

Amnesia and Memory Loss

Helping to Remember

Amnesia occurs when the portion of the brain responsible for retrieving stored memories is somehow compromised. This region of the brain is known as the limbic system; it comprises the hippocampus, the amygdala, and portions of the cortex. Besides retrieving memory, the limbic system is responsible for coordination of emotion and motivation and for some of the functions of the endocrine system.

People are amnesiac when the memory retrieval portion of the limbic system isn't working properly but there is otherwise no change in language, attention span, visual/spatial functioning, or motivation.

Memories are not actually stored in the limbic system or the hippocampus. Rather, several areas of the brain are involved in memory; the type of information being assimilated determines where it is stored. For example, visual and auditory patterns are stored in the temporal lobe, whereas the parietal lobe stores language, speech, word usage, and comprehension.

FORMS OF AMNESIA: DIFFERENT WAYS TO FORGET

There are two types of memory. Short-term or "working" memory stores information one needs to remember in the next few seconds, minutes, or hours (e.g., a telephone number or driving directions). Long-term memory includes relational and procedural memory. Relational memory is concerned with relationships among objects and depends on the hippocampus. In amnesia, both relational memory and short-term memory may be impaired. Procedural memory represents memory for single objects or tasks (e.g., riding a bicycle) and depends on cortical processors that remain intact in amnesia. This helps explain why amnesiacs often remember basic skills and motor function.

There are several forms of amnesia:

- Anterograde amnesia is the most common. It is characterized by the inability to store, retain, or recall new knowledge after the event that triggers the onset of amnesia. Patients in this state often cannot remember what they ate for their last meal or events from the immediate past. They may fill in gaps in their memory with fabricated events (confabulation). This is the type of amnesia seen in dementia and Alzheimer's disease.
- Retrograde amnesia is the loss of memories of events that occurred before the onset of amnesia. This is the form of amnesia most people think of when they hear the word amnesia. It often occurs after a head injury.
- Transient global amnesia is a temporary loss of all memory, especially the ability to form new memories, with milder loss of past memories, going back several hours. This form is rare and seen mostly in older people. It usually dissipates within 24 to 48 hours. Transient global amnesia may be caused by migraine, small seizures in the temporal lobe, or transient ischemic attacks. Patients with this condition may become disoriented and repeatedly ask who they are, where they are, and what they are doing. Because this form of amnesia typically resolves on its own and only rarely recurs, there is no recommended treatment for it.

There are many possible causes of amnesia. The most common include Alzheimer's disease, traumatic brain injury (head trauma), brain infection (such as encephalitis or meningitis), dementia, seizures, and stroke. Less common causes include a brain tumor or psychiatric disorders (schizophrenia, depression, criminal behavior, or psychogenic amnesia). Psychogenic amnesia usually happens in close association with a stressful event that involves serious threat to life or health. Criminals frequently present with amnesia: reports indicate that 23 percent to 65 percent of murderers claim amnesia for their crimes (Taylor PJ et al 1984)

Amnesia can occur because of brain damage that interferes with memory storage, retrieval, or consolidation. What ultimately causes the memory loss—a failure to store memories or a failure to retrieve them—remains unclear. However, a study using rats suggested that memory loss is probably due to an error in memory retrieval, which explains why amnesiacs can usually recover their memories (de Hoz L et al 2004).

Amnesia is also a symptom of Wernicke-Korsakoff syndrome. Wernicke-Korsakoff is caused by a severe thiamine (vitamin B1) deficiency due to chronic alcoholism or malnourishment. Thiamine is necessary for the body to process carbohydrates. Besides amnesia, symptoms of Wernicke-Korsakoff include confusion, loss of balance, drowsiness, and problems with vision, such as double vision or rapid movement of the eye. In severe cases, the memory loss may be accompanied by agitation and dementia. The standard treatment is intravenous thiamine, administered as soon as possible after symptoms become apparent. This therapy does not correct the condition, however, and recovery may be gradual and incomplete.

Drugs besides alcohol can lead to amnesia. These include recreational drugs such as cocaine, LSD, PCP, and mescaline. Several prescription medications, including aminophylline, barbiturates, bromide, digoxin, diuretics, isoniazid, methyl dopa, and tricyclic antidepressants, can also cause transient amnesia (Brna TG et al 1990). Any drug-related impairment is usually resolved once the drug is discontinued.

LIFE EXTENSION'S APPROACH TO AMNESIA

Any neurological disorder represents a challenge, not only for the patients and their families, but also for the treating physician. Patients with amnesia may be occasionally disoriented, and their symptoms may strongly resemble psychiatric disorders (Kasper DL et al 2005). If the amnesia is caused by an underlying condition such as Alzheimer's or dementia, physicians may prescribe drugs for that condition. Patients with these conditions are encouraged to read *Alzheimer's Disease and Preserving Mental Sharpness* for more detailed information.

Life Extension's approach to amnesia is based on the assumption that taking supplements that have been shown to boost memory and brain function will help in amnesia. It is important to visit a physician if amnesia is present because amnesia usually occurs as a result of another condition. In most cases, the supplements discussed in this chapter have not been studied specifically for amnesia but have been researched more generally for their ability to enhance cognitive function, memory retention, and recall, especially in the context of dementia and Alzheimer's disease.

NUTRITIONALLY SUPPORTING HEALTHY MEMORY

There are several herbs, vitamins, and supplements that may help boost memory and provide support for the brain. These work through various mechanisms: enhancing cerebral blood flow, increasing neurotransmitter levels, reducing free radicals, and restoring cell membrane fluidity.

Glyceryl phosphoryl choline (GPC). GPC is a form of choline that is naturally present in all the body's cells. Among aging adults, the rationale for GPC therapy goes back to the hypothesis, developed more than 30 years ago, that declining levels of acetylcholine—and a concurrent decrease in the number of neurons that are its intended target—are responsible for a range of cognitive deficits (Koistinaho M et al 2005). Acetylcholine is an essential neurotransmitter involved in muscle control, sleep, and cognition. By boosting acetylcholine levels in the brain, the hypothesis proposes, it may be possible to reverse cognitive deficits (Parnetti L et al 2001).

Early clinical trials with GPC used daily dosages of 1200 mg. After an initial two to four weeks at this dose, some people reduce their dose to 600 mg daily. A daily dose of 300 mg may be appropriate for healthy young people.

Ashwagandha. A medicinal plant used in India to treat a wide range of age-related disorders (Bhattacharya SK et al 2000; Mishra LC et al 2000; Owais M et al 2005; Mohan R et al 2004; Prakash J et al 2002; Padmavathi B et al 2005; Andallu B et al 2000; Dhuley JN 2001; Chaudhary G et al 2003; Choudhary MI et al 2004; Kuboyama T et al 2005). Its most remarkable effect may involve its ability to preserve the health of the aging brain.

Phosphatidylserine. Phosphatidylserine is essential for brain health because it helps the brain use its fuel. By boosting glucose metabolism and stimulating production of acetylcholine, supplemental phosphatidylserine has been shown to improve the condition of patients experiencing age-associated memory impairment or cognitive decline (Amenta F et al 2001; Schreiber S et al 2000; Delwaide PJ et al 1986; Funfgeld EW et al 1989; Crook TH et al 1991).

Grape seed extract. Recent research indicates that grape seed extract may play a specific role in protecting the brain by preventing the kind of neuronal toxicity experienced by patients with Alzheimer's disease. Korean scientists pretreated rat brain cells with grape seed extract in the laboratory before exposing the cells to beta-amyloid (Abeta), a toxic protein implicated in the formation of senile plaques in the brains of Alzheimer's patients. Untreated cells exposed to Abeta accumulated damaging reactive oxygen species (free radicals) and underwent programmed cell death. However, the rat brain cells pretreated with grape seed extract were significantly protected from the toxic effects of Abeta (Li MH et al 2004).

Pregnenolone and DHEA. Many studies have shown that hormone levels in the brain are closely tied to cognitive function and memory. Pregnenolone, the "master" sex hormone, is the first hormone in the cascade. It is derived from cholesterol. In the body, pregnenolone is converted into other important hormones, including dehydroepiandrosterone (DHEA), estrogens, progesterone, and testosterone (Meieran SE et al 2004). Aging causes a sharp decline in pregnenolone production, and levels of the hormones for which it is a precursor tend to decline with age as well (Karishma KK et al 2002; Goncharova ND et al 2002; Zietz B et al 2001).

DHEA levels have also been shown to decline with age, and patients with cognitive disorders such as Alzheimer's experience a steep decline in DHEA (Tan RS et al 2001). Like pregnenolone, DHEA is a neuroactive steroid that can help regulate brain function (Racchi M et al 2001). Animal studies have shown that DHEA interacts with amnesiac mice by stimulating the sigma-1 receptor in

the brain, which is involved in memory. Other animal studies have shown that DHEA improves short-term and long-term memory in a variety of amnesia models (Mathis C et al 1999).

Ginkgo biloba. Ginkgo biloba leaf extract is the most widely sold phytoextract in Europe with 5 million prescriptions written in Germany alone every year for dementia. It has been used in Chinese medicine for thousands of years to treat respiratory ailments, improve circulation, aid digestion, and combat memory loss in the elderly.

In the United States, ginkgo is mostly used as an aid for mental acuity and memory and as an antioxidant. A review of eight randomized studies demonstrated that ginkgo has modest effects on symptoms of dementia, including memory loss (Kleijnen J et al 1992). Another analysis of 50 articles examined the effect of ginkgo on cognitive function in patients with Alzheimer's. Four of the studies met criteria for adequate clinical trial design. Each study showed that Alzheimer's patients who received ginkgo experienced 10 percent to 20 percent improvement in standardized tests of attention, short-term memory, and reaction time compared to patients who took placebo. The reviewer reported that ginkgo's effects were comparable to the benefits of donepezil (Aricept®) (Rogers SL et al 1998).

An analysis of 33 trials concluded that ginkgo appears safe and shows promising evidence of offering improvement in cognition and function. However, overall trial results were inconsistent, which has prompted the National Institutes of Health to sponsor a multicenter, 6-year, randomized trial of 2000 patients. The trial will evaluate safety and efficacy of ginkgo in preventing dementia and age-related cognitive decline. Another trial is ongoing at the Oregon Health Sciences Center to study the effects of ginkgo on cognitively intact elderly patients older than age 85 and its effect on their progression to mild cognitive impairment.

Vinpocetine. Vinpocetine (vinpocetine-ethyl apovincamate) is a synthetic compound extracted from the seeds of the periwinkle plant (*Vinca minor*). It has been used widely in Hungary, Poland, and Germany for cerebral-related pathologies and became available in the United States in 1998 as an herbal supplement. It has several pharmacological properties, including antioxidant, vasodilator, and neuroprotective benefits. Animal studies have shown that it crosses the blood-brain barrier and is absorbed by cerebral tissue (Gulyas B et al 1999).

Vinpocetine is an effective scavenger of hydroxyl radicals (Stole S 1999) and has been shown to inhibit lipid peroxidation in mouse brain tissue. It leads to enhanced cerebral circulation and decreased platelet aggregation (Chiu PJ et al 1988). It has also been found to have antioxidant properties comparable to vitamin E (Miyamoto M et al 1989).

A double-blind study testing vinpocetine's effect on short-term memory in 12 healthy women showed that those who took 40 mg three times a day for two days scored about 30 percent higher on short-term memory tests than the placebo group. (Subhan Z et al 1985). Another study demonstrated the effects of 30 to 60 mg of vinpocetine daily in patients with mild to moderate dementia: after 16 weeks, 21 percent who took it reported their symptoms subsided, compared with 7 percent of those who took a placebo (Szatmari SZ et al 2003)

A meta-analysis of six randomized, controlled trials involving 731 patients with degenerative cerebral dysfunction showed that vinpocetine was highly effective on cognitive and motor function (Nagy Z et al 1998).

Memory endurance can be measured in the laboratory by the presence of electrical potentials. In lesioned brains of rats, reduced long-term memory was restored by vinpocetine as measured by the normalization of electrical potentials (Molnar P et al 1994).

Huperzine A. Derived from the leaves of the Chinese club moss *Huperzia serrata*, huperzine A demonstrates beneficial characteristics similar to those of ginkgo. It acts like an antioxidant and has neuroprotective properties, including the ability to inhibit the breakdown of acetylcholine, an important neurotransmitter. Most of the studies examining huperzine A have been conducted in Alzheimer's patients.

Huperzine A has been shown to increase acetylcholine levels in rat brains. It also increases norepinephrine and dopamine. Several Chinese studies suggest that this herb may be as effective as tacrine and donepezil against Alzheimer's disease. A study in China (Cheng DH et al 1996) demonstrated a connection between huperzine and improved cognitive function in dementia patients. Another study involved 50 Alzheimer's patients who were given huperzine A or placebo for eight weeks. Significant improvement was reported in 58 percent of patients treated with huperzine A in terms of memory, cognitive, and behavioral functions; only 36 percent of those who took placebo improved. No adverse side effects were reported (Xu SS et al 1995).

Clinical efficacy and safety of huperzine in treatment of mild to moderate Alzheimer's was conducted in a randomized, placebo-controlled trial in China (Zhang Z et al 2002). This study included 202 patients from various centers. One group received 400 mcg/day of huperzine A, and the other group received placebo. Cognitive function, activity of daily life, and overall clinical efficacy were reported: 70 percent in the huperzine group showed improvement in cognition, behavior, mood, and activity of daily life versus only 36 percent in the placebo group.

A study in rats (Liang YQ et al 2004) compared the effects of huperzine A, donepezil, and rivastigine (current drugs for treating

Alzheimer's) on cortical acetylcholine levels. Results showed that huperzine A was 8-fold more potent than donepezil and twice as potent as rivastigmine in increasing cortical acetylcholine levels with a longer-lasting effect than either of them.

The National Institute on Aging is currently sponsoring a clinical trial in the United States to evaluate the safety and efficacy of huperzine A in the treatment of Alzheimer's disease. It will evaluate whether a regimen of 200 mcg/day improves cognitive function. Visit www.clinicaltrials.gov for more information.

Vitamins and Antioxidants

Many studies have examined the effects of antioxidants, especially vitamins E and C, on memory and cognitive function.

Vitamin E (alpha-tocopherol) is lipid-soluble and interacts with cell membranes, traps free radicals, and disrupts the pathway that leads to cell damage (Halliwell B et al 1985). It has also demonstrated (in animal models) the ability to reduce degeneration of hippocampal cells after cerebral ischemia (Hara H et al 1990).

One study examined the effect of these two vitamins on older women's performance on cognitive tests. Dietary information was collected from women older than age 70 who were not diagnosed with stroke. A total of 22,213 women were interviewed. Long-term, current users of vitamin E with vitamin C had significantly better performance than women who never used vitamin E or C. Higher mean scores were seen with increasing duration of use. Benefits were less consistent for women taking vitamin E alone. The researchers concluded that the specific use of vitamin E supplements, especially when combined with vitamin C, may be beneficial in maintaining cognitive function during later adult years (Grodstein F et al 2003).

Vitamins B1 and B12. Vitamin B1 (thiamine) is water-soluble and necessary for the metabolism of proteins, carbohydrates, and fats. It has been shown to mimic acetylcholine in the brain (Meador K et al 1993a), which may account for its possible effects in Alzheimer's and other dementias (Meador K et al 1993b; Benton D et al 1995). Thiamine is also involved in nerve transmissions within cholinergic neurons, which are known to deteriorate in Alzheimer's disease.

A one-year study involved 127 young adults given 15 mg thiamine with other B vitamins at dosages ten times the recommended daily allowance. The most significant effect was enhanced cognitive function in women (Benton D et al 1995). Another study involved 80 elderly women given 10 mg of thiamine daily for ten weeks. Compared to the placebo group, those who took thiamine had significant increases in appetite, activity levels, energy intake, and general well-being, as well as improved sleep patterns and decreased fatigue (Smidt LJ et al 1991).

Symptoms of thiamine deficiency are varied and include memory loss, depression, weakness, insomnia, back pain, myalgia, weight loss, hypothermia, constipation, pain sensitivity, and dyspnea. It also manifests as Wernicke-Korsakoff syndrome, mentioned earlier.

Researchers have pursued the possible connection between B12 deficiency and dementia (Carmel R 1994). A review examined correlations between cognitive skills, homocysteine levels, and blood levels of folate, B6, and B12. The authors suggested that B12 deficiency might decrease levels of substances required for the metabolism of neurotransmitters (Hutto BR 1997).

Piracetam: An Overseas Solution

Although not approved by the FDA for use in the United States, piracetam is prescribed in Europe to treat amnesia, dementia, stroke, dyslexia, senility, and other cognitive problems. Developed more than 30 years ago by a Belgian company (UCB Laboratories), it is a derivative of the neurotransmitter gamma-aminobutyric acid and has been shown to restore cell membrane fluidity. At the neuronal level, it modulates neurotransmission and has neuroprotective and anticonvulsant properties (Winblad B 2005). One of its most interesting effects is the ability to promote the flow of information (via increased blood flow) between the right and left hemispheres of the brain in rats (Buresova O 1976). This may also account for piracetam's usefulness in treating dyslexia (Ackerman PT et al 1991).

One study suggests that piracetam may increase cholinergic receptors in the brain. Older mice were given it for two weeks, and the density of muscarinic cholinergic receptors in the frontal cortex was measured. The older mice had 30 percent to 40 percent higher density of these receptors than before taking the drug (Pilch H et al .1998). The jury is still out on whether piracetam is beneficial for dementia or cognitive impairment (Flicker L et al 2001). However, one study using high doses (8 g/day) demonstrated that piracetam might slow the progression of cognitive deterioration of Alzheimer's disease. It seemed to improve recent incident and remote memory (Croisile B et al 1993). For more information on piracetam, visit www.piracetam.info.

LIFE EXTENSION FOUNDATION RECOMMENDATIONS

For amnesia, Life Extension's recommendations are based on the assumption that supplements having beneficial effects on cognitive function and memory will be helpful in amnesia. Supplements that have been shown to boost memory and brain function

include the following:

- **Cognitex with Neuroprotection Complex**—3 capsules in the morning with or without food. This product was formulated by Life Extension Foundation. It contains many of the nutrients listed above, including GPC, ashwagandha, phosphatidylserine, grape seed extract, vinpocetine, and pregnenolone (optional).
- **DHEA**—15 to 75 milligrams (mg) daily, followed by blood testing after 3 to 6 weeks to make sure that youthful levels of this vital hormone are being maintained
- **Ginkgo biloba**—one 120-mg capsule daily
- **Huperzine A**—50 micrograms (mcg) daily
- **Vitamin E**—400 international units (IU) daily
- **Vitamin C**—500 to 1000 mg daily
- **Complete B Complex**—3 capsules daily. This product was formulated by Life Extension Foundation to provide the complete range of B vitamins. Each capsule contains:
 - Thiamin, 100 mg
 - Riboflavin, 50 mg
 - Niacin, 200 mg
 - Vitamin B6, 75 mg
 - Folic acid, 800 mcg
 - Vitamin B12, 1000 mcg
 - Biotin, 600 mcg
 - Pantothenic acid, 1000 mg
 - Betaine free base, 50 mg
 - Choline, 45 mg
 - Inositol, 250 mg
 - Para-aminobenzoic acid, 100 mg
- **Piracetam**—4800 mg daily until memory is restored

AMNESIA SAFETY CAVEATS

An aggressive program of dietary supplementation should not be launched without the supervision of a qualified physician. Several of the nutrients suggested in this protocol may have adverse effects. These include:

DHEA

- Do not take DHEA if you could be pregnant, are breastfeeding, or could have prostate, breast, uterine, or ovarian cancer.
- DHEA can cause androgenic effects in woman such as acne, deepening of the voice, facial hair growth and hair loss.

Choline

- Do not take choline if you have primary genetic trimethylaminuria.
- Choline can cause fishy body odor, excessive perspiration, hypotension (low blood pressure), depression, and gastrointestinal symptoms such as nausea and diarrhea.

Folic acid

- Consult your doctor before taking folic acid if you have a vitamin B12 deficiency.
- Daily doses of more than 1 milligram of folic acid can precipitate or exacerbate the neurological damage caused by a vitamin B12 deficiency.

Ginkgo biloba

- Individuals with a known risk factor for intracranial hemorrhage, systematic arterial hypertension, diabetes, or seizures should avoid ginkgo.
- Do not use prior to or after surgery.
- Avoid concomitant use of ginkgo with NSAIDS, blood thinners, diuretics, or SSRI's.
- Gastrointestinal symptoms (nausea and diarrhea) may occur.
- Allergic skin reactions may occur.

- Elevations in blood pressure may occur.

Huperzine A

- Do not take huperzine A if you have a seizure disorder, cardiac arrhythmias, asthma, irritable bowel syndrome, inflammatory bowel disease, or malabsorption syndrome.
- Huperzine A can cause excessive perspiration, blurred vision, fasciculations (involuntary muscle twitching), dizziness, bronchospasm, bradycardia, arrhythmias, seizures, urinary incontinence, increased urination, excessive salivation, and gastrointestinal symptoms such as nausea, abdominal cramps, diarrhea, and vomiting.

Niacin (nicotinic acid)

- Do not take high doses of nicotinic acid (1.5 to 5 grams daily or more) if you have liver dysfunction, an unexplained elevation in your serum aminotransferase (transaminase) level, active peptic ulcer disease, arterial bleeding, or if you consume large amounts of alcohol.
- Consult your doctor before taking high doses of nicotinic acid if you have a history of jaundice, peptic ulcer disease, gastritis, disease of the liver or bile ducts, gout, kidney dysfunction, or cardiovascular disease (especially acute myocardial infarction or unstable angina).
- Consult your doctor before taking high doses of nicotinic acid if you have diabetes. High doses of nicotinic acid can negatively affect glucose tolerance. Monitor your serum glucose level frequently if you take nicotinic acid and have diabetes.
- Have your doctor monitor your serum aminotransferase level if you take high-doses of nicotinic acid.
- Nicotinic acid may cause flushing, principally of the face, neck, and chest. This flushing is thought to be prostaglandin-prostacyclin mediated. Histamine may also play a role in the flushing.
- Nicotinic acid can cause dizziness, palpitations, rapid heartbeat, shortness of breath, sweating, chills, insomnia, nausea, vomiting, abdominal pain, and muscle pain.
- High doses of nicotinic acid can cause blurred vision, macular edema, toxic amblyopia, and cystic maculopathy.

PABA (Para-aminobenzoic Acid)

- Do not take PABA if you are taking sulfonamides or have a kidney disease.
- PABA can cause anorexia, nausea, vomiting, fever, and rash.

Phosphatidylcholine

- Phosphatidylcholine can cause increased salivation, a metallic taste, headache, drowsiness, and gastrointestinal symptoms such as nausea and diarrhea.

Phosphatidylserine

- Phosphatidylcholine can cause gastrointestinal symptoms such as nausea and indigestion.

Pregnenolone

- Do not take pregnenolone if you could be pregnant or are breastfeeding, or if you have prostate, breast, uterine, or ovarian cancer.
- Do not take pregnenolone if you have a seizure disorder.
- Pregnenolone can cause gastrointestinal symptoms such as nausea and diarrhea.
- Pregnenolone can be converted to steroids such as dehydroepiandrosterone (DHEA).

Vinpocetine

- Do not take vinpocetine if you have a history of allergic or hypersensitivity reactions to any vinca alkaloids.
- Consult your doctor before taking vinpocetine if you take warfarin (Coumadin). Have your international normalized ratio monitored frequently by your doctor if you take vinpocetine and warfarin.
- Consult your doctor before taking vinpocetine if you have low blood pressure (including transient low blood pressure or orthostatic hypotension). Prolonged use of vinpocetine may lead to slight reductions in systolic and diastolic blood pressures.
- Vinpocetine can cause temporary rapid heartbeat, pressure headache, facial flushing, dizziness, insomnia, drowsiness, and gastrointestinal symptoms such as nausea and diarrhea.

Vitamin B1 (Thiamin)

- Consult your doctor before taking vitamin B1 for a thiamin deficiency, lactic acidosis secondary to thiamin deficiency, Wernicke-Korsakoff syndrome, Wernicke's encephalopathy, or Korsakoff's psychosis.

Vitamin B2 (riboflavin)

- High doses of vitamin B2 (riboflavin) may interfere with the Abbott TDx drugs-of-abuse assay.
- Riboflavin absorption is increased in hypothyroidism and decreased in hyperthyroidism.
- If you are taking nucleoside reverse-transcriptase inhibitors, even a mild riboflavin deficiency can increase your risk of lactic acidosis.

Vitamin B6

- Individuals who are being treated with levodopa without taking carbidopa at the same time should avoid doses of 5 milligrams or greater daily of vitamin B6.

Vitamin B12 (cyanocobalamin)

- Do not take cyanocobalamin if you have Leber's optic atrophy.

Vitamin C

- Do not take vitamin C if you have a history of kidney stones or of kidney insufficiency (defined as having a serum creatine level greater than 2 milligrams per deciliter and/or a creatinine clearance less than 30 milliliters per minute).
- Consult your doctor before taking large amounts of vitamin C if you have hemochromatosis, thalassemia, sideroblastic anemia, sickle cell anemia, or erythrocyte glucose-6-phosphate dehydrogenase (G6PD) deficiency. You can experience iron overload if you have one of these conditions and use large amounts of vitamin C.

Vitamin E

- Consult your doctor before taking vitamin E if you take warfarin (Coumadin).
- Consult your doctor before taking high doses of vitamin E if you have a vitamin K deficiency or a history of liver failure.
- Consult your doctor before taking vitamin E if you have a history of any bleeding disorder such as peptic ulcers, hemorrhagic stroke, or hemophilia.
- Discontinue using vitamin E 1 month before any surgical procedure.

For more information see the Safety Appendix

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