

advances in our understanding of the effects of aging on cognition than have been possible during the entire twentieth century.

Further Reading

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Aging, Neural Changes in

Introductory article

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Dementia is not an obligatory consequence of aging. Normal aging does, however, result in changes in cognition, caused by a combination of neurotransmitter abnormalities and alterations in the structure and function of the brain.

normal brain activation during performance of specific cognitive operations (functional neuroimaging using positron emission tomography or magnetic resonance imaging).

INTRODUCTION

The increasing proportion of older adults in the population of many countries has heightened interest in the cognitive and neural changes that accompany normal aging. The following sections elucidate the effect of aging on a range of cognitive capacities, and the neural changes that may explain the pattern of spared and impaired function. Particular attention is devoted to short-term and working memory (allowing the temporary storage and manipulation of information); declarative memory (requiring conscious awareness); and nondeclarative memory (formed without conscious awareness). The evidence presented here comes from behavioral studies in healthy volunteers (cognitive psychology), patients with brain lesions (neuropsychology), analyses of brain structure (volumetric magnetic resonance imaging) and observations of focal task-related changes in

DECLARATIVE MEMORY

Long-term memory is broadly divided into two components: declarative and nondeclarative memory. Declarative (explicit) memory is formed with conscious awareness, and requires the participation of medial temporal lobe structures, including the hippocampus. Declarative memory is what we use to help us remember the items we need to pick up at the grocery store, or the name of a friend whom we haven't seen in years. In general, declarative memory is more affected by normal aging than is nondeclarative memory. As an example of declarative memory, read the following words, and try to remember them. After reading through the list, write down as many words as you can remember, without looking back at the list: table, orange, calendar, computer, paper, needle, napkin, chair, sneeze, movie, sleep, castle, build, lunch, flower, dragon, plant, cushion, dolphin, muscle.

SENSORY MEMORY (PERCEPTION)

The sensory systems are those through which we receive direct input from the world via receptors: touch, taste, smell, vision, and hearing. Our perception of that input, however, can be influenced by a number of factors, such as our internal state, or the context in which the sensation occurred. Imagine you hear a loud banging sound while you are walking by a construction site. Now imagine that you hear the identical sound while walking alone on a deserted street late at night. Although the sound (sensory information) is identical in the two instances, the interpretation of the sound (perception) may differ greatly because it occurs in different contexts.

Some sensory systems are degraded as part of the normal aging process. Most commonly, older adults experience hearing loss. By the age of 80 years the majority of older adults have significant hearing loss. They also have visual deficits, including poorer color and luminance contrast, and many have a loss of central vision due to macular degeneration. Sensory and perceptual deficits can hinder adults' performance on many tasks. For example, Murphy and colleagues found that older adults are more affected by background noise when trying to remember word pairs than are young adults. These researchers proposed that part of this increased effect is due to degraded sensory representations, though attentional reductions probably also contribute. Older adults also may have more difficulty discriminating isoluminant colors such as blue and green, and are slower and less accurate on tasks that require color discrimination, such as color-naming tests, the Stroop Test, and the Wisconsin Card Sorting Test.

Nevertheless, with modifications to testing procedures (such as louder stimuli) most older adults are successfully able to perceive information. Perceptual priming, requiring visual processing, is spared with normal aging. Older adults are also able to repeat word lists or digit strings (presented either aurally or visually), suggesting that their sensory deficits are not profound enough to affect immediate memory. It is, nonetheless, important to control for perceptual confounds when interpreting the performance of older adults, and to match (equate) the perceptual capabilities of young and older adults (either by matching individuals or, more feasibly, matching the stimuli so that young and older adults perceive them equally). Without taking these measures, it is unclear whether impaired performance in older adults stems from a purely cognitive deficit or from

impaired perception. Particularly in memory studies, reduced perception may result in older adults having a degraded memory representation, subject to faster disruption over time.

SHORT-TERM MEMORY AND WORKING MEMORY

Short-term memory is a limited-capacity storage buffer for information to be remembered over a very short duration (a few seconds). The term 'short-term memory' is commonly used to mean recent memory, but that definition is not used by cognitive psychologists. Short-term memory consists of two components: a passive information store and an active rehearsal system. Working memory, in contrast, not only stores information but also updates and manipulates that information.

Read the following words, and try to keep them in mind for 10s: hill, milk, goat, tool, foot, pie. This type of storage requires short-term memory. To succeed in repeating the words 10s later, you might also have felt that you were 'rehearsing' those words (e.g. internally vocalizing them) to allow yourself to remember them. This phenomenon highlights the active rehearsal component of short-term memory. Now, read the words again, look away, and this time try to say them in alphabetical order. Simply rehearsing the words is insufficient to complete this task; rather, you also need to manipulate the words to place them in the proper sequence. This task, therefore, requires working memory.

The most widely accepted model of working memory, proposed by Baddeley and Hitch, defines working memory as consisting of three components: the central executive, the phonological or articulatory loop, and the visuospatial sketchpad. The central executive controls the allocation of attention, as well as the coordination and monitoring of activities, while the phonological loop and visuospatial sketchpad are slave systems of the central executive that temporarily maintain and manipulate verbal and nonverbal material, respectively.

Short-term memory is usually spared with aging, whereas working memory shows age-related decrements. This decline probably does not occur equally across all components of working memory, but rather targets only a subset of processes. Three components probably account for the majority of age-related working memory decline: processing speed, storage capacity, and inhibitory ability.

Processing Speed

Older adults are known to have a slowed speed of processing. Salthouse and colleagues proposed that decreased processing speed could account for some of the age-related declines in cognition. They suggested that cognitive performance suffers because (a) the slowed mental operations cannot be carried out within the necessary time frame, and (b) the increased time between mental operations makes it more difficult to access previously processed information. Processing speed can affect encoding because the quality and availability of perceptual information degrades over time, so information that is processed quickly will be encoded more effectively and, therefore, will have a more durable representation or memory trace.

The hypothesis of a relation between processing speed changes and cognitive decline has been confirmed in a number of studies. Longitudinal studies have shown that changes in speed of processing may predict longitudinal cognitive decline, and a number of researchers have found that controlling for speed eliminates age effects on various memory tasks.

Storage Capacity

Storage capacity is one component of short-term and working memory: the passive storage buffer that dictates how much information can be stored without rehearsal being used to 'refresh' that information. A reduction in storage capacity is likely to contribute to age-related working memory decline: older adults may be able to hold less information in mind. Reduced storage capacity could provide an alternate explanation to reduced processing speed. Thus, remembering what information was processed, or carrying out mental operations, would be restricted not by time pressure but by reduced storage capacity. Although storage capacity declines with age, it is not clear that this deficit is sufficient to explain the cognitive decrements in working memory that occur with aging.

Inhibitory Ability

Hasher and Zacks proposed the inhibitory deficit theory to account for changes in cognitive performance with age. 'Inhibition', in this theory, is the ability to ignore irrelevant information while focusing attention on pertinent information. The inability to filter out irrelevancies causes older participants' working memory to be filled with unneeded information, leaving less space for

task-relevant memories. This explanation, therefore, is not completely dissociable from a storage capacity explanation for cognitive aging.

Researchers have found evidence for inhibitory deficits in older adults on a variety of tasks. Commonly used paradigms for assessing inhibition are task-switching or set-shifting. On these tasks, participants must first remember one set of rules or pay attention to one salient characteristic, and then must switch rules or attend to a different characteristic. Most investigators have found that these tasks are sensitive to aging effects, with older adults being less able to ignore the previously relevant information.

LONG-TERM MEMORY

Long-term memory can be divided into two categories: episodic and semantic. Episodic memory entails retrieving information from a particular episode, localized in space and time (e.g. remembering seeing the Eiffel Tower on your first trip to Paris), while semantic memory requires retrieving factual information independent of any specific episode (e.g. knowing that the Eiffel Tower is in Paris). Recall of the word list given at the beginning of this article required episodic memory. You had to bring the word to mind, and also correctly remember that the word was on the list you had just read. Accessing the meaning of the words, however, required semantic memory.

Episodic Memory

Episodic memory appears to be more affected by normal aging than other memory processes. All aspects of episodic memory are not affected uniformly, however.

Factual and source memory

Episodic memory can be subdivided into two components: factual memory and contextual or source memory. Normal aging results in a disproportionate impairment in source memory as compared with fact memory. Even when older adults remember a fact or event, they have more difficulty than younger adults pinpointing the specific contextual details, such as where and when they learned a fact. For example, Spencer and Raz tested young and older adults on a test requiring them to remember facts, some true and some fictitious (e.g. 'Angela Lansbury regularly consults with astrologists'). After a delay, participants were asked to complete the fact ('Angela Lansbury regularly consults with ...'), and to say where they had learned the

fact (experiment or elsewhere) and whether the fact had been presented on a blue or pink card. Older adults were disproportionately impaired on the source recall than on the fact recall.

Source memory is believed to rely on the brain's frontal lobes. Measures of frontal lobe function correlate with measures of source memory, and reductions in source memory have been shown to occur in amnesic patients with frontal lobe lesions. The frontal lobes are also critical for linking events together in time. Aging results in frontal lobe dysfunction, probably connected to the source memory deficits seen in older adults.

Recall and recognition

Older adults show poorer performance on recall tests ('What words were on the word list?') where no cue is provided, than on recognition tests ('Was "cloud" or "table" on the word list?') where retrieval cues are provided. In general, older adults show improved performance on episodic memory tests when cues are provided during encoding or retrieval phases.

The source memory decrement, and the benefit provided to older adults with cues, are probably related to the robustness of the memory trace encoded by the older adults. Aging seems to affect the quality of the representation, such that general gist-based information is more easily encoded and retrieved than richer, item-specific information that includes not only the to-be-remembered information, but also the context in which it was learned. This hypothesis is supported by the finding that on recognition tasks, older adults are more likely than young adults to say that an item is 'familiar' (they feel they have encountered the item before), but less likely to say that they 'recollect' the item (remember something specific about the item's presentation).

Semantic Memory

One of the most readily reported complaints by older adults is their declining ability to recall the names of people and objects. Word finding difficulties are among the most severe deficits in normal aging. Naming deficits result in slower speed of picture naming, a greater number of speech disfluencies, and an increased number of tip-of-the-tongue effects.

Picture naming

Older adults' naming deficit is particularly pronounced for proper names, though studies have also reported longer naming times for nonproper

objects. The difficulty may be related in part to deficits in associative memory: the ability to form associations between a name and an object may be reduced in normal aging.

Tip-of-the-tongue effect

The tip-of-the-tongue effect occurs when a person has access to a word's meaning, but is unable to produce the phonological code. Older people report more tip-of-the-tongue experiences with everyday objects and with proper names than younger people. In addition, the accuracy of available information during a tip-of-the-tongue state is higher for young than old adults. For example, younger participants are more likely to state correctly the first letter of the word they are trying to remember than older participants. As with naming deficits, tip-of-the-tongue effects are more pronounced for proper names than for everyday objects. Better performance with everyday objects may be related to what Burke and colleagues refer to as 'summation of priming'. With everyday objects, connections from a variety of semantic associates converge on the correct name; but with proper names, older adults are handicapped without this type of summation.

NONDECLARATIVE MEMORY

Nondeclarative (implicit) memory is encoded and strengthened, across trials, without conscious awareness. It encompasses a heterogeneous group of processes and kinds of performance, including skill (motor) learning, repetition priming, and classical conditioning. These domains rely on distinct and separable neural substrates. Because of the task diversity, and range of necessary neural substrates, it is perhaps logical that nondeclarative memory is not uniformly impaired with aging.

Skill (Motor) Learning

In the 1960s, Milner demonstrated that the amnesic patient HM, while unable to form new declarative memories, could successfully learn a new motor skill. She asked HM to perform a mirror tracing task, in which he had to trace the outline of a star seen only in mirror-reversed view. Over 3 days of practice, his error scores decreased dramatically, and he maintained the learning from one day to the next, but he had no conscious recollection that he had done the task before. Corkin and colleagues administered additional skill-learning tasks to HM, confirming that he generally showed preserved

learning. Other investigators have also reported that amnesic patients can learn and retain motor skill learning without awareness of prior exposure to the task.

These results indicate that the brain structures that support conscious, declarative memory and which are damaged in amnesia (the hippocampus and other medial temporal lobe structures) are not critical for skill learning. Skill learning is thought to rely on the motor cortex, supplementary motor area, cerebellum, basal ganglia, and posterior parietal cortex. Older adults have reductions in the amount of dopamine and acetylcholine in the basal ganglia; they also show cerebellar dysfunction. These changes may result in slower acquisition of some motor learning tasks.

No consensus exists as to how skill learning is affected by aging. Researchers have found every possible outcome: equal performance in young and older adults, better performance in older adults, and poorer performance in older adults.

Repetition Priming

Priming is broadly defined as a faster or biased response to a stimulus based on prior exposure to that stimulus, or a related stimulus. As an example of priming, try to complete these word stems with the first word that comes to mind: nap—, dol—, cas—, cus—, tab—, dra—. You may have responded with words from the list given at the beginning of this article, without being consciously aware that you had done so. This effect, based on prior exposure to a stimulus, is an example of repetition priming.

Priming is not a unitary construct; rather, multiple processes contribute to priming effects. For discussion purposes, we will divide priming into two categories: perceptual priming and conceptual priming. These types of priming are dissociable and rely on separate neural substrates.

Perceptual Priming

Perceptual priming is based on the sensory characteristics of a stimulus. For example, if participants are shown the pseudoword 'pabhan', they will later be more likely to recognize that pseudoword when it is flashed briefly, than another pseudoword flashed at the same rate. Keane and colleagues proposed that perceptual priming effects are mediated by a structural-perceptual memory system localized to the occipital lobe; this hypothesis has been supported by neuropsychological and functional imaging studies.

Older participants frequently perform as well as younger adults on perceptual priming tasks. For example, Schacter and colleagues presented young and older adults with black-and-white drawings of three-dimensional objects in either structurally possible or impossible configurations. When participants had to judge whether the briefly presented stimuli were possible or impossible objects, young and older adults showed the same magnitude and pattern of priming, with robust priming for possible objects and no priming for impossible objects.

The finding of spared perceptual priming with aging is consistent with its reliance on the occipital and temporoparietal cortex because aging is thought to spare primary cortices and modality-specific association areas, including the occipital lobe.

Conceptual Priming

In contrast to perceptual priming, conceptual priming relies primarily on the semantic representation of the stimulus. For example, if participants are first presented with the category word 'fruit', they will be faster at determining that the word 'apple' is a real word than if they were first presented with the category word 'furniture'. Keane and colleagues proposed that conceptual priming is mediated by a lexical-semantic memory system recruiting temporoparietal regions. This hypothesis has been supported also by neuropsychological and neuroimaging studies.

Some studies have reported age-related deficits in priming experiments that are conceptual in nature, including lexical priming and priming for new word associations. Other researchers, however, have reported spared performance in older adults. The discrepancy may have stemmed from different task designs, or individual variation within the older populations.

CLASSICAL CONDITIONING

One of the most commonly used forms of classical conditioning is the eyeblink response. In delay conditioning, a neutral stimulus (a tone) is followed repeatedly by a biologically relevant stimulus (an air puff to the eye), and the two stimuli coterminate. The measure of learning is the subsequent ability of the tone, by itself, to elicit a biologically relevant response (an eyeblink), the conditioned response. The strength of the conditioned response increases gradually with repetition, making it possible to document the number of trials needed to

learn to a particular criterion. Older rabbits and older humans require significantly more trials than younger ones to acquire the association between the tone and the air puff, but considerable variability exists among older individuals. Results from neuroimaging and neuropsychology converge on the conclusion that the cerebellum is the critical neural substrate for delay conditioning. Because the cerebellum is affected by normal aging, the reduction in classical conditioning with normal aging is believed to result from less efficient cerebellar communication and output.

Trace conditioning differs from delay conditioning in that there is an unfilled interval between the offset of the neutral stimulus (the tone) and the onset of the biologically relevant stimulus (the air puff). The participant must therefore build up a representation, across trials, as to the relation between the tone and air puff. In addition to cerebellar recruitment, the hippocampus is critical for trace conditioning. The hippocampal contribution is likely to stem from the fact that delay conditioning is not purely a nondeclarative memory task; conscious awareness of the relation is mandatory for successful conditioning.

On the trace conditioning paradigm, young and middle-aged adults condition at a similar rate, but older animals and humans are impaired. These deficits may occur at an earlier age than deficits in delay conditioning, and may be more pronounced.

AGE OF COGNITIVE DECLINE

Methods of Assessment

The age of cognitive decline can be assessed using one of two designs: cross-sectional or longitudinal.

Cross-sectional studies use data collected from individuals considered to be representative of an entire population, and interpret differences among those individuals as indicative of differences across two or more populations. For example, a cross-sectional study of aging might examine the performance of adults in their twenties, fifties and eighties. If the 80-year-olds performed more poorly than the other groups, this difference would be attributed to age. This design requires that groups be equated (matched) on as many variables as possible (e.g. overall intelligence and perceptual ability, as well as lifestyle, psychological and medical factors) to assure that group differences are due to age and not to other differences.

A longitudinal study avoids many of these confounds by tracking the same group of individuals across time, and comparing their performance at

different time points. For example, a group of adults might be tested every 5 years for 20 years. Because each individual serves as his or her own baseline, the investigator does not have to worry about confounds such as intelligence or education level. Changes in overall health or perceptual ability over time must still be considered, and longitudinal studies can also be confounded by non-random drop-out rates (e.g. in a memory study, individuals who believe their memory is failing might be more likely to drop out of the study than those who believe their memory is good).

Cognitive Performance

The worsening performance across an extensive age range has led many researchers to divide the older adult population into 'young-old' and 'old-old' subgroups. This dichotomy was first proposed by Neugarten, who noted that these groups were dissociable not only by chronological age, but also by lifestyle changes. The young-old have fewer health limitations than the old-old, and the old-old are more likely to be widows or widowers than the young-old.

Researchers have used this dissociation to examine the progression of cognitive changes into the later decades of life. Most studies have confirmed that memory loss does not reach a plateau in the sixth or seventh decades; rather, memory decline continues throughout the later decades. Adults over the age of 70 years perform significantly worse on a range of recognition tasks compared with individuals in their seventh decade of life. Deficits in semantic memory and conditioning can also become more pronounced in the old-old.

The age at onset of decline differs depending on the type of function assessed. Semantic memory, as measured by tip-of-the tongue effects, has been found to be altered between the fifth and sixth decades. Woodruff-Pak and colleagues, however, found that eyeblink classical conditioning decrements began almost a decade earlier, with 40-year-olds showing significant impairments. Episodic memory, in contrast, remains relatively stable until around the seventh decade.

ANATOMICAL CHANGES

Longitudinal studies have found decreases in overall grey and white matter volumes with age, as well as increases in volumes of ventricular cerebrospinal fluid. The changes are not uniform across all brain regions, however. For example, the prefrontal cortex and medial temporal areas are more

affected than primary association cortices. The pattern of neural changes helps to clarify why some types of cognition are particularly affected by normal aging, while other cognitive processes are relatively spared.

Hippocampus and Other Medial Temporal Lobe Structures

Hippocampal function is impaired by normal aging. Functional neuroimaging studies have shown that the hippocampus and other medial temporal lobe structures are less activated by memory tests with aging, and these functional changes often correlate with memory performance. A quantitative imaging study assessing the volume of different brain regions also found that hippocampal volume is significantly correlated with performance on delayed recall tests. In fact, out of a variety of brain regions measured (including overall brain volume), hippocampal volume was the best predictor of delayed memory performance.

It is unclear whether there is substantial cell loss in this region, or whether the hippocampal dysfunction is related to neuropathological changes and cellular dysfunction affecting neuronal communication. On postmortem examination, adults over the age of 55 years typically show at least some entorhinal neurons that contain tangles, or where tangles are beginning to form. Brain neurochemistry also appears to be altered, with reductions in synaptic signaling. For example, long-term potentiation, thought to be a critical neural mechanism for learning and memory, is reduced with normal aging. Reductions in the number of NMDA (*N*-methyl-*D*-aspartate) receptors in the hippocampus, or reductions in the efficiency of the receptor, may mediate some of the age-related hippocampal dysfunction. Glucocorticoids, too, mediate hippocampal function, and increases in glucocorticoid levels may contribute to dysfunction.

Even studies that have found cell loss do not agree on which medial temporal lobe regions are most affected. While a number of studies found evidence for cell loss in the CA1 region of the hippocampus, not all studies have replicated this finding, using unbiased stereologic counting methods.

Cerebellum

Studies of humans, rats and rabbits suggest that the cerebellum, in particular the Purkinje (output) cells, is affected by aging. Older animals have fewer Purkinje cells, and those that remain have a

reduced efficiency. Because Purkinje cells are the major output system of the cerebellum, damage to these cells results in dramatically reduced cerebellar output. Evidence for structural changes in humans comes from a magnetic resonance imaging (MRI) study showing significant negative correlations between age and grey matter volume in the cerebellar vermis and hemispheres.

Prefrontal Cortex

The function of the prefrontal cortex is affected by aging. Older adults perform more poorly than younger adults on tasks that measure frontal lobe capacities, including the Wisconsin Card Sorting Test and the Stroop Test. Neuroimaging studies have indicated changes in prefrontal activation, particularly in dorsolateral prefrontal cortex. Even on tasks where young and older adults perform at similar levels, prefrontal regions in older individuals show different patterns of activation, including recruitment of additional areas, and reduced activation in other regions relative to young adults.

As with the hippocampal region, it is unclear what neuropathological changes account for deficits in frontal lobe capacities. Researchers have proposed that neuronal shrinkage, or reductions in the number of presynaptic terminals, may be responsible for some of the age-related impairment. Axonal abnormalities may also underlie age-related deficits. In a volumetric MRI study, Double and colleagues found frontal lobe white-matter atrophy, suggestive of reductions in axonal processes. They suggested that slowed cognitive processing may occur because of a decrease in the speed of nerve conduction due to such axonal changes. These alterations may account for the working memory deficits with normal aging.

Neurotransmitter and Neuromodulator Abnormalities

A neurotransmitter is a chemical messenger that is released by one neuron, travels across a space between two neurons (a synapse), and binds to the second neuron. In this way, information is passed between neurons. A neuromodulator is a chemical that is not itself a transmitter, but affects the release of neurotransmitters.

Dopamine

Age-related changes in the dopaminergic system are well documented in humans, monkeys, and rodents. Levels of dopamine and tyrosine

hydroxylase (an enzyme important for the production of dopamine) decrease with normal aging, and these reductions are particularly pronounced in the frontal lobes and basal ganglia. Postsynaptic alterations are also reported to occur with aging, including reductions in D2 dopamine receptors and some increases in D1 receptors.

Age-related reductions in dopamine levels may contribute to age-related working memory impairments. Dopamine depletion in the frontal lobes impairs performance on working memory tasks, and dopamine may be particularly important for inhibitory ability. Prefrontal cortex must sort out task-relevant information, and maintain that information in the face of other distractors. Dopaminergic systems may provide the basis for that allocation of attention. Dopamine may potentiate synapses associated with a reward (e.g. correct recall), thereby intensifying links between task-relevant computations, and weakening others.

Acetylcholine

Considerable evidence links acetylcholine to learning and memory. Acetylcholine is released when animals perform spatial memory tasks, and injection of cholinergic antagonists such as hyoscine (scopolamine) impairs memory acquisition in humans and nonhuman primates.

In aged animals, memory loss is correlated with hypoactive cholinergic neurons. For example, older rats show reduced excitatory postsynaptic potential amplitudes resulting from stimulation of the CA1 region of the hippocampus, suggesting that cholinergic neurons are less responsive in older animals. Older animals also show reductions in cholinergic receptor density that are particularly pronounced in the medial and caudal parts of the striatum, and in the frontal lobes.

Adrenal glucocorticoids

Stress hormones, too, are linked to the neural loss and dysfunction associated with normal aging. The adrenal cortex (in the adrenal glands, located near the kidneys) secretes glucocorticoids, which underlie our physical responses to threatening stimuli. In the short term, glucocorticoids are essential to our survival because under stressful conditions they increase the availability of energy substrates (blood glucose). Prolonged exposure to elevated glucocorticoid levels, however, can be detrimental, suppressing anabolic processes and depleting existing energy stores. With age, the stress response is not terminated as efficiently, causing glucocorticoid levels to remain elevated for significantly longer following stress.

The hippocampus, one of the main target sites for glucocorticoids, seems to be hardest hit by prolonged glucocorticoid exposure. When rats underwent experimental removal of the adrenal glands, disrupting glucocorticoid production, aged rats showed little or no evidence of hippocampal neuron loss as compared with control rats. These results link the production of glucocorticoids to the hippocampal atrophy that occurs with aging. Further evidence for this hypothesis comes from studies in Sapolsky's laboratory, showing that young rats treated with corticosterone show patterns of hippocampal cell loss similar to that in aged rats. Sapolsky and colleagues suggest that the effect of glucocorticoids on hippocampal neurons is probably related to metabolic changes stemming from the fact that glucocorticoids inhibit glucose uptake.

Rate of Decline

As discussed above, different cognitive processes decline at differing phases of the aging process. These differences are probably related to the times that neuropathological abnormalities appear in different brain regions. Cerebellar atrophy, thought to cause changes in the acquisition of a conditioned response, may start at an earlier age than most other brain changes, with significant atrophy present by the fifth decade of life. Other regions such as the medial temporal lobe or frontal lobe may not be altered until the seventh or eighth decades. Similarly, neurotransmitter changes, such as dopaminergic reductions, are thought to start around the seventh decade of life and to continue throughout the remaining adult years.

CONCLUSION

Aging does not affect all aspects of cognition uniformly. It does, however, affect a range of cognitive capacities. These changes are not static, but rather continue to intensify. Cognitive alterations are intimately linked to age-related changes in the neurotransmitter systems and in the structure and function of the brain.

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Agreement

Intermediate article

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Agreement in linguistics is a relationship of matching or systematic covariation of the features of constituents of a syntactic construct. All major syntactic categories and many minor categories can entertain agreement relationships of a variety of different kinds, typically involving subject–verb or modifier–head configurations.

AGREEMENT: GENERAL PROPERTIES

Agreement (or concord) is a relationship of matching or systematic covariation of the features of constituents of a syntactic construct. The constituents are said to agree in features: ϕ -features (where ' ϕ ' is a cover for person, number, gender); case (e.g., Latin *illarum duarum bonarum feminarum* – 'of those two good women', with genitive feminine plural

marked throughout); noun class (e.g., Bantu); or some other properties (e.g., categorial features, as in Chamorro complementizer agreement; or tense).

All major syntactic categories can entertain agreement relationships with other constituents. In many languages, finite verbs agree with their subjects ('subject agreement'), and there are also languages in which finite verbs can agree with one or more of their objects ('object agreement'), or with *wh*-extracted constituents ('*wh*-agreement' as in Bantu, Palauan, Chamorro); nonfinite verbs can also show agreement with their dependents (past participle agreement in Romance languages; inflected infinitives in Portuguese, Hungarian). Predicative adjectives can agree with their subjects, attributive adjectives with the head noun. Predicate nominals often agree with their subjects as well;