Memory Impairment in the Aging Brain

An edited collection of articles and news reports from the Mempowered website

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Cognitive function in various clinical conditions

This section is offshoot of my gathering of news items about memory. I am not a medical expert. My background is in psychology. The information I have gathered here should not be taken as providing any advice.

- Short-term declines in cognitive function can occur in elderly subjects after surgery, and persists in a small percentage.
- Heart bypass surgery in particular is associated with cognitive decline - estimates of its prevalence range from 33% to 82%. This decline may persist in as many as 42% of patients. Age and level of education are both factors in determining the likelihood of long-term decline. The presence of a gene (apolipoprotein E4) carried by some 25% of the population may also increase the likelihood of decline after bypass surgery. There also seems to be a link between post-operative fever and cognitive decline. Long-term decline in cognitive function may be more a result of cardiovascular risk factors than the surgery itself.
- High blood pressure in those over 60 seems to be associated with greater risk of cognitive decline.
- High blood pressure and other circulatory problems, such as cardiovascular risk factors and diabetes, are linked to cognitive decline, perhaps through causing abnormalities in the white matter of elderly persons' brains.
- Those with the gene ApoE4 also appear to have more difficulty recovering from traumatic brain injury.
- Two drugs used for Alzheimer's have also been found to help those suffering from dementia following stroke.
- A specific skills approach is having some success in helping those who suffer from attention problems following stroke.

News reports

Cardiac arrest

Heart failure linked to cognitive impairment

A study of 50 patients with chronic heart failure, matched with 50 people without HF, has found that patients with HF scored significantly lower than controls on 14 of 19 cognitive tests, and 46% of the HF patients were rated as having mild to severe cognitive impairment, compared to 16% of mild impairment in controls. The degree of cognitive impairment was closely related to the number of myocardial infarctions experienced. These findings have important implications for the care of patients with HF. [1]

Heart disease linked to worse cognition

Another report has come out from the large Whitehall study, this time on the subject of coronary heart disease and cognition. The study found that coronary heart disease was associated with a worse performance in mental processes such as reasoning, vocabulary and verbal fluency, and that the longer ago the heart disease had been diagnosed, the worse was the person's cognitive performance. This effect was particularly marked in men. Although there has been quite a lot of research on cardiovascular disease and impaired cognition, this is the first, large study to specifically examine the association between coronary heart disease and cognition. The major risk factors for coronary heart disease are all modifiable: cigarette smoking, diabetes, high cholesterol levels and high blood pressure. The findings also support the growing view that it is events happening in earlier life that have an impact on whether or not dementia develops in older age. [2]

Why cardiac arrest may hinder ability to learn certain tasks

Cardiac arrest can take a particularly harsh toll on the hippocampus, the area of the brain that plays a critical role in memory and navigation. A new mouse study found that mice that had had a (surgically induced) heart attack had far more difficulty learning a new spatial task than did healthy mice (controls were given the surgery, but didn’t have a cardiac arrest induced). Mice in the heart attack group spent about eight minutes in cardiac arrest – enough time to stop the flow of

oxygen to the brain. Analysis of the brain tissue found an overall 18% decrease in dendritic spine density in the hippocampus in the cardiac arrest mice compared to the control mice (dendritic spines are projections from neurons involved in sending signals throughout the central nervous system and the body). The researchers are now looking at how different types of social interactions influence the number and health of neurons that survive a heart attack. [3]

Cardiovascular risk factors

More evidence bypass surgery not responsible for cognitive impairment

A 6-year study of 326 heart patients has found no differences in brain impairment between those who had on-pump coronary artery bypass surgery (152 patients), off-pump bypass surgery patients (75 patients), and those who had drugs and arterial stents to keep their blood vessels open instead of bypass surgery (99 patients). However, all of them were found to have experienced significant cognitive decline over the six-year study period on tests of verbal memory, visual memory, visuoconstruction, language, motor speed, psychomotor speed, attention, and executive function, when compared to 69 heart-healthy people who had no known risk factors for coronary artery disease. The findings provide more evidence that it is the disease and not the surgery that causes long-term cognitive problems. [4]

Long-term cognitive decline in bypass patients not due to surgery

Another study has come out supporting the view that coronary bypass patients have no greater risk of long-term cognitive decline than patients not undergoing surgery. The study involved 152 patients who had bypass surgery and 92 patients with coronary artery disease who did not have surgical intervention. Patients had memory and other cognitive tests at the beginning of the study period, and after 3, 12, 36 and 72 months. The results showed that there were no significant differences in cognitive scores between the two groups at the beginning of the study. Both groups showed modest decline in cognitive performance during the study period, but there were no significant differences in the degree of decline

between the groups after six years. It was suggested that the decline in both groups was related to the presence of risk factors for vascular disease. [5]

Stroke risk factors may signal faster cognitive decline in elderly

Analysis of the stroke risks of over 17,000 people aged 45 and older (average 65.9) has found that a higher stroke risk score was associated with a significantly higher rate of cognitive decline. The study also identified three specific risk factors significantly associated with memory loss – high systolic blood pressure, diabetes, and left ventricular hypertrophy. [6]

Review supports link between lifestyle factors and cognitive function in older adults

A review of 96 papers involving 36 very large, ongoing epidemiological studies in North America and Europe looking at factors involved in maintaining cognitive and emotional health in adults as they age has concluded that controlling cardiovascular risk factors, such as reducing blood pressure, reducing weight, reducing cholesterol, treating (or preferably avoiding) diabetes, and not smoking, is important for maintaining brain health as we age. The link between hypertension and cognitive decline was the most robust across studies. They also found a consistent close correlation between physical activity and brain health. However, they caution that more research is needed before specific recommendations can be made about which types of exercise and how much exercise are beneficial. They also found protective factors most consistently reported for cognitive health included higher education level, higher socio-economic status, emotional support, better initial performance on cognitive tests, better lung capacity, more physical exercise, moderate alcohol use, and use of vitamin supplements. Psychosocial factors, such as social disengagement and depressed mood, are associated with both poorer cognitive and emotional health in late life. Increased mental activity throughout life, such as learning new things, may also benefit brain health. [7]

Inflammation associated with higher risk of age-related cognitive impairment

So-called “metabolic syndrome” is characterized most obviously by wide girth about the middle (being “apple-shaped”), as well as by high blood pressure and unhealthy levels of cholesterol, triglycerides and glucose in the blood. The syndrome is a well-known risk factor for cardiovascular disease. A new study finds the syndrome is also associated with a greater risk of cognitive impairment (hardly surprising, since many studies now indicate that cardiovascular risk factors are also risk factors for age-related cognitive impairment). The study tracked 2600 people, average age 74 years, over five years. Some 26% of those with the syndrome showed significant cognitive decline, compared to 21% of those without the syndrome. However, it appears the problem is not the syndrome so much as the high levels of inflammation that can result. About 30% of those with the syndrome plus high levels of inflammatory markers in their bloodstream showed significant cognitive decline. Those with the syndrome but no inflammation showed no increased risk. [8]

Cognitive decline after bypass surgery appears more transient than feared

Recent studies have found a high occurrence of cognitive problems in patients who undergo coronary artery bypass surgery, with such problems still found six weeks after surgery. In a new study comparing 140 patients who underwent bypass surgery and a second group of 92 coronary artery disease patients who did not have surgery, no differences in cognitive abilities were found when patients were re-tested at three and 12 months. This supports recent research suggesting that it is the disease itself that is the major problem, rather than the surgery. [9]

Age-related changes in the brain's white matter affect cognitive function

From around age 60, "white-matter lesions" appear in the brain, significantly affecting cognitive function. But without cognitive data from childhood, it is hard to know how much of the difference in cognitive abilities between elderly individuals is due to aging. A longitudinal study has been made possible by the Scottish Mental Survey of 1932, which gave 11-year-olds a validated cognitive test. Scottish researchers have tracked down healthy living men and women who

took part in this Survey and retested 83 participants. Testing took place in 1999, when most participants were 78 years old. It was found that the amount of white-matter lesions made a significant contribution to general cognitive ability differences in old age, independent of prior ability. The amount of white-matter lesions contributed 14.4% of the variance in cognitive scores; early IQ scores contributed 13.7%. The two factors were independent.
Although white-matter lesions are viewed as a normal part of aging, they are linked with other health problems, in particular to circulatory problems (including hypertension, diabetes, heart disease and cardiovascular risk factors). [10]

Cognitive impairment following bypass surgery may last longer than thought

More support for a link between cardiopulmonary bypass surgery and cognitive impairment comes from a new study. In particular, it seems, that attention may be most affected. The study also found evidence of longer-lasting cognitive decline than previously thought. Bypass patients also demonstrated poorer cognitive performance before the surgery, and it is now being suggested that it may be the disease itself that is the major problem, rather than the surgery itself. This is consistent with recent research connecting cardiovascular risk factors with risk factors for cognitive decline. [11]

Cognitive decline after heart bypass

More evidence bypass surgery not responsible for cognitive impairment

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Inflammatory system genes linked to cognitive decline after heart surgery

The finding that people with variants of two genes involved in the inflammatory system appear to be protected from suffering a decline in mental function following heart surgery raises the possibility that therapy involving drugs known to dampen the inflammatory response may be effective in preventing cognitive decline after heart surgery. The specific genes involved were those for C-reactive protein (which plays an important role in the body's initial response to injury) and P-selectin (which helps recruit circulating white blood cells to the site of an injury). Patients with the variation of the C-reactive protein gene were 20.6% less likely to suffer mental decline, and patients with the P-selectin variant had a 15.2% risk reduction. The risk of cognitive decline for those with both gene variants was only 17% compared to 43% for patients who had neither variant. [12]

'Off-pump' CABG surgery appears to have no benefit on cognitive or cardiac outcomes at 5 years

A five-year study of 281 cardiac patients, half of whom received off-pump coronary artery bypass surgery and half on-pump surgery, has found that there was no difference in cognitive performance five years after surgery. The findings suggest that factors other than cardiopulmonary bypass may be responsible for cognitive decline, such as anesthesia and the generalized inflammatory response that is associated with major surgical procedures. [13]

Cognitive loss following coronary artery bypass surgery due to surgical technique?

A surgical strategy designed to minimize trauma to the body's largest artery – the aorta – during heart bypass surgery can significantly reduce cognitive loss that often follows the operation. The study found that at least 60% of patients showed neurological deficits following bypass surgery, but that at 6 months, 57% of patients who had traditional surgery still had deficits while only 32% of those who didn't use the heart-lung machine during surgery, and 30% of those who had the new surgical technique still had deficits. Researchers conclude that surgical technique is the primary cause of cognitive decline following bypass surgery. [14]

Use of heart pump during bypass surgery not implicated in cognitive decline

A study involving 380 individuals has found that those patients undergoing coronary artery bypass grafting (CABG) surgery that used a cardiopulmonary heart pump had no significant differences in their mental functions compared to CABG patients whose surgery did not involve a heart pump. Patients with coronary heart disease all performed lower on cognitive tests than healthy controls, prior to surgery. By three months, both cardiac patients who had undergone surgery (with or without use of a heart pump) and those who had not, had improved cognitive function. [15]

Review finds bypass surgery free of long-term brain effects for most

A broad retrospective review of the effects of coronary artery bypass surgery on cognitive functions concludes that, although the research confirms the existence of mild deficits in the period up to three months after surgery, the procedure itself probably does not cause late or permanent neurological effects. Rather, they argue, the late cognitive declines seen in some long-term studies are for most people likely associated with progression of underlying conditions such as cerebrovascular disease. However, this is not true for all. The exceptions might include older patients and those with risk factors for cerebrovascular disease or a history of stroke. [16]

Elderly experience long-term cognitive decline after surgery

Researchers have found that two years after major non-cardiac surgery, 42% of elderly patients will have experienced a measurable cognitive decline. 59% of patients experienced cognitive decline immediately after surgery — these are the ones at greatest risk of long-term decline. Three months after surgery, 34% of patients had cognitive declines. The study involved 354 patients, with an average age of 69.5 years. [17]

Lower temperatures improve outcomes after bypass surgery

One of the possible adverse effects of cardiac bypass surgery is cognitive decline. Researchers have found that patients who were allowed an additional 10 to 12 minutes to return to normal body temperature after surgery scored almost one-third better on standard tests of cognition six weeks after surgery. (In order to protect the brain and other organs from damage while the heart is stopped during surgery, physicians cool a patient's blood as it passes through a heart-lung machine. However, toward the end of the operation, this blood needs to be rewarmed.) [18]

Cognitive decline after bypass surgery appears more transient than feared

Recent studies have found a high occurrence of cognitive problems in patients who undergo coronary artery bypass surgery, with such problems still found six weeks after surgery. In a new study comparing 140 patients who underwent bypass

surgery and a second group of 92 coronary artery disease patients who did not have surgery, no differences in cognitive abilities were found when patients were re-tested at three and 12 months. This supports recent research suggesting that it is the disease itself that is the major problem, rather than the surgery. [9]

Lowered immunity puts older coronary bypass patients at higher risk for cognitive decline

Older patients with lowered immunity to certain common bacteria found in the gastrointestinal tract are more likely than younger patients to suffer cognitive decline after coronary artery bypass surgery. [19]

Cognitive impairment following bypass surgery may last longer than thought

More support for a link between cardiopulmonary bypass surgery and cognitive impairment comes from a new study. In particular, it seems, that attention may be most affected. The study also found evidence of longer-lasting cognitive decline than previously thought. Bypass patients also demonstrated poorer cognitive performance before the surgery, and it is now being suggested that it may be the disease itself that is the major problem, rather than the surgery itself. This is consistent with recent research connecting cardiovascular risk factors with risk factors for cognitive decline. [11]

Fever immediately after heart bypass surgery associated with cognitive decline

Elevated temperatures within 8-10 hours after surgery are often seen in patients who have undergone coronary bypass surgery. This has not however been regarded as anything other than a nuisance. Many bypass patients also suffer measurable cognitive decline. A new study reports on a relationship between these fevers and cognitive decline six weeks following surgery. Patients who suffered the highest post-operative temperatures also suffered the highest amount of cognitive decline. [20]

More on implications of having the Alzheimer's gene

Researchers have found an association between nerve cell changes associated with aging and the presence of a variation of the apolipoprotein gene known as apolipoprotein E4 (APOE4). This form is carried by approximately 25% of the population and has been linked to increased risk of Alzheimer's disease, cardiovascular disease and memory loss after head injury or bypass surgery. [21]

Frequency of cognitive decline after bypass surgery

Heart bypasses are becoming increasingly common - in the U.S., more than half a million people undergo coronary-artery bypass grafting (CABG) each year. A common side-effect of the procedure is postoperative cognitive decline (frequency of occurrence estimates range from 33% to 82%, depending on the method of evaluation used). A recent study looked at the longer-term picture: in this study, cognitive decline was found in 53% of the patients at time of discharge; at 6 weeks, the rate was assessed at 36%; at 6 months, 24%. However, five years after the surgery the rate of cognitive decline was 42%. Older age, a lower level of education, a higher preoperative score for cognitive function, and the presence of cognitive decline at discharge were all predictors of cognitive decline at 5 years after CABG. Of these, the most significant predictor was a decline in cognition seen at discharge.
Note that there was no control group, so these results must be treated with caution. Note also that short-term declines in cognitive function are also reported in elderly subjects after non-cardiac surgery, and this can persist in a proportion of these patients - in fact, in 10% after 2 years. [22]

High blood pressure

High blood pressure linked to memory problems in middle age

A study involving nearly 20,000 people age 45 and older, of whom nearly half were taking medication for high blood pressure, has found that those with high diastolic blood pressure (the bottom number of a blood pressure reading) were more likely to have cognitive impairment than those with normal diastolic

readings. For every 10 point increase in the reading, the odds of a person having cognitive problems was 7% higher. There was no correlation with systolic blood pressure. The results were adjusted for age, smoking status, exercise level, education, diabetes and high cholesterol. High diastolic blood pressure is known to lead to weakening of small arteries in the brain. [23]

A diet that may reduce age-related cognitive decline

The Dietary Approaches to Stop Hypertension (DASH) diet lowers blood pressure and is often recommended by physicians to people with high blood pressure or pre-hypertension. An 11-year study of over 3800 seniors found that those with higher DASH diet adherence scores had higher cognitive scores at the beginning of the study and increasingly so over time. Four of the nine food-group/nutrient components were independently associated with cognitive scores -- vegetables, whole grains, low-fat dairy, nut/legumes. When a score based on just these four components was used, the difference between those in the highest quintile and those in the lowest was even greater, particularly by the end of the study. [24]

Factors helping you maintain cognitive function in old age

An 8-year study of over 2,500 seniors in their 70s, has found that 53% showed normal age-related decline, 16% showed major cognitive decline, and an encouraging 30% had no change or improved on the tests over the years. The most important factors in determining whether a person maintained their cognitive health was education and literacy: those with a ninth grade literacy level or higher were nearly five times as likely to stay sharp than those with lower literacy levels; those with at least a high school education were nearly three times as likely to stay sharp as those who have less education. Lifestyle factors were also significant: non-smokers were nearly twice as likely to stay sharp as smokers; those who exercised moderately to vigorously at least once a week were 30% more likely to maintain their cognitive function than those who do not exercise that often; people working or volunteering and people who report living with someone were 24% more likely to maintain cognitive function. [25]

High blood pressure may make it difficult for the elderly to think clearly

A study involving 36 community-dwelling elderly (60-87 years old) whose blood pressure and cognitive functioning was monitored for 60 days has found that those with high blood pressure tended to perform more poorly on one of the three cognitive tasks, and this was particularly so when their blood pressure was higher than normal. The finding suggests that high blood pressure impacts on inductive reasoning, and thus the ability to work flexibly with unfamiliar information and find solutions. It also suggests that, for those with high blood pressure, such reasoning will be particularly difficult when they are stressed. [26]

High blood pressure associated with risk for mild cognitive impairment

A study of nearly 1000 older adults (average age 76.3) without mild cognitive impairment at the start of the study found that over the follow-up period (average: 4.7 years), 334 individuals developed mild cognitive impairment, of which 160 were amnestic (reduced memory) and 174 were non-amnestic. Hypertension (high blood pressure) was associated with an increased risk of non-amnestic mild cognitive impairment; but not with amnestic mild cognitive impairment. [27]

Memory tasks require more coordinated brain blood flow for people with high blood pressure

Previous studies have found an association between high blood pressure and cognitive decline in older adults, but the evidence hasn't been entirely consistent. Now a new study helps explain why the situation is not entirely straightforward. It appears that people with high blood pressure required more blood flow to the parts of the brain that support memory function than those with normal blood pressure. Moreover, and surprisingly, it turned out that antihypertensive medication actually made it worse, increasing the inefficiency of the brain's work during memory tasks. [28]

Lowering blood pressure doesn't prevent cognitive impairment, dementia

A review of three large-scale studies of patients with hypertension who were treated with either medication or lifestyle strategies found no convincing evidence

that lowering blood pressure prevents the development of dementia or cognitive impairment in hypertensive patients without apparent prior cerebrovascular disease. However, there is some evidence that midlife hypertension but not late life hypertension is related to cognitive decline; these studies involved patients aged 60 and older. [29]

Review supports link between lifestyle factors and cognitive function in older adults

A review of 96 papers involving 36 very large, ongoing epidemiological studies in North America and Europe looking at factors involved in maintaining cognitive and emotional health in adults as they age has concluded that controlling cardiovascular risk factors, such as reducing blood pressure, reducing weight, reducing cholesterol, treating (or preferably avoiding) diabetes, and not smoking, is important for maintaining brain health as we age. The link between hypertension and cognitive decline was the most robust across studies. They also found a consistent close correlation between physical activity and brain health. However, they caution that more research is needed before specific recommendations can be made about which types of exercise and how much exercise are beneficial. They also found protective factors most consistently reported for cognitive health included higher education level, higher socio-economic status, emotional support, better initial performance on cognitive tests, better lung capacity, more physical exercise, moderate alcohol use, and use of vitamin supplements. Psychosocial factors, such as social disengagement and depressed mood, are associated with both poorer cognitive and emotional health in late life. Increased mental activity throughout life, such as learning new things, may also benefit brain health. [7]

Uncontrolled high blood pressure means more cognitive problems in old age

A study involving a subset of men (average age 67 years) in the VA Normative Aging Study has found that those men with uncontrolled hypertension performed significantly worse on tests of verbal fluency and short-term memory. Those whose hypertension was controlled did as well as those with normal blood pressure. In the United States, hypertension affects 60% of adults age 60 and older, and a high proportion of these are untreated or inadequately treated. [30]

High blood pressure has stronger effect on cognitive function in African-Americans

Analysis of a large longitudinal study (the Maine-Syracuse Longitudinal Study 1976—2002) has found significant associations of high blood pressure to lower cognitive performance in the areas of abstract reasoning, psychomotor skills and visual organization skills. This association, moreover, was significantly greater for African-Americans, although it should be noted that there were only 147 African-Americans among the 1,563 participants. The effect was independent of age. [31]

High blood pressure may be a factor in "senior moments"

An imaging study of seniors (average age 60) found that those with high blood pressure showed reduced blood flow to active brain areas when performing various everyday memory tasks, such as looking up a phone number then walking to another room to pick up the phone and dial the number. The diminished blood flow correlated to slightly worse scores on the memory tests. The differences weren't large, but may help account for "senior moments" - memory problems commonly associated with age. It's estimated that as many as a third of those with high blood pressure are not aware they have it. [32]

Effects of high blood pressure on cognition may have been overstated

Epidemiological studies have suggested hypertensive patients perform worse than individuals with normal blood pressure on cognition tests. A new study has investigated performance on specific cognitive tasks (visual and memory search involving computer displays) by those with high blood pressure who were not on medication and had no detectable cardiovascular disease. Participants ranged in age from 20 to 80. Contrary to expectation, high blood pressure slowed performance only in the middle-aged group (40-59), not in those younger or older. [33]

Treatment to lower blood pressure reduces risk of cognitive decline in stroke patients

High blood pressure and stroke are associated with increased risks of dementia and cognitive impairment. In a study aimed to determine whether blood pressure lowering would reduce the risks of dementia and cognitive decline among individuals with cerebrovascular disease, 6105 people with prior stroke or transient ischemic attack were given either active treatment (perindopril for all participants and indapamide for those with neither an indication for nor a contraindication to a diuretic) or matching placebo(s). Over some 4 years, dementia was found in 6.3% of those given active treatment and 7.1% of those in the placebo group. Cognitive decline occurred in 9.1% of the actively treated group and 11.0% of the placebo group. The researchers concluded that blood pressure lowering with perindopril and indapamide therapy was helpful for those with cerebrovascular disease, in terms of reduced risks of dementia and cognitive decline. [34]

Age-related changes in the brain's white matter affect cognitive function

From around age 60, "white-matter lesions" appear in the brain, significantly affecting cognitive function. But without cognitive data from childhood, it is hard to know how much of the difference in cognitive abilities between elderly individuals is due to aging. A longitudinal study has been made possible by the Scottish Mental Survey of 1932, which gave 11-year-olds a validated cognitive test. Scottish researchers have tracked down healthy living men and women who took part in this Survey and retested 83 participants. Testing took place in 1999, when most participants were 78 years old. It was found that the amount of white-matter lesions made a significant contribution to general cognitive ability differences in old age, independent of prior ability. The amount of white-matter lesions contributed 14.4% of the variance in cognitive scores; early IQ scores contributed 13.7%. The two factors were independent.
Although white-matter lesions are viewed as a normal part of aging, they are linked with other health problems, in particular to circulatory problems (including hypertension, diabetes, heart disease and cardiovascular risk factors). [10]

Sunflower seeds helpful in reducing hypertension and associated cognitive impairment

Research in rats has found that linoleic acid improved not only blood pressure, but also hypertension-induced memory decline, suggesting that the early incorporation of linoleic acid in the diet, may not only help in controlling hypertension, but may also improve hypertension-induced cognitive impairment. Linoleic acid is found in vegetable seed oils, such as safflower, sunflower, and hemp seed. [35]

Diabetes and hypertension associated with greater risk of age-related cognitive decline

A large-scale six-year study of people aged 40 to 70 years old found that people with diabetes and high blood pressure are more likely to experience cognitive decline. Diabetes was associated with greater cognitive decline for those younger than 58 as well as those older than 58, but high blood pressure was a risk factor only for the 58 and older group. [36]

Untreated high blood pressure associated with greater risk of severe cognitive decline

A large-scale study of French people aged 59 to 71 found that, after four years, 21.7% of those with untreated high blood pressure experienced severe cognitive decline. Of those with high blood pressure whose treatment didn't bring the blood pressure down to normal, 12.5% had severe cognitive decline. Of those whose high blood pressure was successfully treated, 7.8% had severe cognitive decline. Only 7.3% of those with normal blood pressure had severe cognitive decline. [37]

Stroke

Different effects of ministrokes & strokes

A study involving 679 seniors (65+) has found that those with small areas of brain damage called white matter hyperintensities, often referred to as ministrokes, were nearly twice as likely to have mild cognitive impairment that included memory loss (amnestic MCI), while those who had infarcts (areas of dead tissue usually

called strokes) were more likely to experience mild cognitive impairment in abilities other than memory loss (non-amnestic MCI). In other words, ministrokes predicted memory problems, while strokes predicted non-memory problems. [38]

Stroke patients regain sight after intensive brain training

In a surprising and exciting finding, stroke victims left partially blind have been trained to use undamaged parts of their brains to improve their vision. The training program, involving an hour a day for at least nine months, forced them to process visual signals with parts of their brain that had not been damaged by the stroke. The seven patients in the study ranged in age from their 30s to 80s, and had suffered a stroke between eight months and three-and-a-half years previously. Impaired vision is a very common result of a stroke. [39]

Social support may protect brain during stroke

A mouse study has found that male mice that lived with a female partner before and after a stroke had a much higher survival rate compared to those mice that lived alone, and also suffered much less brain damage. The findings suggest that high levels of social support may provide some protection against strokes by reducing the amount of damaging inflammation in the brain, and provides some idea of the mechanism. Significantly fewer neurons died in the brains of pair-housed mice. They also had significantly less edema (excess water in the brain), less expression of two genes associated with damaging inflammation in the brain, and significantly higher levels of interleukin-6 (IL-6), a cytokine that has an anti-inflammatory response in the brain. [40]

Daily dose of ginkgo may prevent brain cell damage after a stroke

A study using genetically engineered mice has found that daily doses of ginkgo biloba can prevent or reduce brain damage after an induced stroke. More research is needed before its use in humans can be recommended, but the finding does lend support to other evidence that ginkgo biloba triggers a cascade of events that neutralizes free radicals known to cause cell death. [41]

Psychological distress, not depression, linked to increased risk of stroke

A study following 20,627 people for an average of 8.5 years has found that psychological distress was associated with an increased risk of stroke and that the risk of stroke increased the more distress the participants reported. This association remained the same regardless of cigarette smoking, systolic blood pressure, overall blood cholesterol, obesity, previous heart attack, diabetes, social class, education, high blood pressure treatment, family history of stroke and recent antidepressant medication use. However, there was no increased risk for people who had experienced an episode of major depression in the past year or at any point in their lifetime. [42]

Listening to music improves stroke patients' recovery

A Finnish study involving 60 patients who had suffered a stroke of the left or right hemisphere middle cerebral artery (MCA) has found that if stroke patients listened to music for a couple of hours a day, their verbal memory and focused attention recovered better and they had a more positive mood than patients who did not listen to anything or who listened to audio books. Patients were randomly assigned to a music listening group, a language group or a control group. During the next two months the music and language groups listened daily to music they chose themselves or to audio books respectively, while the control group received no listening material. All groups received standard stroke rehabilitation. Three months after the stroke, verbal memory improved from the first week post-stroke by 60% in music listeners, by 18% in audio book listeners and by 29% in non-listeners. Similarly, focused attention improved by 17% in music listeners, but no improvement was observed in audio book listeners and non-listeners. The differences were essentially the same six months after the stroke. The music listening group also experienced less depressed and confused mood than the patients in the control group. [43]

Not enough 'good' cholesterol makes it harder to recover from stroke

A large study involving men and women over age 35 in the United States, Canada, and Scotland who had suffered a mild to moderate stroke within the past three

months, found several factors predicted memory and disability problems after stroke: increased age, non-Caucasian race, recurrent stroke, diabetes, stroke in the left hemisphere of the brain, higher levels of homocysteine and lower levels of high-density lipoproteins (HDL), otherwise known as "good" cholesterol. "People with low levels of HDL, high levels of homocysteine, and diabetes are twice as likely as those without such problems to have poorer cognitive function and greater disability after stroke … (and) stroke recovery was the most difficult for people over the age of 57 with high levels of homocysteine ..." [44]

Antidepressants improve thinking after a stroke

Executive dysfunction is common after stroke and may impair long-term outcome. A small study of people who had had a stroke during the previous six months has found that, although there was no difference in executive function between those given antidepressants and those given a placebo at the end of the 12-week treatment period, there was a significant difference 21 months after the treatment ended. Those who had been given the placebo showed continued worsening of executive functions, whereas the group treated with antidepressants had clear and significant improvement, regardless of how their depressive symptoms changed. The researchers speculate that antidepressants may foster recovery of neural tissue not directly destroyed by the stroke, yet because the process is slow, it takes months. [45]

Simulator training benefits stroke patients

A study involving 83 stroke patients found that a 5-week 15-hour training program improved driving ability. Those given experimental simulator-based training improved more than those given driving-related cognitive tasks. Those with more education and those with less disability benefited most. 73% of the simulator group were legally allowed to resume driving compared to 42% of the other group. However, there were a large number of dropouts. [46]

Carotid artery stenting improves thought process

Around a quarter of strokes are caused by a narrowing of the carotid arteries. A less invasive technique — carotid artery stenting — is increasingly taking the place of surgery to treat this problem. A study involving 26 patients who had undergone the procedure has tested their cognitive function at least 24 hours before and three months after the stenting procedure. The results showed that cognitive speed increased significantly after stenting, regardless of the patient's age or the severity of the stenosis, and an increase in memory function in patients with decreased blood flow in the brain. [47]

Shift in brain's language-control site offers rehab hope

Language activity in right-handed people is initially localized in the left side of the brain, but a new study shows that this gradually becomes a function shared by both sides. From ages 5 to 25, language activity increases in the dominant hemisphere; from 25 to 67, the nondominant hemisphere increasingly shares the load. The discovery gives new hope for rehabilitation of brain function in adults after stroke or traumatic brain injuries. [48]

Antioxidant-rich diets reduce brain damage from stroke in rats

A new rat study suggests antioxidant-rich fruits and vegetables may limit brain damage from stroke and other neurological disorders. The study built upon previous research showing that diets enriched with blueberries, spinach or spirulina reversed normal age-related declines in memory and learning in old rats, and found that the same diet significantly reduced brain cell loss and improved recovery of movement in rats who had an ischemic stroke induced. The size of the stroke in the rats fed blueberry or spinach supplements was half that seen in the brains of untreated rats. Rats fed spirulina-enriched diets had stroke lesions 75 percent smaller than their untreated counterparts. [49]

Saving the most vulnerable brain cells in stroke

New research reveals why particular neurons in the hippocampus are most vulnerable to death from oxygen starvation during a stroke, and may lead to drugs

that selectively protect those cells, leaving other brain cells unaffected. The findings could also lead to drugs that protect vulnerable brain cells in sufferers of amyotrophic lateral sclerosis, or Lou Gehrig's disease. [50]

Risk for lowered cognitive performance greater in people at high risk for stroke

A new large-scale study supports earlier suggestions that those with a high risk for stroke within 10 years are also at risk for lowered cognitive function and show a pattern of deficits similar to that seen in mild vascular cognitive impairment. It is speculated that the reason may lie in structural and functional changes in the brain that do not rise to the level of clinical detection, and this is supported by a recent brain imaging study showing that abnormal brain atrophy is related both to higher risk of stroke and poorer cognitive ability. The probability of experiencing stroke within 10 years was calculated using weighted combinations of age, systolic blood-pressure, presence of diabetes, cigarette smoking, history of cardiovascular disease, treatment for hypertension and atrial fibrillation. [51]

Chinese herb effective in treating vascular dementia

The herb gastrodine has been used in China for centuries to treat disorders such as dizziness, headache and even ischemic stroke. Now a 12-week, randomized, double-blind trial comparing gastrodine with Duxilâ (a drug used to treat stroke patients in China) has been done in Beijing Dongzhimen Hospital. The trial involved 120 stroke patients who were diagnosed with mild to moderate vascular dementia. Both treatment groups showed similar improvement in memory, orientation, calculation, and language (as measured by the MMSE). The gastrodine group also showed a significant difference in the Blessed Behavioral Scale (BBS) score - including behavior, activities of daily living, and also suffered fewer side effects. Researchers say combined results showed the gastrodine group improvement was 51.43 percent, with 16 of the 70 cases showing much improvement, 20 cases with some improvement, and 34 cases with no change. The improvement rate for patients treated with Duxilâ was 52 percent, with seven of the 50 cases showing much improvement, 19 cases with some improvement, and 24 cases with no change. [52]

Pilot study finds ginseng may improve memory in stroke dementia patients

Following mouse studies showing that ginseng increased the activities of the brain chemicals acetylcholine and choline acetyltransferase, a pilot study of 40 patients (average age 67) with mild to moderate vascular dementia was undertaken by Chinese researchers. 25 patients were randomly selected to receive ginseng extract, while 15 received the drug Duxil® (used to improve memory in elderly dementia patients). Overall, researchers found that patients who took the ginseng compound significantly improved their average memory function after 12 weeks. More research (larger samples, placebo-controls) is needed before this finding can be confirmed. [53]

Right side of brain learns language skills after stroke

Every year, about 750,000 Americans suffer a loss of blood flow to the brain, an ischemic stroke. When the stroke occurs on the left side of the brain, language abilities may be lost (aphasia). However, many of those who initially lose language abilities after a stroke recover much of their ability within six to 12 months. Several studies have suggested that such language recovery occurs because the right hemisphere of the brain takes over language functions. A new imaging study demonstrates that, indeed, areas on the opposite side of the brain to the damaged language areas are active during language tasks, and demonstrate expected patterns of activation with practice. [54]

Alzheimer's gene makes it harder to recover from TBI

People with the E4 type of the apolipoprotein E (APOE) gene (implicated in Alzheimer’s disease) makes it harder for the brain to recover memory functions after traumatic brain injury. [55]

Another Alzheimer's drug helps vascular dementia

Treatment with ARICEPT® (donepezil hydrochloride tablets) significantly improved the cognitive and overall function of patients with vascular dementia (VaD), compared with placebo. VaD, cognitive decline caused by a single, localized stroke, or series of strokes, is second only to AD as a cause of dementia.

Up to one-third of all diagnosed dementia cases are VaD. Of the patients over 65 years old diagnosed with dementia in the United States, approximately 9 to 39 percent have VaD; in Europe, the prevalence of VaD is estimated to be 1.5 to 4.8 percent for people 70 to 80 years of age. [56]

Alzheimer's drug helps vascular dementia

Reminyl (galantamine) may be effective in treating dementia in patients with cerebrovascular disease, such as stroke. Data from a study presented at the XVII World Congress of Neurology show that Reminyl improves memory, orientation and language skills of patients with vascular dementia or a combination of Alzheimer's disease and cerebrovascular disease ("mixed" dementia) for at least 12 months. The results also showed that Reminyl improved or maintained the ability of these individuals to perform normal activities of daily living, such as bathing, dressing and doing housework. However, Reminyl is not yet approved for the treatment of vascular dementia. [57]

Skill-specific exercises better for people who suffer from attention problems following stroke or brain injury

Treatment programs for people who suffer from attention problems following a stroke or other traumatic brain injuries often involve abstract cognitive exercises designed to directly restore impaired attention processes. But a review of 30 studies involving a total of 359 participants shows that an alternative and lesser-used therapy that teaches patients to relearn the tasks that affect their daily lives the most may be more effective. In this specific skills approach, people with brain damage learn to perform attention skills in a way that is different from non-brain-damaged people. In one study, for example, participants whose brain injuries affected their ability to drive a car used small electric cars in the lab to practice specific driving exercises, such as steering between pylons that were moved closer and closer together. Those that practiced specific exercises showed substantial improvement on a variety of driving related tasks compared to those who drove the car, but did not practice the exercises. [58]

Drugs

Common medications associated with cognitive decline in elderly

A study of over 500 relatively healthy men aged 65 years or older with high blood pressure has found that chronic use of medications with anticholinergic properties was associated with impairment in verbal memory and the ability to perform daily living tasks. The degree of impairment increased proportionally to the total amount of drug exposure. This effect was independent of age, education, morbidities, and severity of hypertension. [59]

Using anti-cholinergic drugs may increase cognitive decline

The Religious Orders Study has thrown up more data, this time on the subject of anticholinergic medication. Over an eight year period, 679 of the 870 elderly participants took at least one medication with anticholinergic properties. The study found those people who took anticholinergic drugs saw their rate of cognitive function decline 1.5 times as fast as those people who did not take the drugs. Anticholinergic properties are found in many medicines, such as medicines for stomach cramps, ulcers, motion sickness, and urinary incontinence. [60]

Injection of human umbilical cord blood helps aging brain

A rat study has found that a single intravenous injection of human umbilical cord blood mononuclear cells in aged rats significantly improved the microenvironment of the aged hippocampus and rejuvenated the aged neural stem/progenitor cells. The increase in neurogenesis seemed to be due to a decrease in inflammation. The results raise the possibility of cell therapy to rejuvenate the aged brain. [61]

Relationship between statins and cognitive decline more complex than thought

Previous studies of a link between statins (which protect against cardiovascular disease) and cognitive decline have produced inconsistent results. A three year epidemiological study of older African Americans has now found cognitive decline in statin users was less than those who did not take statins, but those who continued to take statins from 2001 to 2004 had greater cognitive decline than

those who were taking statins in 2001 but were no longer taking them in 2004. The finding that the benefit is stronger for those who had discontinued use than for continuous users points to a complex association between statins and cognitive decline. [62]

Drug reverses aging effect on memory process

Rat studies suggest that a drug made to enhance memory triggers a natural mechanism in the brain that fully reverses age-related memory loss, even after the drug itself has left the body. In middle-aged rats given ampakines twice a day for four days, there was a significant increase in the production of brain-derived neurotrophic factor (BDNF), a protein known to play a key role in memory formation, and in long-term potentiation (LTP), the process by which the connection between the brain cells is enhanced and memory is encoded. Deficits in LTP occur with age. This restoration of LTP was found in the brains even after the ampakines had been cleared from the animals' bodies. [63]

Nicotine patch may alleviate 'senior moments'

A small preliminary clinical trial has found that four weeks of nicotine skin patches helped decision-making and attention in people with age-associated memory impairment (the mildest form of cognitive impairment in seniors). Given the health risks of smoking, and health risks associated with nicotine patches, it is too early to recommend the use of nicotine to improve memory, however. Nicotine mimics the brain chemical acetylcholine, a nerve signal that plays a role in learning and memory. [64]

Statins associated with rare cases of temporary amnesia

Two recent studies have documented cases of amnesia and other nervous-system side effects after taking statins, the cholesterol-lowering drugs being prescribed to millions of people at risk of heart disease. It is emphasized that this is a rare problem, but given the vast numbers of people taking statins, it might still add up to a significant number of problems. [65]

Diabetes

Poor glucose control linked to cognitive impairment in diabetics

The ongoing Memory in Diabetes (MIND) study, involving some 3,000 type 2 diabetics 55 years and older, has found that cognitive functioning abilities drop as average blood sugar levels rise. However, there was no connection between daily blood glucose levels and cognitive performance. The study adds to growing evidence that poorer blood glucose control is strongly associated with poorer memory function, that may eventually lead to mild cognitive impairment, vascular dementia and Alzheimer's disease. It is also possible that people with impaired cognitive ability are less compliant in taking medications and controlling their diabetes. Further research will test the hypothesis that improving glucose control results in improved cognitive function. [66]

Blood sugar linked to normal cognitive aging

Following research showing that decreasing brain function in the area of the hippocampus called the dentate gyrus is a main contributor of normal age-related cognitive decline, an imaging study has been investigating the cause of this decreasing function by looking at measures that typically change during aging, like rising blood sugar, body mass index, cholesterol and insulin levels. The study of 240 community-based nondemented elders (average age 80 years), of whom 60 had type 2 diabetes, found that decreasing activity in the dentate gyrus only correlated with levels of blood glucose. The same association was also found in aging rhesus monkeys and in mice. The finding suggests that maintaining blood sugar levels, even in the absence of diabetes, could help maintain aspects of cognitive health. It also suggests that one reason why physical exercise benefits memory may be its effect on lowering glucose levels. [67]

Diabetic seniors may experience memory declines after eating high-fat food

Growing evidence links diabetes to cognitive impairment. Now a small study of 16 adults (aged 50 years and older) with type 2 diabetes compared their cognitive performance on three separate occasions, fifteen minutes after consuming different

meals. One meal consisted of high fat products – a danish pastry, cheddar cheese and yogurt with added whipped cream; the second meal was only water; and the third was the high-fat meal plus high doses of vitamins C (1000 mg) and E (800 IU) tablets. Researchers found that vitamin supplementation consistently improved recall scores relative to the meal alone, while those who ate the high fat meal without vitamin supplements showed significantly more forgetfulness of words and paragraph information in immediate and time delay recall tests. Those on water meal and meal with vitamins showed similar levels in cognitive performance. The finding indicates not only that diabetics can temporarily further worsen already underlying memory problems associated with the disease by consuming unhealthy meals, but also that this can be remedied by taking high doses of antioxidant vitamins C and E with the meal, suggesting that the effect of high-fat foods si to cause oxidative stress. However, this is hardly a recommended course of action, and the real importance of this finding is that it emphasizes the need for diabetics to consume healthy foods high in antioxidants, like fruits and vegetables. Of course, this is a very small study, and further replication is needed. [68]

Age-related vision problems may be associated with cognitive impairment

Age-related macular degeneration (AMD) develops when the macula, the portion of the eye that allows people to see in detail, deteriorates. An investigation into the relationship between vision problems and cognitive impairment in 2,946 patients has been carried out by The Age-Related Eye Disease Study (AREDS) Research Group. Tests were carried out every year for four years. Those who had more severe AMD had poorer average scores on cognitive tests, an association that remained even after researchers considered other factors, including age, sex, race, education, smoking, diabetes, use of cholesterol-lowering medications and high blood pressure. Average scores also decreased as vision decreased. It's possible that there is a biological reason for the association; it is also possible that visual impairment reduces a person's capacity to develop and maintain relationships and to participate in stimulating activities. [69]

Review supports link between lifestyle factors and cognitive function in older adults

A review of 96 papers involving 36 very large, ongoing epidemiological studies in North America and Europe looking at factors involved in maintaining cognitive and emotional health in adults as they age has concluded that controlling cardiovascular risk factors, such as reducing blood pressure, reducing weight, reducing cholesterol, treating (or preferably avoiding) diabetes, and not smoking, is important for maintaining brain health as we age. The link between hypertension and cognitive decline was the most robust across studies. They also found a consistent close correlation between physical activity and brain health. However, they caution that more research is needed before specific recommendations can be made about which types of exercise and how much exercise are beneficial. They also found protective factors most consistently reported for cognitive health included higher education level, higher socio-economic status, emotional support, better initial performance on cognitive tests, better lung capacity, more physical exercise, moderate alcohol use, and use of vitamin supplements. Psychosocial factors, such as social disengagement and depressed mood, are associated with both poorer cognitive and emotional health in late life. Increased mental activity throughout life, such as learning new things, may also benefit brain health. [30]

Risk for lowered cognitive performance greater in people at high risk for stroke

A new large-scale study supports earlier suggestions that those with a high risk for stroke within 10 years are also at risk for lowered cognitive function and show a pattern of deficits similar to that seen in mild vascular cognitive impairment. It is speculated that the reason may lie in structural and functional changes in the brain that do not rise to the level of clinical detection, and this is supported by a recent brain imaging study showing that abnormal brain atrophy is related both to higher risk of stroke and poorer cognitive ability. The probability of experiencing stroke within 10 years was calculated using weighted combinations of age, systolic blood-pressure, presence of diabetes, cigarette smoking, history of cardiovascular disease, treatment for hypertension and atrial fibrillation. [51]

Age-related changes in the brain's white matter affect cognitive function

From around age 60, "white-matter lesions" appear in the brain, significantly affecting cognitive function. But without cognitive data from childhood, it is hard to know how much of the difference in cognitive abilities between elderly individuals is due to aging. A longitudinal study has been made possible by the Scottish Mental Survey of 1932, which gave 11-year-olds a validated cognitive test. Scottish researchers have tracked down healthy living men and women who took part in this Survey and retested 83 participants. Testing took place in 1999, when most participants were 78 years old.
It was found that the amount of white-matter lesions made a significant contribution to general cognitive ability differences in old age, independent of prior ability. The amount of white-matter lesions contributed 14.4% of the variance in cognitive scores; early IQ scores contributed 13.7%. The two factors were independent.
Although white-matter lesions are viewed as a normal part of aging, they are linked with other health problems, in particular to circulatory problems (including hypertension, diabetes, heart disease and cardiovascular risk factors). [10]

Diabetes and hypertension associated with greater risk of age-related cognitive decline

A large-scale six-year study of people aged 40 to 70 years old found that people with diabetes and high blood pressure are more likely to experience cognitive decline. Diabetes was associated with greater cognitive decline for those younger than 58 as well as those older than 58, but high blood pressure was a risk factor only for the 58 and older group. [36]

Cognitive decline after noncardiac surgery

Older surgical patients at greater risk for developing cognitive problems

There’s been quite a lot of research on the effects of cardiac surgery on cognitive function, but less is known about the effects of any surgery. Now a study of more than 1000 adult patients of different ages has tested memory and cognitive function before undergoing elective non-cardiac surgery, at the time of hospital discharge,

and three months after surgery. It was found that many patients, regardless of age, experienced postoperative cognitive dysfunction (POCD) at the time they left the hospital (36.6% of young adults, 30.4% of the middle-aged, 41.4% of elderly). But three months later, those aged 60 and older were more than twice as likely to exhibit POCD (12.7% compared to less than 6% for both young and middle-aged). POCD was more common among those patients with lower educational level and a history of a stroke that had left no noticeable neurologic impairment. Those with POCD at both the time of hospital discharge and three months after surgery also were more likely to die within the first year after surgery. The reason for this is unclear, but it's speculated that patients with prolonged cognitive dysfunction might be less able to take medicines correctly or may not recognize the need to seek medical care for symptoms of complications. [70]

Depression

Factors helping you maintain cognitive function in old age

An 8-year study of over 2,500 seniors in their 70s, has found that 53% showed normal age-related decline, 16% showed major cognitive decline, and an encouraging 30% had no change or improved on the tests over the years. The most important factors in determining whether a person maintained their cognitive health was education and literacy: those with a ninth grade literacy level or higher were nearly five times as likely to stay sharp than those with lower literacy levels; those with at least a high school education were nearly three times as likely to stay sharp as those who have less education. Lifestyle factors were also significant: non-smokers were nearly twice as likely to stay sharp as smokers; those who exercised moderately to vigorously at least once a week were 30% more likely to maintain their cognitive function than those who do not exercise that often; people working or volunteering and people who report living with someone were 24% more likely to maintain cognitive function. [71]

Depression increases risk of executive dysfunction in older people

A two-year study of more than 700 older adults (65 and older) has found that depression increased the risk of declining executive function (high-level mental

processes, such a making decisions, organizing, planning and doing a series of things in sequence). [72]

Depressed older adults more likely to become cognitively impaired

A study involving 2,220 participants in the Cardiovascular Health Study, a longitudinal prospective study of adults 65 and older, has found that 19.7% of subjects with moderate to high depression developed mild cognitive impairment within six years, compared to 10% of subjects with no depressive symptoms and 13.3% of subjects with low depressive symptoms. There was no correlation between depression and vascular disease, although it has been hypothesized that vascular disease might lead to both depression and cognitive impairment by causing inadequate blood flow to different brain structures. [73]

Treatable depression often accompanies mild memory loss

A large-scale study of older adults begun in 1989 has revealed that 43% of those with mild cognitive impairment had psychiatric symptoms (such as depression, irritability, loss of interest in activities, or changes in sleep or appetite) in the month before examination. Such symptoms are often shrugged off as emotional reactions to memory decline, but they may be due to changes in brain function, and may respond to treatment. [74]

vision and hearing loss

Age-related eye disease associated with cognitive impairment

Age-related macular degeneration (AMD) is the leading cause of visual impairment in industrialized nations, and like Alzheimer's disease, involves the buildup of beta-amyloid peptides in the brain, as well as sharing similar vascular risk factors. A study of over 2000 older adults (69-97) has revealed an association between early-stage AMD and cognitive impairment, as assessed by the Digit Symbol Substitution Test (a test of attention and processing speed). There was no association with performance on the Modified Mini-Mental State Examination (used to assess dementia).
It’s worth noting that in the same journal two studies into the association between

dietary fat intake and AMD appeared. The first, four-year, study involved over 6700 older adults and found that higher trans-unsaturated fat intake was associated with a higher incidence of AMD, while higher omega-3 fatty acid and higher olive oil intake were each associated with a lower incidence. The second, ten-year, study involving nearly 2500 older adults, found regular consumption of fish, greater intake of omega-3 fatty acids, and low intake of linoleic acid (perhaps because a higher intake implies a lower intake of omega-3 oils? linoleic acid is an omega-6 fatty acid), were all associated with a lower incidence of AMD. Fish and omega-3 oils have of course been similarly associated with lower rates of dementia and age-related cognitive impairment. [75]

Memory impairment associated with sound processing disorder

Central auditory processing dysfunction refers to the situation where hearing in quiet settings is normal or near normal but is substantially impaired in the presence of competing noise or in other difficult listening situations. Such a problem is not helped by amplification and requires alternative rehabilitation strategies. Central auditory processing has been found to be impaired in those with dementia. Now a study comparing individuals with dementia, those with mild memory impairment but without a dementia diagnosis, and those without memory loss, has found that scores on central auditory processing tests were significantly lower in both the group with dementia and in the group with mild memory impairment, compared to controls. [76]

Age-related vision problems may be associated with cognitive impairment

Age-related macular degeneration (AMD) develops when the macula, the portion of the eye that allows people to see in detail, deteriorates. An investigation into the relationship between vision problems and cognitive impairment in 2,946 patients has been carried out by The Age-Related Eye Disease Study (AREDS) Research Group. Tests were carried out every year for four years. Those who had more severe AMD had poorer average scores on cognitive tests, an association that remained even after researchers considered other factors, including age, sex, race, education, smoking, diabetes, use of cholesterol-lowering medications and high blood pressure. Average scores also decreased as vision decreased. It's possible

that there is a biological reason for the association; it is also possible that visual impairment reduces a person's capacity to develop and maintain relationships and to participate in stimulating activities. [69]

Hearing loss in older adults may compromise cognitive resources for memory

A study involving older adults with good hearing and a group with mild-to-moderate hearing loss has found that even when older adults could hear words well enough to repeat them, their ability to memorize and remember these words was poorer in comparison to other individuals of the same age with good hearing. The researchers suggest that the effect of expending extra effort comprehending words means there are fewer cognitive resources for higher level comprehension. Working memory capacity tends to diminish as we age. [77]

Other

Overweight and obese elderly have smaller brains

Analysis of brain scans from 94 people in their 70s who were still "cognitively normal" five years after the scan has revealed that people with higher body mass indexes had smaller brains on average, with the frontal and temporal lobes particularly affected (specifically, in the frontal lobes, anterior cingulate gyrus, hippocampus, and thalamus, in obese people, and in the basal ganglia and corona radiate of the overweight). The brains of the 51 overweight people were, on average, 6% smaller than those of the normal-weight participants, and those of the 14 obese people were 8% smaller. To put it in more comprehensible, and dramatic terms: "The brains of overweight people looked eight years older than the brains of those who were lean, and 16 years older in obese people." However, overall brain volume did not differ between overweight and obese persons. As yet unpublished research by the same researchers indicates that exercise protects these same brain regions: "The most strenuous kind of exercise can save about the same amount of brain tissue that is lost in the obese." [78]

Difficulty identifying odors may predict cognitive decline

Older adults who have difficulty identifying common odors may have a greater risk of developing mild cognitive impairment, increasingly recognized as a precursor to Alzheimer's disease. A study of nearly 600 older adults (average age 79.9) found that 30.1% developed mild cognitive impairment over the five-year period of the study. Risk of developing mild cognitive impairment was greater for those who scored worse on an odor identification test given at the start of the study. For example, those who scored below average (eight) were 50% more likely to develop MCI than those who scored above average (11). This association did not change when stroke, smoking habits or other factors that might influence smell or cognitive ability were considered. Impaired odor identification was also associated with lower cognitive scores at the beginning of the study and with a more rapid decline in episodic memory (memory of past experiences), semantic memory (memory of words and symbols) and perceptual speed. The odor test involved identifying 12 familiar odors given four possible alternatives to choose from. [79]

Memory problems and sleep disturbance linked in older women

A large long-running study, involving older women (average age 69) found that the nearly 25% of women who experienced cognitive decline over the 15 year period were twice as likely as women without memory problems to experience sleep disturbances, specifically problems staying asleep, and also problems falling asleep and being awake for more than 90 minutes during their sleep cycle. Women who declined on one of the two cognitive tests were also nearly twice as likely to nap more than two hours a day. However, cognitive decline was not associated with total sleep time. The association between sleep disturbances and poor cognitive function is of course well-known, but these findings raise the possibility that cognitive decline may increase the risk of sleep problems, rather than vice versa. [80]

High-normal uric acid linked with mild cognitive impairment in the elderly

A study of 96 older adults has found that those with uric-acid levels at the high end of the normal range had the lowest scores on tests of mental processing speed, verbal memory and working memory. The correlation persisted even when controlled for age, sex, weight, race, education, diabetes, hypertension, smoking and alcohol abuse. Uric acid levels increase with age, and higher levels are linked with high blood pressure, atherosclerosis, Type 2 diabetes and the "metabolic syndrome" of abdominal obesity and insulin resistance — all known risk factors for dementia. Because uric acid levels are so easily tested, the finding may suggest a valuable biological marker for very early cognitive problems in old age. [81]

References

Mild Cognitive Impairment

Except in the cases of stroke or traumatic brain injury, loss of cognitive function is not something that happens all at once. Cognitive impairment that comes with age may be thought of as belonging on a continuum, with one end being no cognitive impairment and the other end being dementia, of which Alzheimer's is the most common type.

Most older adults are actually at the "no impairment" end of the continuum. Some 30-40% of adults over 65 will have what is called "age-related memory impairment", which is the type of cognitive loss we regard as a normal consequence of age -- a measurable (but slight) decline on memory tests; a feeling that you're not quite as sharp or as good at remembering, as you used to be.

Only about 1% of these people will develop Alzheimer's.

But around 10% of adults over 65 develop "mild cognitive impairment", and this *is* a precursor of Alzheimer's. This doesn't mean someone with MCI will inevitably get Alzheimer's in their lifetime, but their likelihood of doing so is substantially increased.

Mild cognitive impairment is not necessarily obvious to outside observers. A person with it can function perfectly well, and although they may feel their impairment is obvious to all around them, it's not likely to be obvious to anyone not living with them.

A person suffering from mild cognitive impairment may find that they have problems with:

- finding the right words
- making decisions
- remembering recent events
- placing things in space (for example, getting the proportions right when drawing a simple object such as a box).

Essentially, age-related cognitive impairment might be thought of as slight, non-important, cognitive impairment, while mild cognitive impairment is a condition where significant cognitive impairment exists which nevertheless doesn't affect daily functioning. Dementia is significant cognitive impairment that does interfere with daily life.

Prevalence of MCI

A study of nearly 4000 people from the general population of a Minnesota county is currently being run by the Mayo Clinic to find the prevalence of mild cognitive impairment. A report on the findings so far suggests 9% of those aged 70 to 79 and nearly 18% of those 80 to 89 have MCI. Prevalence varied not only with age but also years of education: 25% in those with up to eight years of education, 14% in those with nine to 12 years, 9% in those with 13 to 16 years, and 8.5% in those with greater than 16 years. [1]

How likely is someone with MCI to progress to Alzheimer's?

The big question for those with MCI is how likely are they to progress to Alzheimer's.

Mild cognitive impairment has now been categorized into two sub-types: those with the amnesic subtype (MCI-A) have memory impairments only, while those with the multiple cognitive domain subtype (MCI-MCD) have other types of mild impairments, such as in judgment or language, and mild or no memory loss. Both sub-types progress to Alzheimer's disease at the same rate, but they do have different pathologies in the brain. For example, shrinking of the hippocampus has been identified as predicting whether those with MCI will progress to Alzheimer's (whose hippocampi show substantial atrophy), but it turns out that this is true only of the MCI-A sub-type. [2]

Behavioral signs

A recent study involving 111 older adults with MCI found only one factor predicted whether the impairment would progress to dementia within the next 2 ½ years (which it did for a quarter of them): the degree of functional impairment (ability to perform routine activities) at the beginning of the study. Other cognitive and neurological variables were not predictive. [3]
Consistent with this, another study found that the 29% of 87 older people with amnestic mild cognitive impairment who developed Alzheimer's within a year had been significantly worse at a money management task than had those who hadn't developed Alzheimer's. The task included counting coins, making grocery purchases, understanding and using a checkbook, understanding and using a bank statement, preparing bills for mailing, and detecting fraud situations. [4]
The studies both point to everyday tasks being a good clue to dementia progression.

Senior's memory complaints should be taken seriously

A study involving 120 people over 60 found those who complained of significant memory problems who still performed normally on memory tests had a 3% reduction in gray matter density in their brains. This compares to 4% in those diagnosed with mild cognitive impairment. This suggests that significant memory loss complaints may indicate a very early "pre-MCI" stage of dementia for some people. [5]

Effective new cognitive screening test for detection of Alzheimer's

A new cognitive test for detecting Alzheimer's has been developed, and designed to be suitable for non-specialist use. The TYM ("test your memory") involves 10 tasks including ability to copy a sentence, semantic knowledge, calculation, verbal fluency and recall ability. It has been tested on 540 healthy individuals and139 patients with diagnosed Alzheimer's or mild cognitive impairment. Healthy controls completed the test in an average time of five minutes and gained an average score of 47 out of 50, compared to 45 for those with mild cognitive impairment, 39 for those with non-Alzheimer dementias and 33 for those with Alzheimer's. Among controls, the average score was not affected by age until after 70, when it showed a small decline. There were no gender or geographical background differences in performance. The TYM detected 93% of patients with Alzheimer's, compared to only 52% by the widely used mini-mental state examination. [6]

New 'everyday cognition' scale tracks how older adults function in daily life

A new, carefully validated questionnaire called Everyday Cognition (ECog) has been developed by seven psychologists. The 39-question screening tool is designed to enable mild functional problems in older adults to be quickly and easily identified. The questionnaire needs to be filled out by someone who knows an older adult well, such as a spouse, adult child, or close friend. It looks at everyday function in seven key cognitive domains: memory, language, semantic (factual) knowledge, visuospatial abilities, planning, organization and divided attention. The test has been shown to be sensitive to early changes present in Mild Cognitive Impairment, and unlike other cognitive tests, does not appear to be strongly influenced by education level. The test even differentiated between people diagnosed with mild impairment in memory only and those mildly impaired in several areas. [7]

More sensitive test norms better predict who might develop Alzheimer's disease

Early diagnosis of Alzheimer's is becoming more important with new medical and psychological interventions that can slow (but not stop) the course of the disease. Given this, it is suggested that more sensitive testing may be necessary for highly intelligent people, who, on average, show clinical signs of Alzheimer's later than the general population. Once they show such signs, they decline much faster. A study of 42 older people with IQ's of 120 or more, used two different test norms to forecast problems: the standard norm, derived from a large cross-section of the population, or an adjusted high-IQ norm that measured changes against the individual's higher ability level. The raised cutoffs predicted that 11 of the 42 individuals were at risk for future decline – compared with standard cutoffs, which indicated they were normal. True to the former prediction, three and a half years later, nine of those 11 people had declined. Six of those went on to develop mild cognitive impairment (MCI), a transitional illness from normal aging to a dementia (of which one type is Alzheimer's). Five of these individuals have since received a diagnosis of Alzheimer's disease, two years after this study was submitted. It is also suggested that, at the other end of the scale, those with below-average intelligence have the potential for being misdiagnosed as 'demented' when they are not, and the norms should be adjusted downwards accordingly. [8]

Researchers have developed a brief telephonic questionnaire that helps distinguish between persons with early signs of dementia and persons with normal cognitive function. The questionnaire provides a way to reach out to persons with dementia whose impairment otherwise may go undetected until substantial cognitive deterioration has occurred. The questionnaire consists of a test of delayed recall and 2 questions that ask whether the person needs help with remembering to take medications or with planning a trip for errands. It is estimated that of 100 people who score positive on this test, 42 will actually have cognitive impairment. In other words, this does not provide a diagnosis of Alzheimer’s, but provides evidence that further evaluation is required. The rate of false positives compares favorably to other types of screening tests. A further study is underway to confirm the validity and reliability of the test. [9]

An analysis of data from 40 participants enrolled in a long-term study at the UCSD Alzheimer's Disease Research Center (ADRC) found that "paper-and-pencil" cognitive skills tests administered to normal subjects averaging 75 years of age contained early signs of cognitive decline in those subjects who later developed Alzheimer's disease. All participants were symptom-free when they took the test. The differences were quite subtle - only some performance measures were affected. [10]

Difficulty identifying odors may predict cognitive decline

Older adults who have difficulty identifying common odors may have a greater risk of developing mild cognitive impairment, increasingly recognized as a precursor to Alzheimer's disease. A study of nearly 600 older adults (average age 79.9) found that 30.1% developed mild cognitive impairment over the five-year period of the study. Risk of developing mild cognitive impairment was greater for those who scored worse on an odor identification test given at the start of the study. For example, those who scored below average (eight) were 50% more likely to develop MCI than those who scored above average (11). This association did not change when stroke, smoking habits or other factors that might influence smell or cognitive ability were considered. Impaired odor identification was also associated with lower cognitive scores at the beginning of the study and with a more rapid decline in episodic memory (memory of past experiences), semantic memory (memory of words and symbols) and perceptual speed. The odor test involved identifying 12 familiar odors given four possible alternatives to choose from. [11]

Clinical signs

Brain atrophy

A study of 55 people succeeded in predicting which patients with mild cognitive impairment would decline to Alzheimer's with 75% accuracy, on the basis of rate of hippocampal atrophy and rate of total brain volume loss. [12]

A fully automated procedure called Volumetric MRI (that can be done in a clinical setting) has recently been shown to accurately and quickly measure parts of the medial temporal lobe and compare them to expected size. It also found that not only atrophy in the hippocampus but also the amygdala is associated with a greater risk of conversion to Alzheimer's. [13]

An imaging study of 64 Alzheimer's patients, 44 people with mild cognitive impairment, and 34 people with no memory or thinking problems, has found that those with smaller hippocampal volumes and higher rates of shrinkage were two to four times as likely to develop dementia over the study period (average 18 months) as those with larger volumes and a slower rate of atrophy. During that time, 23 of the people with MCI developed Alzheimer's, and three of the healthy participants. [14]

A study of 84 patients with mild Alzheimer's, 175 patients with MCI and 139 healthy controls has revealed a pattern of regional brain atrophy in patients with MCI that indicates a greater likelihood of progression to Alzheimer's. Brain scans results showed widespread cortical atrophy in some patients with MCI, most importantly, atrophy in parts of the medial and lateral temporal lobes and in the frontal lobes — a pattern also present in the patients with mild Alzheimer's disease. Those exhibiting such atrophy declined significantly over a year and were more likely to progress to a probable diagnosis of Alzheimer's. MCI patients without that pattern of atrophy remained stable after a year. It should be noted that such atrophy affects not only memory, but also planning, organization, problem solving and language. [15]

A study of 503 seniors (aged 50-85) with no dementia found that 453 of them (90%) reported having occasional memory problems such as having trouble thinking of the right word or forgetting things that happened in the last day or two, or thinking problems such as having trouble concentrating or thinking more slowly than they used to. Such problems have been attributed to white matter lesions, which are very common in older adults, but all of the participants in the study had white matter lesions in their brains, and the amount of lesions was not tied to occasional memory problems. However it was found that those who reported

having such problems had a smaller hippocampus than those who had no cognitive problems. This was most noteworthy in subjects with good objective cognitive performance. [16]

A study of 20 older adults with mild cognitive impairment has found that the hippocampus was smaller in those who developed into Alzheimer's during the 3 year period. [17]

New computer program may enable early prediction of Alzheimer's risk

Researchers have developed a brain scan-based computer program that quickly and accurately measures metabolic activity in the hippocampus, a key brain region that shrinks with the development of Alzheimer's. The study followed 53 normal subjects aged 54 to 80 for at least 9 years and in some cases for as long as 24 years, and found that hippocampal glucose metabolism was significantly reduced on the first scan of those 25 individuals who would later experience cognitive decline related to either mild cognitive impairment or to Alzheimer's. The findings bring hope of being able to predict who will develop Alzheimer's at least 9 years ahead of symptoms. [18]

Biomarkers

A study involving 60 patients with subjective cognitive impairment, 37 patients with non-amnestic mild cognitive impairment, and 71 with amnestic mild cognitive impairment, has found that 52% of those with SCI, 68% of those with naMCI, and 79% of those with aMCI showed decreased concentrations of Aβ42 and increased concentrations of tau protein in the cerebrospinal fluid. The findings confirm the use of biomarkers in the CSF for very early diagnosis. [19]

Cerebrospinal fluid samples from 410 volunteers (100 with mild Alzheimer's; 196 with MCI; 114 cognitively normal older adults) has revealed that concentrations of amyloid beta-42 peptide and tau protein successfully assessed brain status and predicted development. The test diagnosed Alzheimer's with 96% accuracy; ruled out Alzheimer's with 95% accuracy; and predicted the conversion from MCI to Alzheimer's with 82% accuracy. [20]

Technique shows brain aging before symptoms appear

A new chemical marker called FDDNP, which binds to plaque and tangle deposits in the brain, has enabled PET scans to reveal exactly where these abnormal protein deposits are accumulating, and has found that older age correlated with higher concentrations of FDDNP in the medial and lateral temporal regions of the brain, areas involved with memory, where plaques and tangles usually collect. Of the 76 study volunteers, 34 carried the 'Alzheimer's gene'. This group demonstrated higher FDDNP levels in the frontal region of the brain than those without the gene variant. Thirty-six of the volunteers had mild cognitive impairment, and these had higher measures of FDDNP in the medial temporal brain regions than normal volunteers. Those who had both MCI and the APOE-4 gene also had higher concentrations of FDDNP in the medial temporal brain regions than those who had MCI but not APOE-4. The pilot study offers hope of early diagnosis of brain impairment, before symptoms show themselves. [21]

Eye tracking test detects mild cognitive impairment

A test first developed for use with nonhuman primates is now being used to detect mild cognitive impairment (MC (MCI) in humans. The infrared eye-tracking test involves showing one image and then another after a 2-second delay, and then repeating the test 2 minutes later. Those without cognitive impairment spend most of their time looking at the new image, but it was found that those with MCI spent less time looking at the new picture, presumably because they have less memory of seeing the original image before. Those with Alzheimer's disease look at both images equally. It's hoped that this test may allow dementia to be spotted much earlier. [22]

Post-mortem brain studies reveal features of mild cognitive impairment

Autopsies have revealed that the brains of patients with mild cognitive impairment display pathologic features that appear to place them at an intermediate stage between normal aging and Alzheimer's disease. For instance, the patients had begun developing neurofibrillary tangles, but the number of plaques was similar to that in healthy patients. All patients with mild cognitive impairment had

abnormalities in their temporal lobes, which likely caused their cognitive difficulties, and many also had abnormalities in other areas that did not relate to the features of Alzheimer's disease. In a second study, of 34 patients with mild cognitive impairment who had progressed to clinical dementia before their deaths, 24 were diagnosed (post-mortem) with Alzheimer's, and 10 with other types of dementia. As in the other study, all patients had abnormalities in their temporal lobes. [23]

Brain scans predict cognitive impairment

A three-year study of 48 healthy people from 60 to 80 years old, by New York University School of Medicine researchers, predicted which healthy elderly men and women would develop memory impairment based on scans of their brains. At the beginning of the study, everyone scored within the normal range on a battery of tests typically used to detect early loss of memory and other mental skills. However, PET scans revealed a reduction in glucose metabolism in an area of the brain called the entorhinal cortex among 12 people. Three years later, 11 of these people had experienced mild cognitive impairment and one had developed Alzheimer's disease. "Our work extends the use of PET scanning to identifying in normal aging subjects the earliest metabolic abnormalities that may lead to the memory losses referred to as mild cognitive impairment (MCI). The diagnosis of MCI carries a high risk for future Alzheimer's disease." [24]

What factors affect whether MCI will progress to Alzheimer's?

There's evidence that ministrokes (small areas of brain damage called white matter hyperintensities) are more likely to lead to memory problems than strokes. [25] Consistent with this, MCI is more likely in those with quickly growing white matter lesions; importantly, the crucial factor appears to be the rate of growth, not the amount of lesions (all brains develop a certain amount of white matter lesions as they age). [26]

Confirming earlier indications, autopsies of the brains of 38 Catholic nuns in the Nun Study has found that those who had no memory problems, whether or not their brains showed Alzheimer's disease hallmarks, had higher early language

scores compared to those who showed symptoms of Alzheimer's or mild cognitive impairment. Early language was assessed in terms of the number of ideas produced every 10 words in the essays they wrote in their late teens or early 20s when they entered the Order. There was no effect in terms of grammatical complexity. Those with Alzheimer's disease hallmarks and no memory problems also had enlarged neurons in the CA1 region of the hippocampus. [27]

Depression may increase risk of Alzheimer's disease in people with memory problems

A three-year study involving 756 people with mild cognitive impairment found increases in depressive symptoms was positively associated with increased risk in developing Alzheimer's. The study also found that, for those who were depressed, taking the Alzheimer's drug donepezil significantly reduced the risk of developing Alzheimer's, compared to those taking vitamin E or placebo. Donepezil had little effect on those who were not depressed. [28]

Less risk of developing dementia than thought

Data from 41 studies has revealed the risk of those with mild cognitive impairment developing dementia is much less than thought. MCI is found in about 1 in 6 people seen in general practice, and it was thought that the risk of developing dementia was up to 15% per year, making deterioration almost inevitable within 5 to 10 years. It now appears that the risk is 10% per year in high risk groups (9.6% for dementia overall; 8% for Alzheimers; 2% for vascular dementia) and only 5% per year in low risk groups (5% for dementia overall; 7% for Alzheimers; 1.6% for vascular dementia). More importantly, only 20-40% developed dementia even after 10 years and the risk appeared to reduce slightly with time. [29]

Mild cognitive impairment more likely in men

A study involving over 2000 people between 70 and 89 years old, found 15% had mild cognitive impairment, and men were one-and-a-half times more likely to have MCI than women. [30]

High blood pressure associated with risk for mild cognitive impairment

A study of nearly 1000 older adults (average age 76.3) without mild cognitive impairment at the start of the study found that over the follow-up period (average: 4.7 years), 334 individuals developed mild cognitive impairment, of which 160 were amnestic (reduced memory) and 174 were non-amnestic. Hypertension (high blood pressure) was associated with an increased risk of non-amnestic mild cognitive impairment; but not with amnestic mild cognitive impairment. [31]

Risk of mild cognitive impairment increases with less education

A study of 3,957 people from the general population of Olmsted County, Minnesota is currently in train to find how many of those who did not have dementia might have mild cognitive impairment. A report on the findings so far suggests 9% of those aged 70 to 79 and nearly 18% of those 80 to 89 have MCI. Prevalence varied not only with age but also years of education: 25% in those with up to eight years of education, 14% in those with nine to 12 years, 9% in those with 13 to 16 years, and 8.5% in those with greater than 16 years. [32]

Concussions increase chance of age-related cognitive impairment

A study involving retired National Football League players found that they had a 37% higher risk of Alzheimer's than other U.S. males of the same age. Some 60.8% of the retired players reported having sustained at least one concussion during their professional playing career, and 24% reported sustaining three or more concussions. Those with three or more concussions had a five-fold greater chance of having been diagnosed with mild cognitive impairment and a three-fold prevalence of reported significant memory problems compared to those players without a history of concussion. As the study was based on self-reported answers to the health questions, further studies are needed to confirm the findings, but it does seem likely that head injuries earlier in life increase the chance of developing dementia or mild cognitive impairment. [33]

Alzheimer's, cardiovascular disease share risk factors

A study of over 700 elderly men and women has investigated the relationship between diabetes and cardiovascular disease risk factors and cognitive health. Researchers found that the presence of coronary heart disease, high cholesterol, or hypertension increased the risk of later cognitive decline, with a particularly strong effect in African Americans. They also found that although the diagnosis of diabetes increased the risk of cognitive decline by as much as two fold, the presence of high levels of fasting glucose (therefore, low insulin levels) substantially decreased the risk of cognitive decline in diabetic patients.

Data from a 24-week pilot trial of the diabetes drug rosiglitazone in patients with mild cognitive impairment (MCI) or very mild Alzheimer's disease found those who received rosiglitazone – a drug that boosts insulin activity – did not decline in their performance on memory and attention tests. [34]

Minorities hardest hit by Alzheimer's disease

A study of 119 Latinos and 55 non-Latino white Alzheimer patients suggests that Latinos in the U.S. develop Alzheimer's symptoms much earlier than their white, non-Latino peers. There are several known factors which may be responsible for this apparent vulnerability in Latinos: high rates of vascular disease, leave school earlier, and less likely to use medical services or have health insurance than other Americans.

South Carolina, as the only U.S. state that keeps a comprehensive database of people with a diagnosis of Alzheimer's disease, has found that African Americans aged 55 to 64 years were more than three times as likely to have Alzheimer's as their European American counterparts. At ages 65 to 84, African Americans were more than twice as likely to have Alzheimer's. South Carolina has greater rates of obesity, diabetes, and related health problems than the rest of the country, especially amongst African Americans.

Another study has found that, in order to avoid overestimating the number of African Americans who may have early signs of Alzheimer's disease, screening

tests must be adapted to cultural differences. The study involved 635 people over the age of 60. Researchers found that, using current scoring methods, African Americans scored lower on various neuropsychological tests. Even when education was taken into account, 35% of African Americans scored low enough to warrant a diagnosis of MCI, compared to only 15% of European Americans. However, when the researchers applied new, racially sensitive scoring methods they've developed, the difference in MCI rates disappeared. [35]

Is there anything I can do to prevent MCI from progressing to Alzheimer's?

Mediterranean diet associated with lower risk of cognitive impairment

A study of 1,393 individuals with no cognitive problems and 482 patients with mild cognitive impairment has found that eating a Mediterranean diet was associated with less risk of developing mild cognitive impairment or of transitioning from MCI to Alzheimer's disease. Over an average of 4.5 years of follow-up, 275 of the 1,393 developed MCI. The third with the highest scores for Mediterranean diet adherence had a 28% lower risk of developing MCI compared to the third with the lowest scores, while the middle third had 17% less risk. Among the 482 with MCI, 106 developed Alzheimer's disease over an average 4.3 years of follow-up. The third with the highest scores for Mediterranean diet adherence had 48% less chance of developing Alzheimer’s and those in the middle third had 45% less chance. A number of the components of the Mediterranean diet have been associated with reduced risk of developing MCI or Alzheimer’s. [36]

Education protects against pre-Alzheimer's memory loss

Another study has come out supporting the view that people with more education and more mentally demanding occupations may have protection against the memory loss that precedes Alzheimer's disease, providing more evidence for the idea of cognitive reserve. The 14-month study followed 242 people with Alzheimer's disease, 72 people with mild cognitive impairment, and 144 people with no memory problems. During the study period, 21 of the people with MCI developed Alzheimer's. The metabolic changes in those with MCI who developed

Alzheimer's indicate the cognitive reserve is already in play in the pre-dementia stage. [37]

Moderate exercise helps mild cognitive impairment

An Australian study involving 138 older adults (50 years and over) with mild cognitive impairment, has found that those who undertook to achieve 2 ½ hours of physical activity each week (three 50 minute sessions), ranging from walking, ballroom dancing to swimming, for a six month period, continually out-scored the control group on cognitive tests during the 18 month testing period — showing that memory improvement was still evident a year after the supervised exercise period. [38]

Helping those with MCI

Pictures better than words for memory-damaged patients

We've long known that pictures are remembered better than words. Now a study has found that this picture superiority still exists in those with mild cognitive impairment and very mild Alzheimer's disease. Moreover, frontally-based brainwave patterns were similar to those of controls when pictures were being retrieved, but not for words. The findings support the idea that those with mild Alzheimer's can successfully use implicit memory (memory without conscious awareness) to support recognition, and this may point to new strategies for dealing with their memory problems. [39]

Donepezil may have short-term benefit for mild cognitive impairment

Preliminary data from a recently completed clinical trial of 769 patients with mild cognitive impairment indicates that those taking the drug donepezil were at reduced risk of progressing to Alzheimer's disease for 18 months. The reduced risk disappeared after 18 months, and by the end of the 3-year study, the probability of progressing to Alzheimer's was the same in the two groups. The study compared donepezil, vitamin E, or placebo. There was no apparent benefit from vitamin E. [40]

References

Other dementias

Prevalence of dementia

Dementia is estimated[1] to afflict over 35.5 million people worldwide -- this includes nearly 10 million people in Europe, nearly 4.4 million in North America, nearly 7 million in South and Southeast Asia, about 5.5 million in China and East Asia and about 3 million in Latin America.

The estimated prevalence for over 60s is 4.7% worldwide. Because this is a disorder of age, prevalence is of course greatly affected by the proportion of people reaching their senior years. Hence the prevalence is higher in the more developed countries: the estimated prevalence in Western Europe and North America is 7.2% and 6.9% respectively, compared to 2.6% in Africa.

What kinds of dementia are most common?

The prevalence of the various dementia types is a complicated story. Certainly Alzheimer's disease is by far the most common type of dementia, accounting for perhaps 70% of all dementias (although a 2006 study[13] suggested that non-Alzheimer dementias were as common as Alzheimer's -- however this was based on dementia among military veterans). The second most common dementia is almost certainly vascular dementia, which may account for some 17% of dementias. However, the actual numbers are made uncertain by the fact that these two dementias often occur together.

At minimum, around a quarter of Alzheimer's cases have been found, on autopsy, to also have vascular pathology; this proportion reaches higher levels when the samples are not restricted to dementia clinics. One such community-based study[2], for example, found 45% of the Alzheimer's cases also showed significant vascular pathology. Another, U.K., study[3] found a similar proportion (46%).

Another, large long-running, study[14] has found that only 30% of people with signs of dementia had Alzheimer's disease alone. 42% had Alzheimer's disease with cerebral infarcts (strokes) and 16% had Alzheimer's disease with Parkinson's disease (including two people with all three conditions). Infarcts alone caused another 12% of the cases. Vascular dementia caused another 12%.

Although there are other types of dementia that also co-occur with Alzheimer's, mixed dementia generally refers to the co-occurrence of Alzheimer's and vascular dementia.

The other important dementia type that co-occurs with Alzheimer's at a high rate is dementia with Lewy bodies, also considered to be one of the most common dementias (although, due to inconsistent criteria, estimates of its actual prevalence vary wildly). It is estimated to co-occur with Alzheimer's pathology around half the time. At a lesser frequency, but still high, is Parkinson's disease dementia -- about 20% of Alzheimer's patients also have Parkinson's disease.

But it is probably fair to say that the distinction between these dementia types is not clear-cut. Lewy bodies are found in a high proportion of both Alzheimer's and Parkinson's patients -- the number of cases of 'pure' Lewy body dementia is much smaller. It's been said, in fact, that the main difference between Lewy body dementia and Parkinson's disease dementia lies in the timing -- Parkinson's disease dementia will be preceded by at least a year and more likely a number of years, by full-blown Parkinson's disease.

Regardless of the difficulties in establishing clear clinical criteria, however, there is no doubt that Alzheimer's co-occurs with vascular pathology or Lewy body pathology at a startlingly high rate.

One of the problems with clearly distinguishing between these types of dementia is a happy one: vascular and Alzheimer's pathology can be found, at autopsy, in many elderly brains that have *not* shown symptoms of dementia.

For example, in one community-based study[4], in which the median age at death was around 85 for the 209 individuals, 48% had had dementia, of whom 64%

showed Alzheimer's pathology. However, 33% of those who had not had dementia showed similar levels of Alzheimer's plaques. Similarly, some amount of tau tangles (another aspect of Alzheimer's pathology) was found in 61% of the demented and 34% of the non-demented individuals. Finally, multiple vascular pathology was found in 46% of the demented group and 33% of the non-demented, and vascular lesions were equally common in both.

And in the large long-running study mentioned earlier[14], in those without dementia, brain autopsy revealed the presence of Alzheimer's in 24% of cases, and infarctions in 18%.

How likely am I to develop dementia?

The question of how likely any person is to develop dementia must begin with estimates of prevalence, but this of course is only the very beginning of the story.

Estimating prevalence is complicated by the fact that dementia is greatly affected by lifestyle, environmental, and genetic factors, and consequently prevalence varies a lot depending on geographic region.

Different dementia sub-types have different causes, and some give a much greater weight to genetic or environmental factors than others. However, the finding that dementia risk is much greater in those with more than one pathology, and that Alzheimer's pathology with cerebral infarcts is a very common combination, adds to growing evidence that dementia risk might be reduced with the same tools we use for cardiovascular disease such as control of blood cholesterol levels and hypertension.

Age as a factor

The first American study to use nationally representative data[5] (rather than extrapolating from regional data) came up with a figure of 13.9% of those aged 71 and older (one in seven). But age of course makes all the difference in the world. The study found 5% of those aged 71 to 79, rising to 37.4% of those age 90 and older.

Although all the dementia types show an increase with age, Alzheimer's is particularly a disorder of age: although the study found only 46.7% of those with dementia in their 70s had Alzheimer's, for those in their 90s, Alzheimer's was the dementia type for 79.5% of them.

An Italian study of over 2000 seniors over 80 years old[6] confirms that dementia does indeed keep increasing with age (it had been thought that risk leveled off for those who reached their 90s). The study found that 13.5% of those aged 80 to 84 had dementia, rising sharply to 30.8% of those 85 to 89, 39.5% of those 90 to 94, and 52.8% among those older than 94.

Gender as a factor

A number of studies have found differences between men and women, or between difference ethnicities, but this large, nationally representative study found that, although on the face of it there were race and gender differences, these differences disappeared once age, years of education, and presence of at least one "Alzheimer's gene" was taken into account.

However, an American study of over 900 seniors over 90 years old[7] found that women of this age were much more likely to have dementia than men (some 45% of them, compared to 28% of the men), and that the likelihood of having dementia kept increasing with age for the women, but not for the men. Of course, more women than men survive to this age (some two-thirds of the participants were women).

Interestingly, education was protective for the women (the risk of dementia decreasing the more years of education the individual had had) but not for the men. The study participants were not, however, a random sampling -- they all came from the same retirement community, and most were white and of high socioeconomic status. Given that, and considering the times in which they were born, it seems likely that there would be far more variability in educational level among the women than the men. The men, while less likely to develop dementia, did tend to decline faster if they did develop it.

The Italian oldest-old study, too, found more women than men had dementia: across all ages, 25.8% of the women and 17.1% of the men.

These figures don't of course tell us how many *develop* dementia at those ages. Obviously, survival rates are a factor, and as we saw in the other study, male and female survival rates do vary. The figures for new cases of dementia developing in these age bands were:

- 6% at 80 to 84 years;
- 12.4% at 85 to 89 years;
- 13.1% from 90 to 94 years; and
- 20.7% among those over 94.

These figures make even more clear what was apparent in the earlier figures: dementia jumps suddenly in the later half of the 80s, and again in the later half of the 90s.

Importantly, however, the incidence of new cases shows us how important the gender difference in survival rates is: the difference in prevalence is much smaller in these terms --9.2% among women and 7.2% among men.

The study, which canvassed everyone in the age group within a specific geographical area and had an 88% response rate, had a ratio of 74 women to 26 men. Because the number of men at the very highest ages was so small, we can't draw any firm conclusions about gender differences at those ages.

The Italian study involves a very different population from that of the American study: Varese is in a heavily industrialised part of northern Italy, with a high immigrant population, and the average amount of education was only 5.1 years.

A review of 26 studies looking at dementia prevalence in Europe[8] confirmed rates for men rising from 1.8% in the 65-69 years age range up to 30% in the over 90 years age group, and for women rising from 1.5% to 30% in the 80-85 years age band. However (and confirming the American study), rates in the oldest old for women rose to over 50% in those over 95 years.

Early onset of dementia

The average age at the onset of dementia is around 80 years. Early-onset dementia is defined arbitrarily (and variably) as occurring before 60-65. Early-onset cases have been estimated to make up about 6-7% of all cases of Alzheimer's disease, and though a lot of attention has been given to them, only about 7% of early-onset cases are in fact familial[9].

Familial cases involve mutations in specific genes (the APP or presenilin genes); they do not include what is popularly referred to as the "Alzheimer's gene" -- variants of APOE. A 1995 study[10] calculated that a person with no family history of Alzheimer's disease who has an e4 allele has a lifetime risk of 29%, compared to a risk of 9% if they don't have an e4 allele. In other words, if you don't have any of the Alzheimer's risk genes, or any family history, you only have a 9% risk of developing Alzheimer's, and even if you do have the "Alzheimer's gene", your chance of *not* getting Alzheimer's is still over 70%. Your risk does, however, go up dramatically if *both* your APOE alleles are e4.

A large study[11] found, however, that there were both ethnic and gender differences for the risk of this genetic factor. The effect of having an e4 allele was much greater among Japanese compared to Caucasian, and greater for Caucasian compared to African American and Hispanic. Additionally, the effect of having an e4 allele becomes less significant after 70.

There is evidence[12] that the age of onset for both Alzheimer's and Parkinson's diseases, for those genetically disposed, is controlled by genes on chromosome 10.

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General

PET's targeted imaging may lead to earlier diagnosis of dementia

Use of PET imaging with the radiotracer fluorodeoxyglucose (FDG) has allowed researchers to classify different types of dementia (Alzheimer's, frontotemporal dementia, and dementia with Lewy bodies) with very high rates of success (94%). [1]

Memory loss becoming less common in older Americans

A new nationally representative study involving 11,000 people shows a downward trend in the rate of cognitive impairment among people aged 70 and older, from 12.2% to 8.7% between 1993 and 2002. It's speculated that factors behind this decline may be that today's older people are much likelier to have had more formal education, higher economic status, and better care for risk factors such as high blood pressure, high cholesterol and smoking that can jeopardize their brains. In fact the data suggest that about 40% of the decrease in cognitive impairment over the decade was likely due to the increase in education levels and personal wealth between the two groups of seniors studied at the two time points. The trend is consistent with a dramatic decline in chronic disability among older Americans over the past two decades. [2]

Vitamin E or C does not reduce risk of dementia or Alzheimer's

A five-year study involving nearly 3000 people has found that use of Vitamin C or E or both was not associated with a reduced risk of developing dementia or Alzheimer's. [3]

PLMI factor in sleep disturbance for dementia patients

A study of 102 people diagnosed with both cognitive impairment and sleep disturbance (average nightly sleep of seven or less hours and daytime sleep of 30 minutes or longer) found that periodic leg movement disorder (a condition that causes people to jerk and kick their legs every 20 to 40 seconds during sleep) was predictive of reduced total sleep time in older adults with Alzheimer disease and

related dementias. Given that sleep disturbance in persons with dementia is a highly prevalent and disabling symptom, and sedative-hypnotics are not recommended, this finding is important because it suggests treatment of periodic leg movements may be beneficial. [4]

Mental and physical exercise delays dementia

A study using genetically engineered mice has found providing the mice with an enriched environment that enhanced their mental and physical stimulation improved performance on memory tests at an early stage of Huntington's disease, when memory impairment has begun. Specific molecular changes were also observed at the synapses in the hippocampus. Those without increased mental and physical activity showed decreased levels of specific proteins that are expressed at the synapse, but those exposed to stimulation didn't. The finding offers hope for slowing the progression of the disease, as well as other dementias. [5]

National study of dementia prevalence

A study using data from 856 men and women who participated in the nationally representative Aging, Demographics and Memory Study estimates one in seven or 13.9% of the American population aged 71 and older, have some form of dementia. About 70% of those, or 9.7% of the population, have Alzheimer's, and 17.4% of them have vascular dementia. As expected, the prevalence of dementia increased dramatically with age, from 5% of those aged 71 to 79 to 37.4% of those age 90 and older. With increasing age, Alzheimer's disease accounted for progressively more of the dementia cases, so that in the 90+ age group, it comprised 79.5% of dementia cases, compared to 46.7% among those in their 70s. There was no difference between genders when corrected for education and age. Previous national estimates of dementia prevalence have been extrapolated from regional samples, and were generally significantly lower. [6]

How whole-brain radiation might cause dementia

Whole-brain radiation is widely used to treat recurrent brain tumors as well as to prevent other cancers from spreading to the brain. About a half of patients later

develop progressive memory problems. A new study has now identified changes in brain chemistry that may be responsible. Using middle-aged rats, researchers found changes in brain receptors for the neurotransmitter glutamate. The changes may impair synaptic communication. [7]

More light on adult neurogenesis; implications for dementia and brain injuries

New research has demonstrated that adult mice produce multi-purpose, or progenitor, cells in the hippocampus, and indicates that the stem cells ultimately responsible for adult hippocampal neurogenesis actually reside outside the hippocampus, producing progenitor cells that migrate into the neurogenic zones and proliferate to produce new neurons and glia. The finding may help in the development of repair mechanisms for people suffering from dementia and acquired brain injury. [8]

Study links adolescent IQ/activity levels with risk of dementia

An analysis of high school records and yearbooks from the mid-1940s, and interviews with some 400 of these graduates, now in their 70s, and their family members, has found that those who were more active in high school and who had higher IQ scores, were less likely to have mild memory and thinking problems and dementia as older adults. [9]

Walking may protect elderly from dementia

A study of more than 2,200 Japanese-American men between the ages of 71 and 93 has found that elderly men who are sedentary or walk less than a quarter of a mile per day are nearly twice as likely to develop dementia and Alzheimer's disease compared to men who walk more than two miles per day. Those who walked less than a mile (and more than quarter of a mile) a day also showed a significantly greater risk of dementia than those walking more than two miles a day. [10]

Drinking too much alcohol, and not enough, increases risk of cognitive impairment

In Finland, researchers re-examined 1018 participants from a study of 1464 men and women aged 65-79 studied in 1972 or 1977. They found that participants who drank no alcohol in midlife as well as those who drank alcohol frequently were twice as likely to have mild cognitive impairment in old age compared to those who drank alcohol infrequently. The effect of alcohol was however modified by the presence of the apolipoprotein e4 allele (implicated in dementia risk). People who were carriers of the apolipoprotein e4 allele had an increased risk of dementia with increasing alcohol consumption, with carriers of the gene significantly reducing their risk by never drinking. [11]

Estrogen-alone hormone therapy could increase risk of dementia in older women

A new report from the Women's Health Initiative Memory Study suggests that older women using estrogen-alone hormone therapy could be at a slightly greater risk of developing dementia, including Alzheimer's disease (AD), than women who do not use any menopausal hormone therapy. Among 10,000 women using conjugated equine estrogens, 37 could be expected to develop dementia, compared to 25 in 10,000 women using the placebo. Previous reports from the Study found a greater risk with hormone therapy involving both estrogen plus progestin: among 10,000 women over age 65 using estrogen plus progestin there might be 45 cases of dementia compared to 22 cases in 10,000 older women on placebo.
It was also reported that beginning estrogen-alone hormone therapy after age 65 can have a small negative effect on overall cognitive abilities and that this negative effect may be greater in women with existing cognitive problems. [12]

More evidence that mental exercise helps prevent or postpone dementia

Another study provides support for the idea that mentally demanding activities can help stave off dementia. The study involved 469 people aged 75 and older. Over the course of the study, dementia developed in 124 of the participants (Alzheimer's disease in 61, vascular dementia in 30, mixed dementia in 25, and other types of

dementia in 8). Those who participated at least twice weekly in reading, playing games (chess, checkers, backgammon or cards), playing musical instruments, and dancing were significantly less likely to develop dementia. Although the evidence on crossword puzzles was not quite statistically significant, those who did crossword puzzles four days a week had a much lower risk of dementia than those who did one puzzle a week. Most physical activities, like group exercise or team games, had no significant impact. The only exception - ballroom dancing - possibly occurred because of the mental demands of remembering dance steps, responding to music and coordinating with a partner. Although the study was careful to include only those who showed no signs of dementia at the start, it cannot be ruled out that people in pre-clinical stages of dementia may be less likely to participate in mentally demanding activities. [13]

HIV-associated dementia

Green tea extract protects against HIV-associated dementia

A compound derived from green tea greatly reduced the neurotoxicity of proteins secreted by the human immunodeficiency virus, suggesting a new approach to the prevention and treatment of HIV-associated dementia. [14]

References

Vascular & Mixed Dementia

Prevalence

Vascular dementia, as its name suggests, is caused by poor blood flow, produced by a single, localized stroke, or series of strokes.

It is the second most common dementia, accounting for perhaps 17% of dementias. It also co-occurs with Alzheimer's in 25-45% of cases. Although there are other types of dementia that also co-occur with Alzheimer's, mixed dementia generally refers to the co-occurrence of Alzheimer's and vascular dementia.

Risk factors

In general, unsurprisingly, vascular dementia has the same risk factors as cerebrovascular disease.

A study of 173 people from the Scottish Mental Survey of 1932 who have developed dementia has found that, compared to matched controls, those with vascular dementia were 40% more likely to have low IQ scores when they were children than the people who did not develop dementia. Because this was not true for those with Alzheimer's disease, it suggests that low childhood IQ may act as a risk factor for vascular dementia through vascular risks rather than the "cognitive reserve" theory. [1]

Prevention

Again, as a general rule, the same things that help you protect you from heart attacks and stroke will help protect you from vascular dementia.

A four-year study involving 749 older adults has found that the top one-third of participants who exerted the most energy in moderate activities such as walking were significantly less likely to develop vascular dementia than those people in the bottom one-third of the group. [2]

Treatment

The herb gastrodine has been used in China for centuries to treat disorders such as dizziness, headache and even ischemic stroke. Now a 12-week, randomized, double-blind trial involving 120 stroke patients who were diagnosed with mild to moderate vascular dementia has found that gastrodine and Duxil® (a drug used to treat stroke patients in China) produced similar overall levels of cognitive improvement -- although more patients showed 'much improvement' with gastrodine (23% vs 14%). [3]

A Chinese pilot study involving 25 patients with mild to moderate vascular dementia found that ginseng compound significantly improved their average

memory function after 12 weeks, but more research (larger samples, placebo-controls) is needed before this finding can be confirmed. Five years on I have still not seen such a study. [4]

References

Dementia with Lewy Bodies

LBD: What is it?

Lewy Body Dementia is so called because the brains of affected people develop abnormal spherical masses of protein, called Lewy bodies, inside nerve cells. Lewy bodies are associated with Parkinson's disease as well as dementia. Thus Lewy body dementia can refer to both Parkinson's disease dementia and "dementia with Lewy bodies". Lewy bodies are also often found in the brains of those with Alzheimer's disease.

Unlike Alzheimer's, however, dementia with Lewy bodies characteristically (but not invariably) begins with visual hallucinations.

Prevalence of LBD

Estimates of its prevalence are complicated by the lack of clearly defined clinical criteria, and vary widely. A 2005 review[1] concluded that the range probably falls between 0 to 5% in the general population, and from 0 to 30.5% of all dementia cases (the very broad range reflects the confusion between Parkinson's disease dementia (PDD), dementia with Lewy bodies, and Alzheimer's where Lewy bodies are present).

How does LBD differ from Alzheimer's & PDD?

A comparison of these three disorders found that cognitive impairment in those with Alzheimer's disease and those with Lewy body dementia was similar, and more severe than in those with Parkinson's disease dementia.

The 1997 study[2] also found that a simple test, in which patients are asked to draw and copy a clock face, distinguished those with Alzheimer's and those with Lewy body dementia — of all the groups, only those with Lewy body dementia had equally poor scores in the "copy" part of the test compared to the "draw" part.

For more information:

Mayo Clinic: http://www.mayoclinic.com/health/lewy-body-dementia/DS00795

Lewy Body Dementia Association: http://www.lewybodydementia.org/

References

Frontotemporal Dementia

What is it?

Frontotemporal dementia is a disorder of the frontal lobes and includes what was known as primary progressive aphasia. Although it occurs far less often than Alzheimer's disease, among dementia sufferers younger than 65 it is estimated to occur at about the same rate. In other words, frontotemporal dementia is, unlike the most common dementias, not a disorder of age. Most sufferers become symptomatic in their 50s and 60s.

Frontotemporal dementia generally begins with a focal symptom, such as aphasia, before (usually a number of years later) progressing to more generalized dementia.

There are several types of frontotemporal dementia. The most common (around 60% of FTD cases) is known as the behavioral variant (also, Pick's disease). This is characterized by impairment in social and emotional skills. The other 40% of FTD cases have language impairments -- about half of these suffer from semantic FTD, characterized by difficulties in remembering the meanings of words; the other half suffer from progressive nonfluent aphasia, characterized by difficulties in producing language (although they understand what they're trying to say).

In around 15% of FTD cases (most usually the behavioral variant), motor neurone disease also develops.

Prevalence

A large-scale epidemiological study[1] in the Netherlands indicated frontotemporal dementia occurs at a rate of 1.1 per 100,000, with the prevalence highest among those ages 60 to 69, at 9.4 per 100,000. The prevalence among people ages 45 to 64 was estimated to be 6.7 per 100,000 (this was after autopsies caused the number of diagnosed cases to go up, with 17 of 50 patients undiagnosed in life). Unlike other forms of dementia, where most occurrences begin in older adults, symptoms began after age 65 in only 22% of patients. The median age of onset was 58, with a range from 33 to 80.

A family history of dementia was present in 43% of patients. Interestingly, whites accounted for 99% of all cases despite an ample nonwhite population.

A large U.K. study[2] found prevalences of early-onset FTD and Alzheimer's were the same in the 45-64 population: 15 per 100,000. The mean age at onset of FTD was 52.8 years and there was a striking male preponderance (14:3).

This rate is notably higher than that found in the Dutch study, and it has been suggested that the reason is ethnicity -- the Dutch study, as mentioned, had a significant proportion of non-Caucasians, while the British (Cambridge) study explicitly mentioned that minorities were under-represented.

It has been estimated that frontotemporal dementia accounts for approximately 8% of patients with dementia, but this is now thought to be an underestimation.

Genes as a factor

There is a high level of genetic involvement in this type of dementia.

As mentioned, the Dutch study found a family history of dementia in 43% of FTD patients. Another large Dutch study[3] found 38% of FTD patients had one or more first-degree relatives with dementia before age 80 compared to 15% of age-

matched controls; 10% had two or more first-degree relatives with dementia compared with 0.9% of the controls. FTD patients were also three times more likely to have two "Alzheimer's genes" (two e4 alleles of the ApoE gene) than the controls: 7% vs 2.3%.

This study also supports findings with other dementias that earlier-onset is more likely to have genetic causes. First-degree relatives of FTD patients (who had twice the risk of dementia before age 80 compared with relatives of controls) were much more likely to develop dementia early: age of onset of dementia in affected first-degree relatives of FTD patients averaged was just under 61, compared to 72.3 for affected first-degree relatives of controls.

The genes implicated in familial cases of FTD are on chromosome 17, in the gene for the tau protein, and in the gene for the progranulin protein. Research[4] has now confirmed that people with these hereditable defects produce only half of the normal amount of progranulin, and recently a simple test for measuring the quantity of progranulin in the blood was developed. The test reveals whether someone has the mutations that carry an increased risk of FTD.

A recent study[5] involving 225 FTD patients found 41.8% of patients had some family history, although only 10.2% had a clear autosomal dominant history (at least 3 cases within the last 2 generations). However, the importance of genes varied across the different clinical subtypes of the disease, with the behavioral variant being the most heritable and FTD–motor neuron disease and the language syndromes (particularly semantic dementia) the least heritable.

For more information:

http://emedicine.medscape.com/article/1135164-overview

http://memory.ucsf.edu/ftd/

References

Parkinson's Disease Dementia

Prevalence of Parkinson's Disease

After Alzheimer's disease, the second most common neurodegenerative disorder is Parkinson's disease. In the U.S., at least 500,000 are believed to have Parkinson's, and about 50,000 new cases are diagnosed every year[1] (I have seen other estimates of 1 million and 1.5 million -- and researchers saying the numbers are consistently over-estimated while others that they are consistently under-estimated!). In the U.K., the numbers are 120,000 and 10,000[2].

Part of the problem in estimating national and global prevalence is that Parkinson's is very much affected by environmental factors. The Amish, Nebraska, the area around the ferromanganese plants in Breccia (Italy), and the Parsi of Mumbai (India), have the highest rates of Parkinson's in the world. Pesticide use, and some occupations and foods, are all thought to increase the risk of Parkinson's. So is head trauma.

There may also be ethnic differences. A recent analysis of Medicare data[3] from more than 450,000 patients with PD in the United States has found substantial variation between whites, African Americans, and Asians, with whites showing dramatically greater rates (158.21 per 100,000 in white men compared to 75.57 and 84.95 for African Americans and Asians, respectively). These differences, however, may well reflect factors other than ethnicity, given the significant role that environmental factors play in Parkinson's. Most patients were found to live in the Midwest and Mid-Atlantic regions (areas with very high proportions of whites).

Of course Parkinson's, like Alzheimer's, is a disorder of age (although in both cases, a minority suffer early onset). Figures from a 1997 European study[4] that estimated the overall, age-adjusted prevalence in Europe at 1.6% gave this age breakdown:
65-69: 0.6%
70-74: 1.0%
75-79: 2.7%
80-84: 3.6%

85-89: 3.5%
As you can see, there is a sharp rise in the later half of the 70s, rising to a peak in the 80s (studies suggest it declines in the 90s).

Risk of developing dementia

Parkinson's is of course primarily a movement disorder, not a cognitive one. However, it can lead to dementia. As with the numbers of Parkinson's sufferers, the risk of that is so variously estimated that estimates range from 20-80%!

Part of the problem is disentangling mortality — as with Alzheimer's, many die before the symptoms of dementia have had time to develop. It is helpful to deconstruct that top statistic.

The 2003 Norwegian study[5] that appears to be the source of this 80% calculated an 8-year prevalence estimate of 78.2% from an 8 year study involving 224 Parkinson's patients. At the beginning of the study, 51 of these 224 had dementia. After 4 years, 36 of the non-demented had died, and 7 refused to continue their participation; of the 51 demented, 42 had died (according to my calculations – this figure, and several others, were not given). Of the 139 patients remaining in the study at year 4, 43 of the previously non-demented had developed dementia, meaning (according to my calculations) that 52 in total now had dementia, and 87 had not. After another 4 years, there were only 87 patients remaining in the study, 19 of those 87 non-demented having died, a further 3 refusing to continue, and (my calculation) 30 of the 52 demented having died. At this time, year 8, 28 of the previously non-demented had now developed dementia, leaving (my calculation) 37 non-demented survivors.

In other words, over a period of 8 years, after having had Parkinson's for over 9 years, on average, when the study began, just over half (54.5%; 122/224) developed dementia. About the same number (56.7%; 127) had died. At that point, after having had Parkinson's for an average of 17 years (they were now on average 73 years old), 50 (22%) were still alive but with dementia, and 37 (16.5%) were still alive and non-demented (the percentage is only slightly increased by subtracting those who refused to continue participating).

Importantly, those 37 had no more cognitive decline than was evident in age-matched controls.

Note also that the average life expectancy after being diagnosed with Parkinson's is about 9 years -- hence, those who participated were already at this point at the beginning of the study. We don't know how many people developed dementia and died between diagnosis and the study beginning, but we do know that 23% (51/224) had dementia at the beginning of the study, after having had Parkinson's for an average of 11 years (their average was higher than the group average) -- which is already longer than the average survival rate.

In other words, we need a study that follows PD sufferers from diagnosis until death to truly give an accurate estimate of the likelihood of developing dementia before death. We can however give an estimate of how many people survive PD for 17 years (nearly twice the average survival time) without developing dementia: 16.5% -- which is approaching half (42.5%) the number of people who survive that long.

We can also estimate how many PD sufferers who have had PD for an average of 9 years will not have dementia: 77% (173/224 - the number of non-demented at the beginning of the study). And how many will not have dementia after 13 years: 63% (87/139 -- the number of non-demented at year 4 of the study).

The big question is of course, are there any signs that indicate which individuals will develop dementia. The researchers found[6] that age, hallucinations, and more severe motor problems were all risk factors for developing dementia.

For more on Parkinson's:

Check out this youtube video:
http://www.youtube.com/watch?v=ZPnpmVWU0Hk

See these websites:
http://www.ninds.nih.gov/disorders/parkinsons_disease/parkinsons_disease.htm

http://www.nhs.uk/Conditions/Parkinsons-disease/Pages/Introduction.aspx

http://viartis.net/parkinsons.disease/

Check out these books:
http://www.amazon.com/Dementia-Lewy-Bodies-Parkinsons-Disease/dp/1841843954

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Glossary

acetylcholine: is what is known as a neurotransmitter -- a chemical produced by brain cells which transmits information within the brain. Acetylcholine is vital for memory, attention and thought. Acetylcholine-producing cells are among the first to die in Alzheimer's disease. Parkinson's disease, dementia due to multiple strokes, multiple sclerosis and schizophrenia, are all, like Alzheimer's, associated with lower levels of acetylcholine in the brain.

amygdala : means "almond", so-named because of its shape and size. The amygdala is part of the basal ganglia, and is situated in the temporal lobe. It has many connections with other parts of the brain, most particularly with the limbic system, for which reason it is considered part of the limbic system (although not part of the limbic lobe itself). The amygdala is critically involved in computing the emotional significance of events, and recent research indicates it is responsible for the influence of emotion on perception, through its connections with those brain regions that process sensory experiences, thus "allowing perception of emotionally significant events to occur despite inattention." Rat studies also suggest that the amygdala, in tandem with the orbitofrontal cortex, is involved in the forming of new associations between cues and outcomes - in other words, it is the work of the amygdala to teach us what happens to us when we do something.

amyloid beta peptides : peptides derived from amyloid precursor protein, these fragments of amyloid beta are the main protein component of plaques, and probably a major cause for the their toxicity. They are thought to bind to a receptor in the brain, blocking the signals needed for learning and memory. The peptides come in two forms: A-beta 42 and A-beta 40. Amyloid beta peptides routinely circulate in the human bloodstream, where they are harmless. Early beta amyloid accumulation within neurons is the trigger for the onset of memory decline in Alzheimer's.

amyloid precursor protein (APP): is found in many tissues besides brain, but its functions are largely unknown. It is anchored across the cell membrane, so part of it is inside and part of it is outside the cell. Enzymes snip it apart into three protein fragments, two of which are released outside the cell and one inside. One of those which is found outside the cell is made of amyloid beta peptides. It's speculated that the creation of amyloid plaque is a byproduct of a misregulation in normal APP processing. Mutation in the APP is thought to be involved in early-onset Alzheimer's; the APP gene is located on chromosome 21, at 21q21.

anterior cingulate: also known as area 24 of Brodmann-1905. The anterior cingulate is a defined area of the cerebral cortex including parts of both the cingulate gyrus and the frontal lobe.

apolipoprotein E (APoE): is a protein whose main responsibility is transporting cholesterol out of the cell. Too much of this protein results in an increase in the level of free cholesterol in the cells. An allele of the gene responsible for this protein has been identified as a major genetic risk factor for Alzheimer's (see APOE gene).

apolipoprotein-E gene (APOE): the e4 allele of the apolipoprotein E gene has been identified as a major genetic risk factor for Alzheimer's. There are three versions (alleles) of the APOE gene; the most common is e3, present in over half the population. Those who inherit one copy of the e4 allele are at higher risk of developing type 2 Alzheimer's, a late-onset form; those who inherit two copies are at greater risk. Most people with familial hypercholesterolemia have 2 copies of

the e4 allele. One study suggests having the e4 allele is particularly risky in combination with a small head size. Similarly, calorie and fat intake appear to increase the risk of developing Alzheimer's in those with the allele. An Australian study has more recently identified the -491A allele as another risk factor. The same study found that people with these alleles were more likely to complain of memory difficulties. APOE is located on chromosome 19. A gene on chromosome 10 has also recently been identified as significantly increasing the risk of Alzheimer's when found in combination with APOE e4.

axon: a long projection extending from the cell body, that carries the output of the neuron away from it.

basal ganglia: are large "knots" (ganglion means knot) of nerve cells deep in the cerebrum. They are thought to be involved in various aspects of motor behavior (Parkinson's disease, for example, is an affliction of the basal ganglia).

beta-amyloid plaques: are considered one of the hallmarks of Alzheimer's disease. The plaques are hard, insoluble aggregations of various peptides and proteins, chiefly and most important amyloid-beta peptides. Recent research suggests plaques attach primarily to blood vessels, damaging them.

brain stem: is the most primitive part of the brain, which also means it controls the most basic functions (such as breathing. It may be thought of as the stem connecting the spinal cord and the cerebral hemispheres.

cerebral cortex: the gray matter outer layer of the cerebrum. The newest part of the brain in evolutionary terms; responsible for primary sensory functions, motor coordination and control, and most particularly, the "higher-order" functions of language and thinking.

cerebrospinal fluid (CSF): a clear salty liquid which cushions the brain

cerebrum: the largest structure of the brain; containing the cerebral cortex (the outer layer), which is made of gray matter, and an inner core composed of white

matter (myelinated nerve fibers and gray basal ganglia); divided into a number of regions known as lobes.

cholinesterase inhibitors: are drugs that slow the breakdown of acetylcholine.

cingulate gyrus: (fold) in the limbic lobe; implicated in self-reflective thought (thinking about yourself and your attributes).

cognitive reserve: the idea that education and mental stimulation during a lifetime can give older adults a cognitive reserve or neuroplasticity that can reduce the effect of brain abnormalities on cognitive function, allowing them to function normally for longer in the presence of such brain abnormalities.

corona radiata: sheet of white matter that is continuous with the internal capsule.

corpus callosum : the main "bridge" between the left and right cerebral hemispheres; a broad bundle of myelinated fibers (white matter) carrying information from regions in one lobe to similarly placed regions in the opposing lobe.

dentate gyrus: a substructure of the hippocampus, highly active during encoding (learning) of face-name pairs.

donepezil: donepezil hydrochloride is a cholinesterase inhibitor, marketed as Aricept.

enzymes: are a type of protein; they are responsible for catalyzing the chemical reactions in a living cell -- that is, they accelerate the rates of reactions.

frontal lobe : the frontal lobes (left and right) are situated at the "front" of the cortex, i.e. behind the forehead. They are the largest of the lobes in the cerebrum, and may be thought of as the "highest" part of our brain. The frontal lobes are critical for those faculties that humans regard as special to our species - reasoning, planning, attention, some aspects of language. Women have up to 15% more brain cell density in the frontal lobe, but with age, appear to shed cells more rapidly from

this area than men. By old age, the density is similar for both sexes. The effect of this on performance is unknown.

genetically engineered mice: mice that are genetically engineered to develop an Alzheimer's-like disease by the introduction of transgenes. Mice ordinarily do not develop symptoms of the disease. see http://www.mni.mcgill.ca/nm/1999f/en/transgenes.html for a description of how this achieved.

glutamate : an amino acid, it's the most prevalent excitatory neurotransmitter in the adult brain.

gray matter : brain tissue is divided into two types: gray matter and white matter. Gray matter is made up of the cell bodies of nerve cells. The volume of gray matter tissue is a measure of the density of brain cells in a particular region.

hippocampus: means "sea horse", and is named for its shape. It is one of the oldest parts of the brain, and is buried deep inside, within the limbic lobe. The hippocampus is important for the forming, and perhaps long-term storage, of associative and episodic memories. Specifically, the hippocampus has been implicated in (among other things) the encoding of face-name associations, the retrieval of face-name associations, the encoding of events, the recall of personal memories in response to smells. It may also be involved in the processes by which memories are consolidated during sleep.

internal capsule: a collection of axons connecting the cerebral cortex and the brain stem

lateral temporal cortex: a part of the temporal lobe that is implicated in language processing, in particular rhyme.

limbic lobe: a lobe that lies deep within the cerebrum - a broad collar of cortex fringing the corpus callosum (limbic means "border"). The limbic lobe includes the hippocampus, cingulate gyrus, dentate gyrus, and the parahippocampal gyrus.

lobes : the cerebrum is highly convoluted - it is this deep and numerous folding that vastly increases the cortical area of the human brain. The deepest fissures provide somewhat arbitrary boundaries for the mapping of the brain. Following these guidelines, the cerebrum is divided into five lobes: the frontal, temporal, parietal, occipital and limbic lobes.

MCI-A: mild cognitive impairment, amnesic subtype; patients with this disorder show memory impairments but not other cognitive impairments (e.g., in reasoning).

MCI-MCD: mild cognitive impairment, multiple cognitive domain subtype; patients with this disorder show mild impairments in cognitive tasks such as judgment or language, and mild or no memory loss.

mediotemporal lobe (MTL): includes the hippocampus, the amygdala, and the entorhinal and perirhinal cortices. Although given this name, the idea that this is an integrated memory system with a common function has recently been questioned. It is observed that the various components evolved at different points. Nevertheless, we may say that the MTL appears to be involved in declarative learning (facts and events), being particularly important during initial learning. There is some evidence that long-term consolidation of memories is guided by the MTL, in particular by the entorhinal cortex (which is damaged in the early stages of Alzheimer's disease). Moreover, a recent study showed that progressive atrophy in the medial temporal lobe was the most significant predictor of cognitive decline in seniors.

myelin : the sheathing that insulates axons and facilitates speedy communication among neurons.

neurofibrillary tangles : are tangled bundles of fibers inside neurons. Like plaques, they are considered one of the hallmarks of Alzheimer's disease, although they also occur in other neurological disorders. Tangles mainly consist of tau protein. By disrupting the structure of the neuron and disabling the transport of nutrients, tangles cause neurons to die. Plaques can induce tangles, but that is only one way in which tangles can form. Nicotine is, apparently, another.

neurogenesis: creation of new neurons; common in young brains, it has only recently been found to occur in adult brains, and then only in specific regions.

neurotransmitter : a messenger chemical in the brain; it is through neurotransmitters that neurons communicate with each other. Examples are GABA, glutamate, acetylcholine, dopamine, serotonin, norepinephrine.

occipital lobe : one of the lobes of the cerebrum, situated at the back of the skull, and above the hindbrain. It borders with the parietal lobe (from which it is not clearly demarcated) and the temporal lobe. The occipital lobe contains the primary visual cortex, where visual information is processed.

parietal lobe : one of the lobes of the cerebrum, situated at the top, behind the frontal lobe. The primary sensory area is located in the parietal lobe - this is where nerve impulses carrying sensations of pain, temperature, touch, and pressure come. Areas in the parietal lobe are also involved in spatial orientation, speech and language development, and attention.

peptide : a compound of two or more amino acids linked by a peptide bond. Peptides differ from proteins by their size; peptides are shorter. Proteins can be broken down into peptides (this occurs during digestion).

progranulin: is a protein growth factor that helps brain cells survive. A gene mutation that leads to a dramatic loss in production of progranulin has been implicated in frontotemporal dementia. Production of too much progranulin has been associated with cancer.

proteins : are essential to living organisms; they are long chains of amino acids linked together by peptide bonds. Enzymes, hormones, and antibodies are all types of protein.

Religious Orders Study (ROS): a long-running prospective study of aging and cognitive function in Catholic clergy.

Scottish Mental Survey: assessed 87,498 eleven-year-olds in 1932, and another 70,805 in 1947. More recently, over 1000 of these students were contacted and re-assessed, on the exact same tests.

synapse: the site where one neuron makes contact with another

synaptic transmission: the process of transfering information at a synapse.

tau proteins : are proteins that form part of a structure called a microtubule, which helps transport nutrients and other important substances from one part of the nerve cell to another. In Alzheimer's disease, however, the tau protein is abnormal and the microtubule structures collapse, causing neuron death.

temporal lobe : one of the lobes of the cerebrum, situated below the frontal and parietal lobes, and above the hindbrain. The temporal lobe is primarily concerned with sensory experience - specifically, with hearing, and with the integration of information from multiple senses. Part of the temporal lobe also plays a role in memory processing. Patients with damaged temporal lobes appear to have impaired lexical retrieval of names of living things.

thalamus: means "inner chamber", and accordingly is located deep within the cerebrum. It is an egg-shaped structure lying at the very top of the brain stem, above the hypothalamus. The thalamus relays all information received from the senses (except smell) to the various processing centers in the cerebral cortex. Recent research also suggests that the thalamus regulates the electrical rhythms that parts of the brain use to communicate with each other. It has been speculated that tips of the tongue experiences (when only part of a memory is recalled) may occur when the rhythms don't synchronize with the regions properly - which would put these memory failures at the door of the thalamus. The thalamus also seems to be involved in memory consolidation processes that occur during sleep.

transgenes: genes from one organism that have been incorporated into another organism.

vascular dementia: dementia caused by poor blood flow, produced by a single, localized stroke, or series of strokes. It's the second most common type of dementia, after Alzheimer's, accounting for up to a third of diagnosed dementia cases.

white matter : Brain tissue is divided into two types: gray matter and white matter. White matter is made up of the axons of neurons -- the long filaments that extend from the cell bodies and carry the electrical signals that carry the messages between neurons. It's the myelin sheathing that makes it look white. There are three major white matter systems, which all connect to form one continuous system: cortical white matter; the corpus callosum; the internal capsule.

Whitehall Study: A British study questioned some 5,350 civil servants aged between 35 and 55 about their participation in 13 leisure activities, ranging from DIY and housework to cultural visits and evening classes. They were then given tests in verbal memory, mathematical reasoning, vocabulary and verbal fluency.

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Elizabeth Devore – Plasma Cholesterol Levels and Cognitive Function in Aging Women (O3-01-02, Tues., 7/20, 3-5 pm)
Jacobo Mintzer – Effects of Diabetes Mellitus and Other Elderly CVD Risk Factors on Change in cognitive Function Later in Life (P4-015, Wed., 7/21, 12:30)
G. Stennis Watson – Rosiglitazone Preserves Cognitive Functions in Patients with Early Alzheimer's Disease (O4-05-05, Wed., 7/21, 3-5 pm)
Ara Khachaturian – Anti Hypertensive Medication Use May Reduce Risk of Incident AD: The Cache County Study (O3-01-07, Tues., 7/20, 3-5 pm) Press release
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Christopher Clark – Latino Patients with AD Have An Earlier Age of Symptoms Onset Compared to Anglos (P1-041)
James Laditka – Epidemiology of Alzheimer's Disease: Race Effects, Area Variation, and Clustering (P3-132)
Marjorie Marenberg – Racial Differences in Screening of MCI in a Primary Care Population (O4-01-02) Press release
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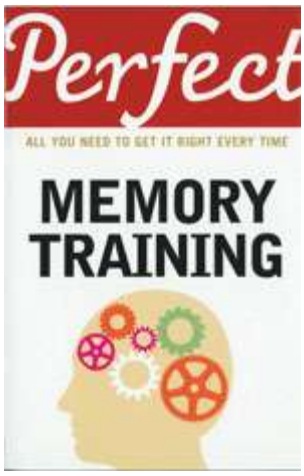

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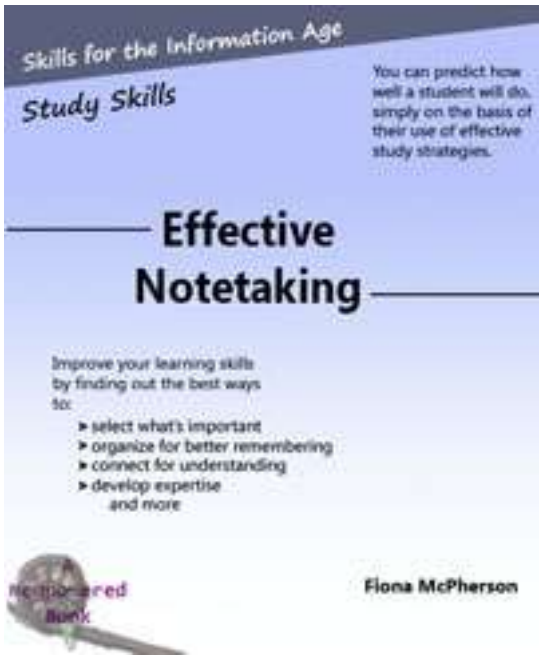

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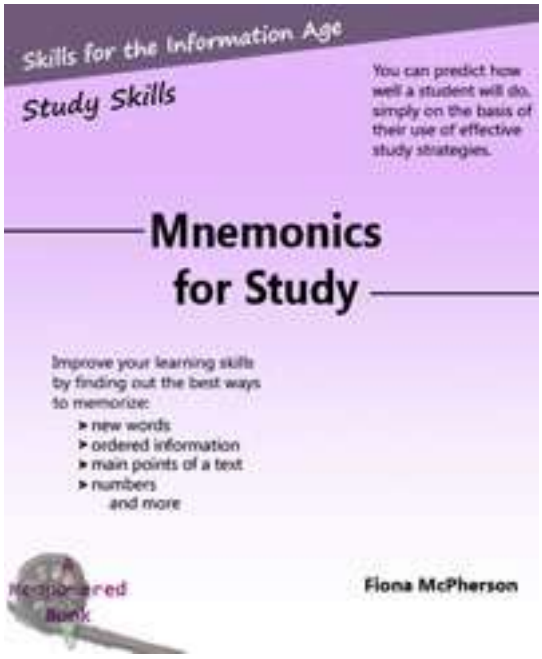

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