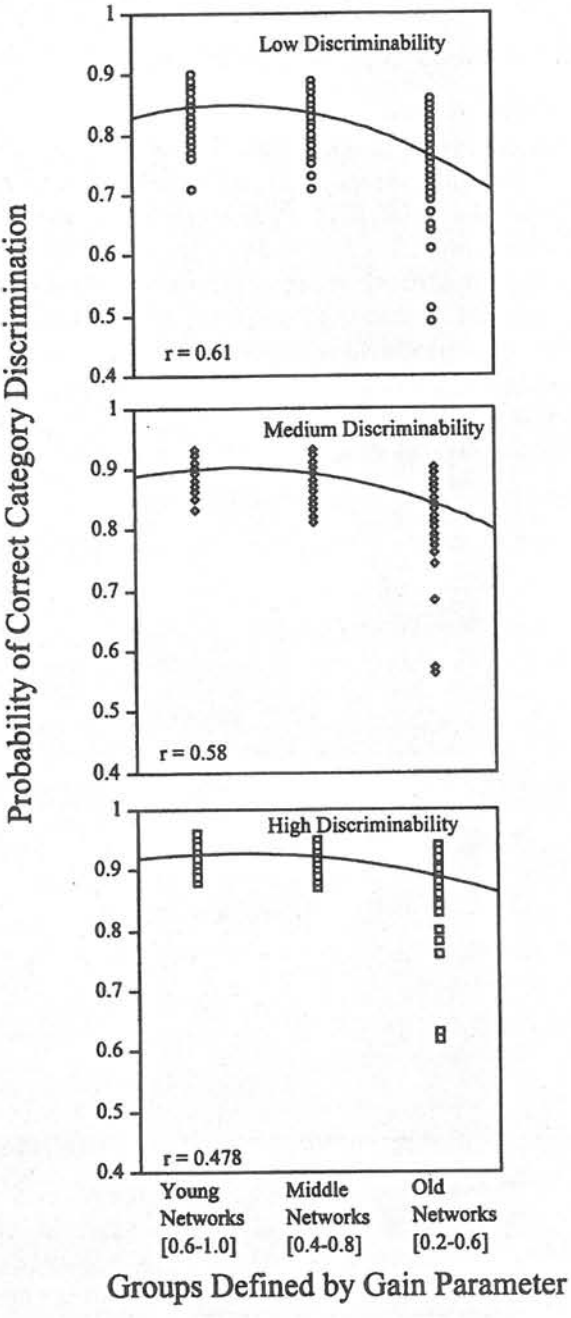

Figure 8. Inter-network variability as a function of the gain parameter in two-choice categorization with three levels of between-category discriminability.

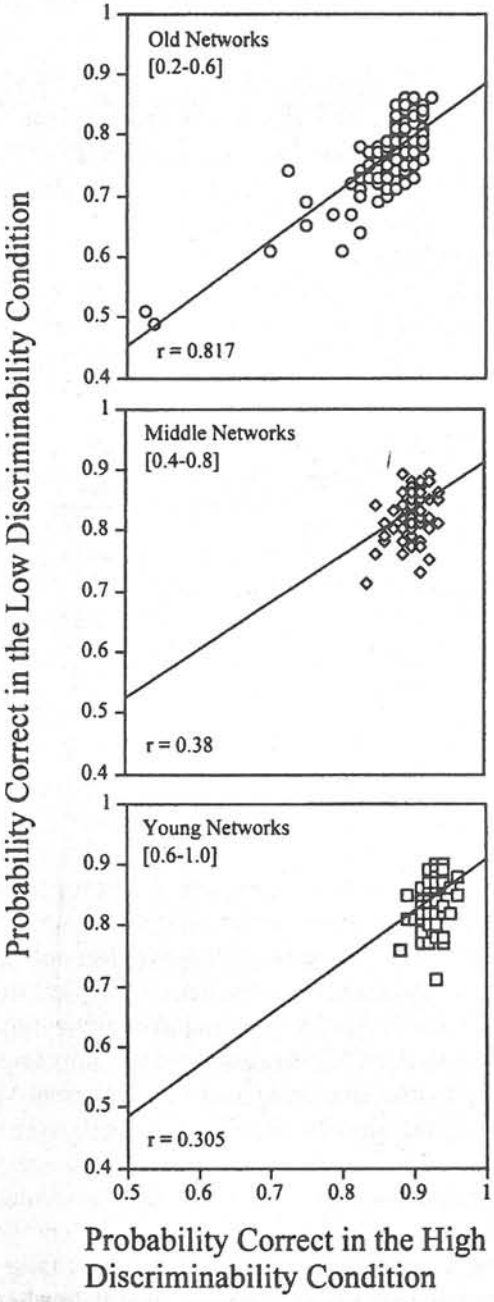

Figure 9. Correlations between performances in conditions of high and low discriminability as a function of the gain parameter.

Gain Parameter and Inter-Network Variability and Patterns of Intercorrelations

Similar to the previous results of paired-associate learning, Figure 8 shows that the magnitude of inter-network variability increases as the values of the gain parameters decreases. Concerning the gain parameter's effect on correlations between tasks, Figure 9 shows that performance in conditions of high and low discriminability are most strongly correlated in the group of old networks ($r_{old} > r_{mid} = r_{young}$, $z = 5.8$). Table 2 shows that as the values of the gain parameter decreases, the correlations between the three categorization tasks increases. Results from principal component analyses of these correlation matrices show that the percentage of variance accounted for by the first principle component increases as the values of the gain parameters decrease.

Table 2. Correlations between categorization tasks with different discriminability.

Young Networks Gain Parameter [0.6–1.0]			Middle Networks Gain Parameter [0.4–0.8]			Old Networks Gain Parameter [0.2–0.6]		
	High	Med.		High	Med.		High	Med.
Medium	0.62	–	Medium	0.70	–	Medium	0.94	–
Low	0.31	0.80	Low	0.38	0.79	Low	0.81	0.9
Variance accounted for by 1st PC: 72.2%			Variance accounted for by 1st PC: 75.0%			Variance accounted for by 1st PC: 92.8%		

General Discussion

This chapter describes a set of computational investigations undertaken to *relate* aging-induced deterioration of the catecholaminergic system *with* age-related dedifferentiation in cognitive abilities. Effects of catecholamines on neural transmission as indicated by some neurobiological studies (i. e., modulating the signal-to-noise ratio of a neuron and consequently raising the level of random variability in the CNS) were computationally implemented in connectionist networks by the stochastic gain manipulation. Values of the gain parameters which regulate the responsivity of the units were sampled randomly from distributions with different means to simulate age-related change in the efficacy of neurotransmitter systems. Results from simulations of paired-associate recall and a two-choice categorization task demonstrated that inter-network variability and the intercorrelations between tasks increased as the values of the gain parameters were reduced, regardless whether the gain manipulation was applied to a subset of the units (Simulation 1) or to all units (Simulation 2).

Disclaimers and Limitations

Before we further discuss the implications of our simulation results, some disclaimers and limitations about the present implementation should be pointed out. It should be made clear that we do not claim that the simulations presented here are, by themselves, sufficient enough to constitute a "theory" that relates the efficacy of neural transmission and the dedifferentiation of ability structure in old age. Rather, we view the connectionist approach taken here as a computational tool to aid the development of such a theory (cf. McCloskey, 1991). We have demonstrated that connectionist networks provided a computational framework for implementing the neuromodulatory effects of catecholamines as the gain parameter's effects on tuning a unit's responsivity and the level of intra-network variability. With this implementation, the simulations allow us to observe two sets of relationships. First, the relation between reducing the responsivity at the level of a single unit and the overall level of intra-network variability during information processing (referring back to Figures 1 and 2). And second, the relation between responsivity and intra-network variability at the system's level and the extent of inter-network variability and the magnitude of intercorrelations among different tasks at the performance level (refer back to Figures 4, 5, 8, and 9). Whether these relationships between the gain parameter, a unit's responsivity, intra-network variability and intercorrelations between tasks mimic closely the mechanisms underlying neural information processing and their behavioral manifestations is an open empirical question awaiting rigorous experimental validation. However, the simulation results do demonstrate a set of computational formalisms that would support these relationships if they indeed exist.

One additional limitation which applies to all quantitative models with even a moderate degree of complexity is the difficulty in discerning the analytical boundary of the specific manipulations implemented. Given that connectionist networks are adaptive learning systems, other network parameters, such as learning rate, momentum, bias, number of units, initial weights and even the task requirement specified by the actual input-output mappings are likely to interact with the gain parameter. We have kept all these other parameters constant within each of the two simulations reported here. However, we have not tested the generalizability of our simulation results in other parameter settings, nor is the investigation entailing a sufficient portion of the entire parameter space possible. Rather than searching through the parameter space, we think a more principled research strategy is to work out analytical solutions that could describe the simulated effects. Note, however, that such analytical solutions are not always attainable.

One observation from the process of implementing the simulations suggests that the gain parameter's effects on the level of inter-network variability and the magnitude of cross-task intercorrelations could dependent on whether the gain manipulation is effective enough to produce a difference in mean-level performance. In other words, given specific task requirements and number of training trials, is the difference in the means of the gain parameters large enough to produce a difference in mean-level performance. On the one hand, this suggests that the gain manipulation demonstrated in our simulations are necessarily constrained by other parameters of the network and the task requirements. On the other hand, this additional condition on the gain parameter's effects is actually not at odds with the experimental data. Results from psychometric studies have shown that the phenomenon of differentiation and dedifferentiation of ability structure can also be found along the dimension of ability, besides the age dimension. Specifically, the ability structure is less differentiated among groups of low performers than among groups of high performers (e. g., Deary et al., 1996; Detterman & Daniel, 1989). In addition, age-related decrements in the cognitive mechanics (e. g., processing speed and memory) are pervasive phenomena. In cases where age-related increase in variability or dedifferentiation are found, there are almost always age-related decrease in performance level as well.

One other limitation concerns the general issue about the level of abstraction that a given task should be represented. Arbitrary random vectors have been commonly used as the stimulus and response patterns in most connectionist simulations. During learning, connection weights are adjusted to reduce error between the network's actual output and the target output that's specified by the stimulus-response mapping defined by a given task. At the end of learning, the network stores the learned internal representation of the stimulus-response mapping in its weight patterns. The network's final weight patterns which determines its performance are jointly defined by a large number of network parameters *and* the to-be-learned task. Even in conditions when all other network parameters are held constant, a network can still "develop" quite different weight patterns, depending on the differences in the input-output mappings that are specified by different tasks. Relations between a network's internal representations of different tasks depend on the similarity between the stimulus-response mappings specified by the tasks. When random vectors are used to represent the stimulus and response patterns, there is no guarantee that a network's internal representations of different tasks would be related to each other; nor would the performances, being the outward expressions of the internal representations, of a group of networks in one task be systematically related to their performances in other tasks. In our simulation of paired-associate recall, similarities between the input-output mappings

of different tasks were created by nesting the shorter lists within the longer lists. Hence, we first generated a certain degree of intercorrelations between the tasks, before observing the effect of the gain manipulation on patterns of covariation. It is foreseeable that in conditions when more realistic representations of the stimuli and responses can be used (such as in simulations of facial recognition, pixel densities from the image of a face can be transcribed into a matrix of values; e. g., Valentin & Abdi, 1996), overlaps between different stimulus-response mappings can arise more naturally from the similarities between the stimuli. However, in many cases it is not clear as to what would be the realistic ways to represent the stimuli. Relatively few work has been done with respect to this issue, and it is not within the purview of this chapter to provide the solutions. Given that our goal is to demonstrate general principles between the gain parameter's effect on regulating intra-network variability, inter-network variability and patterns of covariations, as opposed to answer specific questions about performances in what types of tasks would be related, we feel justified to stay at the more abstract level of representing task requirements. Keeping these limitations in mind, we now turn to discuss implications of our simulation results with respect to the possible sources of interindividual variability and cognitive development.

Sources of Interindividual Variability

In this section, we highlight the implications of our results for the relationship between intraindividual and interindividual variability. One early proposal for the age-related increase in interindividual variability in cognitive performance (Birren et al., 1980; Rabbitt, 1981; Welford, 1980) is interindividual differences in the rate of brain aging (e. g., Birren, Woods, & Williams, 1980; Rabbitt, 1981; Welford, 1980). Results from our simulations together with some empirical results suggest, however, this need not be the case. At least, there exist other plausible explanations. Age-related increase in intraindividual variability or age-related behavioral slowing are two possible alternatives.

In the simulations, we demonstrated that as the stochastic gain manipulation increased the level of intra-network variability within each of the old networks, the inter-network variability measured across all networks in the group at the performance level also increased. This indicates that a greater degree of intra-individual variability in old people's cognitive functioning either at behavioral or biological level is at least one alternative explanation for the greater inter-individual variability observed at the group level. Aging-induced increase in CNS variability alone could also lead to an increase in interindividual variability at the group level. It is, therefore, not necessary to invoke a hypothesis about interindividual differences in the "rate" of neurobiological deterioration, in

order to account for age-related increase in interindividual variability observed at the behavioral level. One should also note a principle difference between these two hypotheses. This intraindividual-variability hypothesis accounts for a group-level phenomenon by an individual-level mechanism, whereas the hypothesis of individual differences in the rates of brain aging still uses a group-level mechanism to account for the group-level phenomenon. Hence, it is reasonable to argue that the hypothesis of increased intraindividual variability, supported by the simulation results, is more parsimonious and fundamental than the hypothesis of individual difference in the rates of neurobiological deterioration.

However, one should note that this mapping from intra-network to inter-network variability is not "unique" in the sense that the increase in inter-network variability can also be produced by reducing the network's learning rate (e. g., Li et al., 1996). Indeed, analyses at the behavioral level have also shown that the relationship between age and variability in RT is, to a great extent, channeled through age-related difference in RT itself. In other words, age-related slowing measured at the performance level is sufficient to account for age-related increase in interindividual variability (e. g., Hale, Myerson, Smith, & Poon, 1988; Salthouse, 1993). Both the simulation results and the empirical findings suggest then that age-related slowing can be yet another alternative account for age-related increase of interindividual variability.

Based on these simulation results alone one cannot choose between the two alternatives, favoring a age-related behavioral slowing or a neural-noise explanation, if the only issue of interest is whether these two alternatives can account for the phenomenon of age-related increase in interindividual variability. Likewise, one also cannot decide which of the two parameters, gain or learning rate, can better capture age-related increase in interindividual variability. However, these two hypotheses can be better contrasted if additional criteria, such as the feasibility of cross-level hypothesis generation and the scope of the explanation, are considered. These two explanations are not entirely compatible in the sense that they were proposed for phenomena at two different levels. Consequently, they also are not in direct conflict with each other. Cognitive aging researchers who subscribe to the behavioral-slowing view in general agree that what they take as a primitive in their explanations (i. e., behavioral slowing) needs to be somehow instantiated at the biological level (e. g., Salthouse, 1996). The stochastic gain manipulation has been shown to be able to account for both age-related slowing and additional cognitive aging phenomena that were formerly shown to be within the purview of the age-related slowing hypothesis (Li et al., 1996). Given that the gain parameter computationally implements the efficiency of neural information transmission, the gain parameter account of cognitive aging deficits provides at least one

version of computational formalism which demonstrates how behavioral slowing might be instantiated biochemically. From a modeling perspective, the learning rate parameter has a more restricted simulation scope than the gain manipulation. Specifically, although reducing the learning rate can also simulate a greater degree of inter-network variability, learning rate alone was not able (or less able in some cases) to account for some benchmark cognitive aging deficits, such as the age difference in asymptotic performance, the age by task complexity effect, and susceptibility to interference, all of which can be better accounted for by the stochastic gain manipulation (Li et al., 1996).

Implications for Cognitive Child Development

The question concerning variations in the structure of mental ability has also been investigated from a cognitive child development perspective. For instance, Garrett, Bryan, and Perl (1935) found that the first unrotated factor accounted for, respectively, 31%, 32% and 12% of the variance in a 10-tests battery for boys aged 9, 12, and 15 years (and 31.5%, 24% and 19.5% for girls of the same ages). These findings led Garrett (1946), who coined the term *differentiation hypothesis*, to state that "with increasing age there appears to be a gradual breakdown of an amorphous general ability into a group of fairly distinct aptitudes" (p. 375). In addition, initial biological evidence suggests that as infants mature from 2 to 17 weeks of age, the variability in both the latency and amplitude of the evoke potential in response to tones decreased (e. g., Thomas, Whitaker, Crow, Little et al., 1997). Behavioral level results with respect to word and phrase duration in speech also indicate a decline in variability from early childhood (age 7 year) to teenage (age 13 year) to adulthood (Chermak & Schneiderman, 1985), and this effect could not be explained by mean speaking rate.

Taken together, results both from developmental and cognitive aging studies seem to suggest two continua as the ontogeny of cognition goes from early childhood to adulthood and then into late adulthood. With respect to the structure issue, cognitive abilities are less differentiated in early childhood, become increasingly differentiated from childhood to adulthood, and start to dedifferentiate again going from adulthood to old age. (e. g., Baltes & Lindenberger, 1997; Burt, 1954; Reinert, 1970). With respect to the variability issue, the trend seems to be that variability is high in early childhood, decreases in adulthood, then increases again in late adulthood. However, empirical data supporting age-related decrease in interindividual and intraindividual variability from infancy to adulthood is not as available as the findings supporting age-related increase in variability during the aging process.

Given the similarities between the developmental and aging patterns, one

can expect that the gain manipulation can also account for related cognitive developmental phenomena at the formal level. However, one should ask whether there are reasons for assuming that the general neurobiological mechanisms associated with aging are also operative in child development but in a reversed direction? Empirical data at both the behavioral and biological level indicated that this might possibly be the case. Behavioral studies of lifespan cognitive development have shown that cognitive abilities, such as processing speed, selective attention, and memory span, show an inverted-U shaped lifespan function. For instance, using two tests of perceptual speed from the Woodcock-Johnson Tests, Kail and Salthouse (1994) showed that perceptual speed increases from age six to early adulthood, then becomes stable until mid-adulthood, and eventually starts to decline. In their review of lifespan development of selective attention, Plude et al. (1994) reported that the abilities to filter visual distractors and to search for attribute conjunctions improve throughout childhood, remain stable in adulthood, and decline in late adulthood. Similarly, others have shown that the ability to resist interference in paradigms involving Wisconsin Card Sorting Test (WCST) and the Stroop Test increases from age seven to early adulthood, remains stable until mid adulthood, then declines again as people age (e. g., Chelune & Baer, 1986; Comalli, Wapner, & Werner, 1962; Haaland, Vranes, Goodwin, & Garry, 1987). With respect to memory performance, a few studies have also shown a similar inverted-U lifespan developmental function for memory span (e. g., Case, 1985; Hasselhorn, 1988; Salthouse, 1990; Siegel, 1994). In light of these data, some developmental psychologists have proposed conceptual accounts to explain the phenomena of cognitive development and aging within a unified framework. For instance, lifespan variations in the efficacy of inhibitory mechanism (e. g., Bjorklund & Harnishfeger, 1995; Dempster, 1992), the amount of neural noise (cf. Plude, Enns, & Brodeur, 1994), and the speed of processing (Kail & Salthouse, 1994; Park, Smith, Lautenschlager, Earles et al., 1996) have all been *independently* proposed as the connecting thread for lifespan cognitive development. Interestingly, at the biochemical level, lifespan data concerning the efficacy of the dopaminergic system also indicated a continuum of age-related increase in dopamine metabolites extracted from human urine samples in the age range from 1 day old to 18 years old and a decrease from 18 to 55 years old (Dalmaz, Peyrin, Sann, & Dutruge, 1979). Given the roles of catecholamines in regulating the spontaneous firing rate of neurons in the prefrontal cortex, the related attentional and inhibitory mechanisms (e. g., Jay, Glowinski, & Thierry, 1995; Mora, Sweeney, Rolls, & Sannguinetti, 1976; Shelley, Catts, Ward, & Andrews, 1997), and processing speed (e. g., MacRae et al., 1988; Schultz et al., 1989; Spirduso et al., 1989), one might speculate that the rise and fall in the efficacy of neural transmission as modulated by the catecholamines (or other transmit-

ter substances showing similar functional properties) could be an important thread at the biological level for cognitive development across the lifespan.

Conclusion

In this chapter, we have reviewed empirical evidence for age-related dedifferentiation in cognitive abilities and age-related increase in variability at both the biological and behavioral levels, along with age-related differences in the integrity of neurotransmitter systems. A computational approach capturing the effects of the catecholaminergic system on regulating the sensitivity and variability of neural information processing was proposed to theoretically link findings at these different levels. Based on the simulation results, we suggest that a causal path from the responsivity of a neuron to the level of random variability within the CNS, and the behavioral manifestations of intraindividual variability, interindividual variability, and the ontogeny of the structure of cognitive abilities can at least be supported by the computational formalism specified here. We acknowledge that such cross-level theorizing runs the risk of losing the specifics for the general, and that the cross-level links we proposed here stay quite speculative, despite initial support from the simulations. However, we have demonstrated in this chapter that theorizing from a cognitive neuroscience orientation offers more possibilities for cross-level data integration, hypothesis generation and testing. Hopefully this will in the future provide us with a more integrated picture of lifespan cognitive development.

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