

Factors Associated with Changing Cognitive Function in Older Adults: Implications for Nursing Rehabilitation

Jamie S. Myers, MN RN AOCN



KEY WORDS

cognitive decline
cognitive rehabilitation
cognitive reserve

This article reviews the significant effects of aging on cognitive function. As people age, brain tissue volume decreases, white matter hyperintensities increase, and associated deficits are seen in working memory, attention, and executive function. Comorbidities include hypertension, diabetes, and cardiovascular risk factors. Another factor that affects cognitive function is the presence of apolipoprotein E-4, which is negatively correlated with cognitive function. In addition, decreased serum levels of endogenous sex hormones are related to changes in cognitive function, but hormone replacement therapy may be detrimental. Improved cognition has been associated with moderate alcohol intake, regular exercise, and exposure to novel stimuli. This article also examines research evaluating brain-plasticity-based training and rehabilitation to reverse losses in sensory, cognitive, and motor processing. Rehabilitation nursing strategies for dealing with the decline of cognitive function include educating patients and developing a program about lifestyle changes that will enhance cognitive stimulation; minimizing risks for and effects of hypertension, diabetes, and cardiovascular disease; recognizing and accommodating sensory deficits; and maintaining awareness of current research outcomes to guide evidence-based practice.

A significant body of literature exists covering the process of aging and its impact on cognitive function. It has been suggested that the 'graying of America' has stimulated this interest (Hillman et al., 2006). Evidence of cognitive decline begins to appear during people's early sixties (Barnes et al., 2007). Age-related declines are exhibited in processing speed, short-term memory, working memory, and long-term memory. These deficits have been associated with changes in brain structure and function (Hillman et al.). The goals of this paper are to summarize a subset of the literature that discusses the cognitive effects of aging, describe the relationship of factors associated with aging to the severity and speed of cognitive aging, and discuss evidence for rehabilitative strategies.

Pathophysiology of the Aging Brain

Postmortem studies have revealed a number of peculiarities in the brains of older adults. In their comprehensive review article, Raz and Rodrigue (2006) outlined a variety of postmortem changes found in the brain of older adults, including reduced brain weight and volume; ventriculomegaly and sulcal expansion; neuronal body loss in the neocortex, hippocampus, and cerebellum; neuronal shrinkage and dysmorphology; synaptic density reduction; dendritic spine loss; mitochondrial damage; and reductions in DNA-repair ability. Magnetic resonance imaging (MRI) makes it possible to measure the volume of brain tissue in living beings

and has shown decreases in volume for the hippocampus, amygdala, cerebellum, neostriatum (Raz & Rodrigue), and prefrontal cortex (Gunning-Dixon & Raz, 2003; Raz et al., 2005; Raz, Rodrigue, & Haacke, 2007) with age. Voxel-based morphometry is a test that measures brain density and structural properties of brain tissue. This technique has been used to measure sulcal morphology and shows that aging is related to sulcal width increases and sulcal depth decreases (Raz & Rodrigue).

White matter hyperintensities (WMH) are present in older adults (Raz & Rodrigue, 2006) and can be seen on T1-weighted MRI scans. The reduced cerebral perfusion associated with aging has been offered as a reason for why WMH develop in older adults. However, a variety of other factors may also be related, including hypertension and several risk factors for cerebrovascular disease such as carotid atherosclerosis (Raz & Rodrigue). WMH occur primarily in the frontal regions of cerebral white matter but can advance into posterior areas as age and cardiovascular risk factors increase (Raz & Rodrigue). Frontal lobe deterioration associated with aging has resulted in the frontal aging hypothesis, which is based on the assumption that the cognitive impairment of aging adults is similar to that observed of patients with frontal lobe lesions (Rodriguez-Aranda & Sundet, 2006). Deficits in both populations are seen in working memory, attention, and executive function (i.e., planning, cognitive flexibility, self-monitoring, and inhibition) (Rodriguez-Aranda & Sundet).

Factors Associated with Changing Cognitive Function in Older Adults: Implications for Nursing Rehabilitation

Aging is also associated with an increase of the ventricular system volume and a decrease in white and gray matter. The decline/reduction of white and gray matter begins during the fifth decade (Raz & Rodrigue, 2006).

Comorbidities and Associated Factors to Cognitive Changes

A variety of factors are associated with cognitive aging. One of the most important factors is hypertension. Chronic blood pressure elevation has been shown to increase the effects of aging on brain structures including the reduction of white and gray matter in the prefrontal lobes, decline of the hippocampus, and increased frontal WMH (Raz & Rodrigue, 2006; Raz, Rodrigue, & Acker, 2003). Uncontrolled hypertension has been shown to have a negative effect on cognitive function that is independent of aging (Brady, Spiro, & Gaziano, 2005; Schretlen et al., 2007). However, controlled hypertension may also be associated with deficits in brain structure and cognition (Raz, Rodrigue, & Acker).

Age-related reductions of white and gray matter may be associated with chronic exposure to glucocorticoids produced by stress. Glucocorticoid release has been shown to be related to hippocampal synaptic loss and atrophy as well as impaired memory in all age groups (Raz et al., 2005). Chronic stress has also been associated with prefrontal and thalamic density (Raz et al., 2005).

Homocysteine is an amino acid that is elevated in patients with cardiovascular risk factors and patients with chronic alcoholism. Some evidence suggests that homocysteines may be biomarkers associated with hippocampal atrophy, reductions in gray matter volume, and ventriculomegaly (Raz & Rodrigue, 2006). Angina pectoris, myocardial infarction, coronary heart disease, and intermittent claudication are all results of vascular disease, and a recent study also showed that these components of vascular disease are associated with poor cognitive function (Singh-Manoux, Britton, & Marmot, 2003). Raz and Rodrigue discussed the relationship of age-related vascular changes to the impairment of cerebral blood flow and subsequent brain injury.

Diabetes mellitus has been associated with impaired cognitive function (Barnes et al., 2007; Brands, Biessels, De Haan, Kappelle, & Kessels, 2005; Brands et al., 2006; Brands et al., 2007; Schretlen et al., 2007). Cognitive impairment and depressive symptoms occur in approximately one-third of Type II diabetic patients (Barnes et al., 2007). Mild-to-moderate effects on cognitive function are seen in Type I diabetics (Barnes, Yaffe, Satariano, & Tager, 2003; Brands et al., 2006).

Genetic abnormalities are related to impaired cognitive function. The apolipoprotein E (ApoE) genotype has been studied in association with the risk for developing Alzheimer's disease (AD) as well as the increased risk for cognitive impairment related to aging (Deary, Whalley, Batty, & Starr, 2006; Deary et al., 2002; Deary et al., 2004; Etnier et al., 2007). The presence of the ApoE-epsilon4 allele is a known risk factor for AD. It is negatively correlated with cognitive function in the older adult. Carriers of this allele have been shown to have a more rapid rate of cognitive function and memory decline than the general population (Etnier et al.).

Endogenous levels of sex hormones also play a role in cognitive function (Grady et al., 2002; Shumaker et al., 2003; Shumaker et al., 2004; Yaffe, Barnes, Nevitt, Lui, & Covinsky, 2001; Yaffe et al., 2007). Low levels of bioavailable estradiol are associated with a decline in global cognitive function and verbal memory (Yaffe et al., 2007). Estrogen receptors have been found in areas of the brain involved in learning and memory, such as the hippocampus (Yaffe et al., 2007). Lower levels of serum estradiol have also been reported in patients with AD (Yaffe et al., 2007). Estradiol is thought to be neuroprotective and can limit oxidative stress injury as well as protect neurons from the amyloid toxicity seen in AD (Yaffe et al., 2007). However, some studies evaluating the use of hormone replacement therapy have shown negative effects on cognitive function including increased risk of strokes, dementia, and cerebral infarcts (Raz et al., 2005; Shumaker et al., 2003; Shumaker et al., 2004). Yaffe and colleagues (2007) postulated that future studies need to evaluate lower doses of hormone replacement therapy to optimize rather than maximize estradiol levels in the brain.

Dietary factors also affect cognitive function. Vitamin-D deficiency is present in an estimated 25%–54% of adults aged 60 and older and in 74% of women with AD (Wilkins, Sheline, Roe, Birge, & Morris, 2006). Vitamin-D metabolism is less efficient in the elderly (Wilkins et al.). Because the primary source of vitamin D is sunlight, diet alone may not be sufficient for maintaining normal serum levels without supplementation (Wilkins et al.). Research evaluating the role of vitamin D in brain function and development is now under way. Vitamin-D receptors are present in the hippocampus. Some evidence of *in vitro* neuroprotection by vitamin D exists (Wilkins et al.).

Uric acid levels are also of interest even though the research on the topic has been somewhat contradictory. Elevated levels of uric acid have been associated with hypertension, atherosclerosis, and Type II diabetes (Schretlen et al., 2007). All of these ailments have been associated with cognitive impairment. However,

low levels of uric acid have been associated with AD. A recent study conducted by Schretlen and colleagues (2007) demonstrated that older adults with higher than normal levels of uric acid performed poorly on cognitive tests. Elevated uric acid levels are associated with an increased risk of cardiovascular disease and cognitive impairment in aging. The authors of this study concluded that taking medication to lower uric acid levels may improve cognitive functioning in older adults and suggested that clinical trials be developed to test this hypothesis.

Factors Associated with Maintaining Cognitive Function

Cognitive decline occurs at varying degrees in older adults. Certain health factors and behaviors may be protective in nature. Barnes and colleagues (2007) conducted a study comparing older women who maintained optimal cognitive function into old age with women who experienced minor cognitive decline. Women aged 65 and older were assessed for cognitive function at the time the study was initiated and throughout a 15-year period. Almost 10,000 women were included in the study; 9% maintained optimal cognitive function, 58% experienced minor decline, and 33% demonstrated major decline. The women who maintained optimal cognitive function exhibited the following characteristics: lack of diabetes, lack of hypertension, moderate alcohol consumption, facility with instrumental activities of daily living, access to a modest social network, and less likely to have comorbid medical conditions. This group was also younger, had more years of education, and had higher baseline cognitive function scores. They were less likely to smoke, refrain from exercise, perform poorly on physical tasks, have poor vision, or have depressive symptoms.

Moderate alcohol consumption has been linked to an improved cognitive performance in older adults as well as a reduction in the risk of dementia (Britton, Singh-Manoux, & Marmot, 2004; Ruitenberg et al., 2002). Evidence also suggests that older adults are more sensitive to alcohol and are less able to metabolize it (Lang, Wallace, Huppert, & Melzer, 2007); however, in a recent study conducted by Lang and colleagues, consuming one to two drinks per day was associated with a reduced risk of dementia. A similar study conducted by Ruitenberg and colleagues reported that moderate alcohol consumption is related to improved cognition and subjective well-being. Moderate alcohol consumption is hypothesized to improve cognition because of a protective effect on cardiovascular risk factors (Lang et al.). The study conducted by Britton and colleagues demonstrated that the positive effects extended to patients consuming approximately

30 drinks per week, and women experienced a stronger effect than men. The effects seen in this study were not limited to individuals with evidence of cardiovascular disease.

Dietary studies have been conducted to evaluate the potential benefits of fruit and vegetable intake on cognitive function (Kang, Ascherio, & Grodstein, 2005; Morris, Evans, Tangney, Bienias, & Wilson, 2006). Both fruits and vegetables are known to have antioxidant properties and are associated with lowered cardiovascular risk. It is interesting to note that only vegetable intake showed a positive impact on cognitive function in the elderly. The study conducted by Morris and colleagues (2006) evaluated fruit and vegetable intake in relation to the food components vitamin E, vitamin C, carotenoids, and flavonoids, which are all antioxidants. Consuming vegetables, particularly the green, leafy variety, had the strongest linear association to maintaining cognitive function. Participants with high fruit intake were more likely to have a myocardial infarction, hypertension, and diabetes. This result may explain why high fruit consumption was not shown to have a positive effect on cognition. The authors speculated that vegetables have a higher vitamin-E content than fruits. Vegetables are also frequently ingested in combination with added fats, such as butter, margarine, or salad dressing. Fats have been shown to increase the absorption of fat-soluble antioxidants. The study conducted by Kang (2005) included only women. Fruits were not associated with cognition or cognitive decline.

Folic acid supplementation also has been studied. Older adults absorb vitamins less efficiently (Morris, Jacques, Rosenberg, & Selhub, 2007), which puts them at risk for low levels of vitamin B-12. Folic acid supplementation in the presence of low vitamin B-12 levels has been associated with cognitive impairment; folic acid supplementation in the presence of normal vitamin B-12 levels has been shown to significantly improve age-related cognitive decline (Durga et al., 2007; Morris et al.).

A significant amount of evidence supports the positive relationship between increased exercise and improved cognition in older adults (Barnes et al., 2003; Deary et al., 2006; Etnier et al., 2007; Hillman et al., 2006; Lytle, Vander Bilt, Pandav, Dodge, & Ganguli, 2004; Raz & Rodrigue, 2006; Weuve et al., 2004; Yaffe et al., 2001). Exercise has been defined as physical activity (Hillman et al.; Weuve et al.; Yaffe et al., 2001), aerobic exercise (Lytle et al.; Raz & Rodrigue), and walking (Weuve et al.; Yaffe et al., 2001). Fitness also has been evaluated in relationship to cognitive function. Fitness has been categorized as cardiorespiratory (Barnes et al., 2003), aerobic (Etnier et al.), and physical (Deary et al., 2006). Some measures of fitness have included grip strength, time to walk 6

Moderate alcohol consumption has been linked to an improved cognitive performance in older adults as well as a reduction in the risk of dementia.

Key Practice Points

1. An important component of health education aimed at reducing the risk of cognitive decline associated with aging is providing older adults with information about healthy behaviors that can minimize the risk of hypertension, cardiovascular disease, and diabetes.
2. Exposing older adults to novel stimuli and engaging them in mentally challenging individual and social activities have been associated with enhanced cognitive reserve. Cognitive reserve is associated with decreased age-related cognitive decline.
3. Cognitive rehabilitation programs for older adults that enhance memory and cognitive function are being developed and studied. The results from this research may provide a foundation for evidence-based practice in the field of rehabilitation nursing.

miles, forced expiratory volume (Deary et al., 2006), and treadmill exercise duration with oxygen uptake efficiency slope and peak oxygen consumption (Barnes et al., 2003). Exercise and fitness have been associated with cognitive reserve, a protective factor for cognitive decline related to aging (Etnier et al.). Studies conducted with animals have shown that the neurochemicals associated with neuronal survival, brain plasticity, and synaptic function (e.g., neurotrophin factor, dopamine, serotonin) are enhanced with increased physical activity (Hillman et al.) and that new cerebellar capillaries develop (Hillman et al.). Studies with human subjects have demonstrated changes in structure and function to the frontal, prefrontal, and parietal cortices (Hillman et al.).

Evidence for Cognitive Rehabilitative Strategies

Five fundamental principles have been postulated as root causes of age-related cognitive decline (Mahncke, Bronstone, & Merzenich, 2006): neuronal atrophy; deteriorating cortical areas related to sensation, cognition, memory, motor control, and affect; neuronal apoptosis; physical and chemical neuropathology correlated with behavioral losses; and the virtually universal outcome of deterioration associated with extended human life. Mahncke and colleagues described “noisy processing” as a consequence of aging (p. 87). They stated that sensory input from all systems, including auditory, visual, tactile, and proprioceptive, is degraded as a result of sensory organ deterioration. As the brain adjusts to these changes, there are longer space and

time integration constants and less accurate details of spatiotemporally complex signals. For example, degraded auditory inputs make interpreting rapid speech patterns, particularly in the presence of background noise, more difficult for older adults (Mahncke et al.). High-frequency hearing loss and decreases in visual acuity also affect response time. Mahncke and colleagues discussed the typical reduction in levels of cognitive stimulation that occur as people age and provide the examples of retirement, “resting on one’s laurels,” or pursuing only activities at which one excels (p. 86) as possible explanations for the reductions. They cited studies that relate the lack of brain engagement with negative changes in the production and function of neurotransmitters and receptors. These changes have been associated with impaired learning and memory capacities.

Mahncke and colleagues (2006) defined *brain plasticity* as the lifelong capacity for physical and functional brain change. Previously, brain plasticity was limited to the developing brain; however, in the last decade, research has shown that new neurons still grow during adulthood (Pinel, 2006). The brain is capable of reorganization throughout the adult life span and can develop new short-range interconnections in response to experiences. Studies have shown that participation in intellectually stimulating activities can slow cognitive decline and help maintain cognitive function (Daffner et al., 2006). Studies with animals have shown that older animals exposed to complex environments demonstrate neurogenesis, synaptogenesis, and dendritic complexity. These animals were more engaged by novel stimuli and exhibited greater preservation of cognitive function (Daffner et al.). Daffner and colleagues conducted studies with humans to compare cognitively high and cognitively average older adults. Their early work led to the hypothesis that high cognitive competence in older adults reflects significant cognitive reserve and compensation for age-related brain changes. They exposed older adults to a combination of routine and novel stimuli. Cognitively high-performing adults demonstrated increased responses to novel stimuli. The authors concluded that cognitively high-performing adults adapted by developing compensatory increases in neural activity and appropriating more brain resources. They also postulated that this compensatory strategy is likely to fail when there are multiple tasks and competition for brain resources.

Using the theory of brain plasticity and the relationship of continued intellectual stimulation to positive outcomes in cognitive function, Mahncke and colleagues (2006) have begun studying a brain-plasticity-based

training program to evaluate the feasibility and potential for improving the cognitive function of older adults. Their research demonstrated that behavioral training can reverse declines in sensory, cognitive, and motor processing. Recently, they conducted a pilot study comprising 73 participants aged 60 or older who met the following criteria: a minimum standard on the Mini-Mental State Examination and scores consistent with normal aging on a repeatable battery for the assessment of neuropsychological status (RBANS). Participants were randomly assigned to the training group, active control group, or no-contact control group. The training group received the program training for 1 hour daily, 5 days per week for 8 weeks. The active control group participated in an auditory and visual learning activity for the same time frame. The no-contact group engaged in no study-related activities during the 8 weeks. All participants completed neuropsychological assessment batteries at baseline and after the 8-week training period. The primary focus of the training program was to renormalize the auditory system. The primary outcome measure was a global auditory memory score based on six auditory tests of the RBANS. The authors concluded this approach was feasible; pre- and posttesting documented significant memory improvement within the training group.

Implications for Future Research

The literature on brain plasticity and factors associated with maintaining cognitive function is encouraging. Despite the inevitable effects of aging on the human brain, it appears there are a variety of viable strategies available for minimizing its effect. Many of the studies reviewed in this article recommend further research to definitively identify interventions for older adults. However, some recommendations can be made now based on research already completed. Health education programs should be revised to include the ramifications of aging on cognitive function. Healthy behaviors shown to minimize the risk of cardiovascular disease, such as regular moderate exercise and the consumption of leafy, green vegetables, should be recommended for minimizing age-related cognitive impairment. Maintaining a balanced diet with sufficient amounts of antioxidants, vitamin B-12, and folic acid should also be recommended (with the caveat that normal vitamin B-12 levels need to be established before folic acid is administered). Vitamin-D supplementation or daily exposure to sunlight for 15 minutes could be recommended if the patient has no contraindications to these treatments. Moderate alcohol intake could be considered depending on the individual's beliefs about alcohol consumption and whether there is any history of substance abuse. Not smoking has a

variety of benefits including a decreased risk for cardiovascular disease. The data about ongoing exposure to intellectually stimulating activities are also important to stress to patients. When older adults are made aware of the potential ramifications of decreasing activities in later life, they may opt for lifestyle changes that will promote cognitive function.

Further study is needed regarding uric acid levels and endogenous sex hormones. Because the results of earlier studies were contradictory in nature, it is premature to speculate whether xanthine oxidase inhibitors, uricosuric medications, or hormone replacement therapy are harmful or beneficial interventions for cognitive decline. It will be very interesting to review future study results from Mahnke and colleagues' (2006) brain-plasticity-based training program. The first pilot study focused on the auditory system. Perhaps future studies will also incorporate the visual, tactile, and motor systems.

According to the 2006 U.S. Census Bureau report, the number of adults aged 65 and older will double between the years 2000 and 2030 (Wan, Sengupta, Velkoff, & DeBarros, 2006). Eighty percent of older adults have at least one chronic health problem, and 50% have two. The most common ailments include cardiac disease, hypertension, and diabetes (Wan et al.). Because these chronic diseases are associated with an increased risk of cognitive impairment, there is the potential for a significant impact on American society related to the provision of health care and the need for assisted-living accommodations. On a positive note, the 2006 U.S. Census Bureau report speculates that in the future, the older population is likely to be better educated and healthier than the elderly population studied during the time the report was written. The report also predicts that ongoing research to understand chronic diseases may produce improvements in treatment and prevention. Minimizing the occurrence of cardiac disease, hypertension, and diabetes would be a positive step toward reducing risk factors for cognitive decline in the older adult.

Implications for Rehabilitation Nursing Practice

Rehabilitation nurses are in a unique position to help older adults maintain an optimal level of cognitive function. One opportunity is to develop or revise community awareness programs to include some or all of the recommendations listed in the previous section. Health education programs for older adults could be provided in a variety of venues, such as acute and long-term rehabilitation facilities, community centers, and churches with senior programs. Rehabilitation nurses could

Despite the inevitable effects of aging on the human brain, it appears there are a variety of viable strategies available for minimizing its effect.

Factors Associated with Changing Cognitive Function in Older Adults: Implications for Nursing Rehabilitation

partner with organizations such as AARP to ensure that the appropriate educational materials are made available to members. Community education could be directed at the “sandwich generation” or middle-aged adults involved with the care of aging parents. As more is learned about the etiology of cognitive changes associated with aging and viable interventions are tested, it will be important to include this information in nursing curricula and continuing education programs.

Rehabilitation nurses working with older adults could consider promoting and providing social activities that involve intellectual effort, such as cards or other games, book clubs, discussion groups, and music appreciation groups. Providing easy access to individual activities that stimulate mental activity, such as puzzles and Sudoku, may be beneficial.

A component of rehabilitation nursing is the recognition of and accommodation for sensory changes that occur in older adults, such as changes in auditory and visual acuity. Reduction of background noise, enhanced lighting, and speaking slowly and clearly are all important techniques for creating an environment conducive to communication and education.

Finally, it is important for rehabilitation nurses to be aware of current research to maintain evidence-based practice. As research matures, rehabilitation nurses will be able to facilitate appropriate referrals to successful programs or lead efforts to incorporate these types of programs into existing patient care in their facilities.

About the Author

Jamie S. Myers, MN RN AOCN, is a doctoral student at University of Kansas Medical Graduate School of Nursing in Kansas City, KS. Address correspondence to her at jamyers@swbell.net.

References

- Barnes, D. E., Cauley, J. A., Lui, L. Y., Fink, H. A., McCulloch, C., Stone, K. L., et al. (2007). Women who maintain optimal cognitive function into old age. *Journal of the American Geriatric Society, 55*(2), 259–264.
- Barnes, D. E., Yaffe, K., Satariano, W. A., & Tager, I. B. (2003). A longitudinal study of cardiorespiratory fitness and cognitive function in healthy older adults. *Journal of American Geriatric Society, 51*(4), 459–465.
- Brady, C. B., Spiro, A., & Gaziano, J. M. (2005). Effects of age and hypertension status on cognition: The veterans affairs normative aging study. *Neuropsychology, 19*(6), 770–777.
- Brands, A. M., Biessels, G. J., De Haan, E. H., Kappelle, L. J., & Kessels, R. P. (2005). The effects of type I diabetes on cognitive performance: A meta-analysis. *Diabetes Care, 28*(3), 726–735.
- Brands, A. M., Kessels, R. P., Hoogma, R. P., Henselmans, J. M., van der Beek Boter, J. W., Kappelle, L. J., et al. (2006). Cognitive performance, psychological well-being, and brain magnetic resonance imaging in older patients with type I diabetes. *Diabetes, 55*(6), 1800–1806.
- Brands, A. M., Van den Berg, E., Manschot, S. M., Biessels, G. J., Kappelle, L. J., De Haan, E. H., et al. (2007). A detailed profile of cognitive dysfunction and its relation to psychological distress in patients with type 2 diabetes mellitus. *Journal of the International Neuropsychological Society, 13*(2), 288–297.
- Britton, A., Singh-Manoux, A., & Marmot, M. (2004). Alcohol consumption and cognitive function in the Whitehall II study. *American Journal of Epidemiology, 160*(3), 240–247.
- Daffner, K. R., Ryan, K. K., Williams, D. M., Budson, A. E., Rentz, D. M., Wolk, D. A., et al. (2006). Increased responsiveness to novelty is associated with successful cognitive aging. *Journal of Cognitive Neuroscience, 18*(10), 1759–1773.
- Deary, I. J., Whalley, L. J., Batty, G. D., & Starr, J. M. (2006). Physical fitness and lifetime cognitive change. *Neurology, 67*(7), 1195–1200.
- Deary, I. J., Whiteman, M. C., Pattie, A., Starr, J. M., Hayward, C., Wright, A. F., et al. (2002). Cognitive change and the ApoE epsilon 4 allele. *Nature, 418*(6901), 932.
- Deary, I. J., Whiteman, M. C., Pattie, A., Starr, J. M., Hayward, C., Wright, A. F., et al. (2004). Apolipoprotein e gene variability and cognitive functions at age 79: A follow-up of the Scottish mental survey of 1932. *Psychology and Aging, 19*(2), 367–371.
- Durga, J., van Boxtel, M. P., Schouten, E. G., Kok, F. J., Jolles, J., Katan, M. B., et al. (2007). Effect of 3-year folic acid supplementation on cognitive function in older adults in the FACIT trial: A randomised, double blind, controlled trial. *Lancet, 369*(9557), 208–216.
- Etnier, J. L., Caselli, R. J., Reiman, E. M., Alexander, G. E., Sibley, B. A., Tessier, D., et al. (2007). Cognitive performance in older women relative to ApoE-epsilon4 genotype and aerobic fitness. *Medicine and Science in Sports and Exercise, 39*(1), 199–207.
- Grady, D., Yaffe, K., Kristof, M., Lin, F., Richards, C., & Barrett-Connor, E. (2002). Effect of postmenopausal hormone therapy on cognitive function: The heart and estrogen/progestin replacement study. *American Journal of Medicine, 113*(7), 543–548.
- Gunning-Dixon, F. M., & Raz, N. (2003). Neuroanatomical correlates of selected executive functions in middle-aged and older adults: A prospective MRI study. *Neuropsychologia, 41*(14), 1929–1941.
- Hillman, C. H., Moti, R. W., Pontifex, M. B., Posthuma, D., Stubbe, J. H., Boomsma, D. I., et al. (2006). Physical activity and cognitive function in a cross-section of younger and older community-dwelling individuals. *Health Psychology, 25*(6), 678–687.
- Kang, J. H., Ascherio, A., & Grodstein, F. (2005). Fruit and vegetable consumption and cognitive decline in aging women. *Annals of Neurology, 57*(5), 713–720.
- Lang, I., Wallace, R. B., Huppert, F. A., & Melzer, D. (2007). Moderate alcohol consumption in older adults is associated with better cognition and well-being than abstinence. *Age and Ageing, 36*(3), 256–261.
- Lytle, M. E., Vander Bilt, J., Pandav, R. S., Dodge, H. H., & Ganguli, M. (2004). Exercise level and cognitive decline: The MoVIES project. *Alzheimer Disease and Associated Disorders, 18*(2), 57–64.
- Mahncke, H. W., Bronstone, A., & Merzenich, M. M. (2006). Brain plasticity and functional losses in the aged: Scientific bases for a novel intervention. *Progress in Brain Research, 157*, 81–109.
- Morris, M. C., Evans, D. A., Tangney, C. C., Bienias, J. L., & Wilson, R. S. (2006). Associations of vegetable and fruit consumption with age-related cognitive change. *Neurology, 67*(8), 1370–1376.
- Morris, M. S., Jacques, P. F., Rosenberg, I. M., & Selhub, J. (2007). Folate and vitamin B-12 status in relation to anemia, macrocytosis, and cognitive impairment in older Americans in the age of folic acid fortification. *American Journal of Clinical Nutrition, 85*(1), 193–200.
- Pinel, J. J. (2006). *Biopsychology (Sixth ed.)*. Boston: Pearson Allyn and Bacon.
- Raz, N., Lindenberger, U., Rodrigue, K. M., Kennedy, K. M., Head, D., Williamson, A., et al. (2005). Regional brain changes in aging healthy adults: General trends, individual differences and modifiers. *Cerebral Cortex, 15*(11), 1676–1689.

- Raz, N., & Rodrigue, K. M. (2006). Differential aging of the brain: Patterns, cognitive correlates and modifiers. *Neuroscience and Biobehavioral Reviews*, 30(6), 730-748.
- Raz, N., Rodrigue, K. M., & Acker, J. D. (2003). Hypertension and the brain: Vulnerability of the prefrontal regions and executive functions. *Behavioral Neuroscience*, 117(6), 1169-1180.
- Raz, N., Rodrigue, K. M., & Haacke, E. M. (2007). Brain aging and its modifiers: Insights from in vivo neuromorphometry and susceptibility weighted imaging. *Annals of the New York Academy of Sciences*, 1097, 84-93.
- Rodriguez-Aranda, C., & Sundet, K. (2006). The frontal hypothesis of cognitive aging: Factor structure and age effects on four frontal tests among healthy individuals. *Journal of Genetic Psychology*, 167(3), 269-287.
- Ruitenbergh, A., van Swieten, J. C., Wittteman, J. C., Mehta, K. M., van Duijn, C. M., Hofman, A., et al. (2002). Alcohol consumption and risk of dementia: The Rotterdam study. *Lancet*, 359(9303), 281-286.
- Schretlen, D. J., Inscore, A. B., Jinnah, H. A., Rao, V., Gordon, B., & Pearson, G. D. (2007). Serum uric acid and cognitive function in community-dwelling older adults. *Neuropsychology*, 21(1), 136-140.
- Shumaker, S. A., Legault, C., Kuller, L., Rapp, S. R., Thal, L., Lane, D. S., et al. (2004). Conjugated equine estrogens and incidence of probable dementia and mild cognitive impairment in postmenopausal women: Women's health initiative memory study. *Journal of the American Medical Association*, 291(24), 2947-2958.
- Shumaker, S. A., Legault, C., Rapp, S. R., Thal, L., Wallace, R. B., Ockene, J. K., et al. (2003). Estrogen plus progestin and the incidence of dementia and mild cognitive impairment in postmenopausal women: The women's health initiative memory study: A randomized controlled trial. *Journal of the American Medical Association*, 289(20), 2651-2266.
- Singh-Manoux, A., Britton, A., & Marmot, M. (2003). Vascular disease and cognitive function: Evidence from the Whitehall II study. *Journal of American Geriatric Society*, 51(10), 1445-1450.
- Wan, H., Sengupta, M., Velkoff, V. A., & DeBarros, A. (2006). 65+ in the United States: 2005. Retrieved February 5, 2008, from www.census.gov/prod/2006pubs/p23-209.pdf.
- Weuve, J., Kang, J. H., Manson, J. E., Breteler, M. M., Ware, J. H., & Grodstein, F. (2004). Physical activity, including walking, and cognitive function in older women. *Journal of the American Medical Association*, 292(12), 1454-1461.
- Wilkins, C. H., Sheline, Y. I., Roe, C. M., Birge, S. J., & Morris, J. C. (2006). Vitamin D deficiency is associated with low mood and worse cognitive performance in older adults. *American Journal of Geriatric Psychiatry*, 14(12), 1032-1040.
- Yaffe, K., Barnes, D. E., Lindquist, K., Cauley, J. A., Simonsick, E. M., Penninx, B., et al. (2007). Endogenous sex hormone levels and risk of cognitive decline in an older biracial cohort. *Neurobiology of Aging*, 28(2), 171-178.
- Yaffe, K., Barnes, D. E., Nevitt, M., Lui, L. Y., & Covinsky, K. (2001). A prospective study of physical activity and cognitive decline in elderly women: Women who walk. *Archives of Internal Medicine*, 161(14), 1703-1708.

Earn nursing contact hours

Rehabilitation Nursing is pleased to offer readers the opportunity to earn nursing contact hours for its continuing education articles by taking a posttest through the ARN Web site. The posttest consists of questions based on this article, plus several assessment questions (e.g., how long did it take you to read the article and complete the posttest?). A passing score of 88% on the posttest and completion of the assessment questions yield one nursing contact hour for each article.

To earn contact hours, go to www.rehabnurse.org, and select "Continuing Education." There you can read the article again, or go directly to the posttest assessment. The cost for credit is \$9 per article. You will be asked for a credit card or online payment service number.

The Association of Rehabilitation Nurses is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation (ANCC COA).



ARN Rehabilitation Nursing

Announcing the 18th Annual

Voices of Rehabilitation Nursing Writers' Contest

Every nurse has a story to tell—even you! If you have had a standout experience in rehabilitation nursing that has inspired you and could benefit your colleagues in their professional practice, *Rehabilitation Nursing* encourages you to write about it. **You could win a year's free membership!**

The Voices of *Rehabilitation Nursing* Writers' Contest is the one opportunity ARN members (and interested nonmembers) have each year to achieve special recognition for submitting an article of compelling human interest in the field of rehabilitation nursing. **All contest entrants receive a \$35 credit toward the purchase of ARN merchandise, and the winner receives a free 1-year membership in ARN.**

As always, the winning article will be published in the "Perspectives" column of ARN's prestigious, peer-reviewed journal, *Rehabilitation Nursing*.

The journal's editorial team emphasizes the need for *inspiring* contest entries. Articles should emphasize a professional experience that

- summarizes an original or highly successful rehabilitation approach;
- describes a unique team-building scenario; or
- illustrates a special rehabilitation-related contribution to care.

Other topics and approaches may be appropriate for contest submission. If you have additional questions, send them to info@rehabnurse.org. Send submissions to gpannozzo@connect2amc.com.

Start planning now!

Entries must be e-mailed or postmarked by **July 18, 2008**.

Association of Rehabilitation Nurses • 4700 W. Lake Avenue, Glenview, IL 60025-1485 • www.rehabnurse.org