

Migraine Headache relief

According to the National Headache Foundation, about 28 million Americans are affected by migraine headaches (National Headache Foundation 2004). Although migraine was recognized by ancient doctors, its cause is still disputed by experts on the disorder. Even today, no single hypothesis has been accepted by conventional science.

Perhaps because of the confusion surrounding the cause of migraines, conventional medicine has not been able to effectively approach this debilitating condition. Fortunately, however, researchers at Life Extension have closely studied migraine headaches and proposed a novel theory that, for the first time, unites the various lines of research. Although the theory, dubbed the Neurohormonal and Metabolic Dysbalance Hypothesis of Migraine, hasn't been tested in clinical trials, anecdotal evidence strongly supports Life Extension's approach, and clinical trials have validated the individual tenets of the hypothesis.

WHAT IS MIGRAINE?

Migraine headaches are usually classified as either common migraine or classic migraine. A classic migraine is preceded by an aura with characteristic visual, sensory, or motor symptoms. Aura usually includes visual abnormalities (e.g., flashes, shimmering, and other hallucinations that seem to migrate through the visual field) and neurological abnormalities such as tingling sensations (Kasper DL et al 2005; Silberstein SD et al 1995b).

Migraine attacks often include features that occur in sequence, including the following:

- **Prodrome.** This stage is marked by a change in mood that begins hours or days before the headache. Symptoms of prodrome include depression, sleepiness, talkativeness, restlessness, or other alterations (Silberstein SD et al 2003).
- **Aura.** Aura is characterized by visual abnormalities, including flashes, shimmering, and other hallucinations.
- **Headache phase.** The headache itself is typically one sided but may affect both sides of the head. It is usually gradual in onset, moderate to severe in pain intensity, throbbing, and worse with physical exertion, and it can last anywhere from 2 hours to 2 days in children and 4 hours to 3 days in adults (Silberstein SD et al 2003; Joubert J 2005). The frequency of migraine attacks is unpredictable (Silberstein SD et al 2003). The headache stage is often accompanied by loss of appetite, nausea, vomiting, sensitivity to light and sound, blurred vision, tenderness of the scalp or neck, lightheadedness, sweating, and pallor (Silberstein SD et al 2003; Silberstein SD 1995a).

Migraines are about three times as common in women as in men (Lim C 2005; Breslau N et al 2001; Lipton RB et al 2001), and they typically begin between the ages of 10 and 40. The frequency of migraine headaches appears to increase with age, with peak frequency in women during their 30s and 40s and in men during their 30s, and then seems to decrease (Henry P et al 1992). Among some women, migraines decrease in severity or disappear entirely during menopause (Silberstein SD et al 2003). Clearly, migraine's close correlation with sexual maturity and menopause in women suggests that steroid hormones are involved in the disease.

Migraine headaches also tend to run in families, and there is a definite genetic predisposition (Kasper DL et al 2005). More than 50 percent of migraine sufferers have a relative who also suffers from migraine (Lim C 2005).

Migraines have frequently been found to coexist with psychiatric disorders (e.g., anxiety and major depression), neurotic personality, stroke, and epilepsy (Breslau N et al 2001; Davey G et al 2002; Breslau N et al 1995). Links between migraine and other disorders, such as cerebrovascular disease, are under evaluation (Breslau N et al 2001).

UNITING THE MIGRAINE THEORIES

Throughout the years, various theories have been advanced to explain what causes migraine headaches. Naturally, each of these theories has been accompanied by a narrow treatment protocol. The problem, however, is that migraine is likely caused by various and overlapping abnormalities. As a result, no single treatment protocol has been uniformly successful, and many migraine patients are unsatisfied with their results.

One of the first theories to explain migraines was the classic theory of vasoconstriction/vasodilation. According to this theory, migraine headaches are caused by the constriction of blood vessels in the brain, followed by dilation (Spierings EL 2003; Deleu D et al 2000). Brain studies during migraine have shown that blood flow to the brain is in fact abnormal, which likely contributes to the

symptoms (Kasper DL et al 2005).

The theory of hyperexcitability builds on the idea of vasoconstriction/vasodilation. According to the theory of hyperexcitability, the brains of migraine sufferers are extra susceptible to normal triggers, such as stress, and the frequency of migraines depends on the level of excitability. An external trigger, such as stress, causes the sudden constriction of the blood vessels in the brain, which launches the migraine headache. The cause of this excitability is thought to be abnormal brain chemistry, especially in the relationship between calcium and magnesium. During periods of excitability, calcium flows from the extracellular fluid to the intracellular space, resulting in vasoconstriction. Therefore, anything that blocks the flow of calcium or restores the balance of magnesium to calcium would be helpful in mitigating migraine. In fact, studies have shown calcium channel blockers, which block the flow of calcium into cells, can successfully prevent migraine attacks (Bartleson JD 1999).

Another theory proposes a derangement of serotonin metabolism and an excess of neurotransmitters (Beckett BE et al 2002). During migraine, serotonin levels are depressed in the brain, and certain drugs, called triptans, that selectively stimulate certain serotonin receptors, have been shown to reduce the symptoms of migraine (Kasper DL et al 2005). This theory is further supported by the fact that melatonin, which is secreted by the pineal gland along with serotonin, is also reduced during migraine, suggesting that the pineal gland is depressed in migraine patients (Claustrat B et al 1989). Finally, high levels of steroid hormones (e.g., estrogen) can interact with the serotonin transport system, further compromising the availability of serotonin.

Other parts of the nervous system, including the sympathetic nervous system, are also implicated in migraines. The sympathetic nervous system is responsible for various functions, including increasing the contractility of smooth muscle and increasing the heart rate. Many of the factors that trigger migraine, such as stress and hormonal changes, also act on the sympathetic nervous system (Kasper DL et al 2005). By the same token, drugs that mimic or enhance norepinephrine (a neurotransmitter in the sympathetic nervous system) can alleviate migraine (Kasper DL et al 2005).

And, as mentioned above, evidence implicates steroid hormonal imbalances in migraine. Many women note that their migraine attacks occur in connection with their menses, and abnormal hormone levels are closely associated with migraine headaches (Recober A et al 2005). This connection will be discussed in greater detail below.

While none of these theories alone can explain migraine headaches, together they provide an excellent framework for understanding the condition and therefore better managing it. According to Life Extension's Neurohormonal and Metabolic Dysbalance Hypothesis of Migraine, migraine headaches are caused by a collection of disorders:

- Malfunctions in the neurohormonal system, including the feedback loop between the hypothalamus, the pituitary gland, and the glands that produce sex hormones
- An imbalance between the parasympathetic and sympathetic nervous systems
- An imbalance between calcium and magnesium
- Improper functioning of the pineal gland

Furthermore, Life Extension's approach to migraine takes into consideration the digestive complaints that frequently plague migraine patients. Many migraine patients report constipation and gastrointestinal upset, which suggests that absorption of nutrients is poor (Diamond S et al 2002).

Migraine Triggers

Several lifestyle factors may trigger a migraine headache (Silberstein SD et al 2003; Peatfield RC et al 1993). Implicated lifestyle factors include the following:

- Lack of sleep
- Consumption of alcohol, especially red wine and beer
- Excessive exercise
- Consumption of foods containing monosodium glutamate or nitrates
- Consumption of other potential food triggers, including chocolates, aged cheese, dairy foods, caffeine, fermented or pickled foods, shellfish, and wheat
- Emotional stress

Use of certain medications may trigger a migraine (Peatfield RC et al 1993); these include the following:

- Birth control pills or conventional hormone replacement therapy with synthetic estrogens and progestins
- Drugs that dilate the blood vessels, such as Viagra® (sildenafil)
- Antimigraine drugs used excessively, which can cause rebound migraine

- Migraines also may be triggered by the overreaction of blood vessels to a variety of factors (Silberstein SD et al 2003; Peatfield RC et al 1993), including the following:
 - Menstruation
 - Fatigue
 - Changes in altitude, weather, or time zone
 - Glaring lights
 - Perfumes or other powerful odors
 - Head trauma

CONVENTIONAL TREATMENT OF MIGRAINE

Generally speaking, conventional treatment of migraine follows one of three approaches—all of which may be used in the same patient. The first is prevention of migraines; the second is stopping a migraine as it is beginning; and the third is to lessen the pain of an ongoing migraine attack. In each approach, certain drugs have been shown to be effective.

In general, migraine patients who suffer from severe, recurrent migraines that are not responsive to acute drugs (e.g., drugs that are used to treat conventional headaches) are candidates for medications that attempt to prevent migraines (Lawrence EC 2004; Silberstein SD et al 2000a). Prophylactic drugs must be taken daily, and there is usually a lag of two to six weeks before the effect is felt (Kasper DL et al 2005). Drugs approved by the Food and Drug Administration to prevent migraines include propranolol, timolol, sodium valproate, methysergide, verapamil, and amitriptyline. The probability of success with any drug is about 50 percent to 75 percent, so it is not unusual for drugs to be rotated until an effective one is found (Kasper DL et al 2005).

Most migraine sufferers, however, rely on drugs that attempt either to abort a migraine once symptoms become apparent or to reduce the pain of an existing headache. Drugs used to abort fledgling migraines or treat the pain associated with migraines include the following:

5-HT (serotonin receptor) agonists (triptans). These agents target serotonin receptors, which results in pain relief (Beckett BE et al 2002). The triptans are currently regarded as either first-line therapy for moderate-to-severe migraine or as rescue management for those patients for whom nonspecific abortive therapies have failed (Silberstein SD 2000a, 2000b).

Ergot alkaloids and ergot derivatives. These agents lead to constriction of the blood vessels within the skull (Silberstein SD 1997), stunt the process of neurogenic inflammation (Silberstein SD 1997), affect neurotransmitter receptors, and lead to arterial and venous constriction (Beckett BE et al 2002; Silberstein SD et al 1995c; Silberstein SD 1997).

Painkillers and painkiller combination drugs. These include over-the-counter painkillers such as acetaminophen and aspirin, as well as prescription headache relief combination medications including Fiorinal (a combination of aspirin, butalbital, and caffeine) and Fioricet (a combination of acetaminophen, butalbital, and caffeine) (Beckett BE et al 2002).

Nonsteroidal anti-inflammatory drugs. These include drugs such as ibuprofen and naproxen sodium (Pfaffenrath V et al 1995).

Opiate analgesics. These drugs are classified as powerful pain medicines. (Rang HP et al 2005).

Antiemetics. These drugs are used as adjunctive agents (in addition to abortive therapies) to alleviate nausea and vomiting associated with the migraine attack or abortive therapies for acute episodes (Beckett BE et al 2002).

Corticosteroids. These drugs may be used when other conventional therapies have failed or are contraindicated (Beckett BE et al 2002; Klapper J et al 1991).

Lidocaine. A 4 percent topical solution provides effective pain relief during acute migraine episodes when applied to the nasal cavities (Beckett BE et al 2002; Maizels M et al 1999; Maizels M et al 1996).

Botox. While the exact mechanism through which botulinum toxin type A (Botox) injections relieve migraine pain and prevent migraines is currently under investigation, it has been hypothesized that Botox works by inhibiting release of transmitters from the pain-sensitive nerve endings (Dodick D et al 2004). Several studies found that Botox is effective in reducing migraine severity, frequency, and associated disability (Gruener G et al 2003; Smuts JA et al 2004; Dodick D et al 2004; Binder WJ et al 2003; Cordivari C et al 2004; Gobel H 2004; Schim J 2004; Behmand RA et al 2003).

What You Have Learned So Far...

- Migraine headaches occur three times more frequently in women than in men. Life Extension's Neurohormonal and Metabolic Dysbalance Hypothesis of Migraine unites for the first time the leading theories that attempt to explain migraine, offering a multifactorial approach to the condition.
- A migraine is a pounding, throbbing headache that can last up to several days in an adult. Common migraines are not associated with any unusual visual symptoms, whereas classic migraines are associated with visual disturbances before the headache; these visual disturbances are known as aura.
- Migraine headaches are treated by conventional medicines that seek to prevent migraines, stop them in their early stages, or lessen their pain.
- Migraines are closely associated with hormonal changes in women, especially with menstruation.

MIGRAINE: THE HORMONE CONNECTION

There is little doubt that migraines are closely related to hormonal imbalance in both men and women. Because migraine occurs most often in women and is closely related to menstruation, most of the clinical studies examining migraines and hormones have been conducted in women. Their findings reveal that a deeply disturbed hormone regulation system is closely linked to the frequency and severity of migraines.

Migraine headaches increase among women after puberty, and many women have migraines that are closely associated with menstruation (Beckham JC et al 1992). Estrogen withdrawal has been described as a trigger for migraine headache, which explains why some women suffer from migraines when estrogen levels are low (Misakian AL et al 2003). However, conventional estrogen replacement therapy with synthetic estrogens is not always able to relieve migraines. It works for some women, but in others, estrogen therapy appears to worsen migraines (Chavanu KJ et al 2002). Similarly, in a recent study examining 17,107 postmenopausal women, migraine was more common among women who were on conventional hormone replacement therapy (Misakian AL et al 2003). Another study noted that it was difficult to predict which postmenopausal women would suffer from worse migraines because of conventional hormone replacement therapy (Hodson J et al 2000). These studies examined women on conventional hormone replacement therapy, which includes strong equine estrogens synthesized from the urine of pregnant mares.

Although researchers are still looking for an explanation, it appears that progesterone levels may help explain the trial data. During a typical monthly cycle of a healthy premenopausal woman, estrogen levels rise during the first part of the cycle. After the egg is released, progesterone levels rise quickly to prepare the uterus for implantation, and the levels of estrogen decline. If implantation does not occur, levels of progesterone and estrogen both decline quickly, and menstruation occurs to prepare the uterus for another cycle. By carefully studying women with menstrual migraine during this cycle, researchers made some interesting discoveries. Essentially, they found that migraines are more severe and disabling during the phase of the cycle when estrogen is dominant and that women with relatively higher levels of progesterone fared better on a headache outcome index (Martin VT et al 2005).

This research suggests that it is not the absolute levels of estrogen that are associated with migraine among women but rather an imbalance between estrogen and progesterone. This theory would also help explain why conventional hormone replacement therapy among postmenopausal women sometimes exacerbates migraine headaches: it is not necessarily the withdrawal from estrogen but perhaps the imbalance between estrogen and progesterone that occurs when postmenopausal women take strong synthetic estrogens during conventional hormone replacement therapy.

Thus, to help balance progesterone and estrogen levels, Life Extension recommends comprehensive hormone testing and, if necessary, hormone restoration with bioidentical hormones that mimic a woman's natural balance of the various estrogens. When used as part of a multifactorial approach, this measure has been shown to help relieve migraine (Dzugas SA et al 2003).

MELATONIN AND OTHER HORMONES

Estrogen and progesterone are not the only hormones involved in migraine headaches. Rather, it appears that in migraine sufferers, the body's regulation of many hormones is abnormal, and each imbalance may contribute to the pathology of migraines. For example, research has shown that the pineal gland in migraine sufferers is depressed, which leads to reduced levels of both serotonin and melatonin during migraine headaches (Claustrat B et al 1997; Claustrat B et al 1989).

Subsequently, several studies have demonstrated that melatonin effectively relieves migraine pain, decreases frequency of migraines, reduces intensity of migraines, and shortens migraine duration (Gagnier JJ 2001; Peres MF et al 2004).

One study, conducted with 23 volunteer participants (21 women and 2 men), found that administration of melatonin at bedtime was well tolerated and resulted in a 100 percent success rate (i.e., none of the patients suffered from migraine afterward). Melatonin was one part of a program that included four components:

- Hormone restoration therapy with bioidentical hormones
- Simultaneous correction of the imbalance between sympathetic and parasympathetic nervous systems and the ratio of calcium to magnesium (Use of calcium in the daytime and magnesium at night reinforces the balance.)
- “Resetting” of the pineal gland through melatonin supplementation (which can be enhanced with the addition of L-theanine if needed)
- Improvement of intestinal absorption by restoration of normal intestinal flora through the use of probiotics

In this study, all the patients had suffered from deficiencies in steroid hormones, especially pregnenolone, before beginning the study. During the course of the study, patients were given complete hormone restoration therapy, including estrogen, progesterone, testosterone, pregnenolone, and dehydroepiandrosterone (DHEA). The researchers concluded that their clinical experience strongly supports the notion that migraine can be managed only when levels of all the basic hormones—pregnenolone, DHEA, testosterone, estrogen, and progesterone (as well as melatonin)—are optimal (Dzuga SA et al 2003).

NUTRIENTS THAT TARGET MIGRAINE

Magnesium. Maintaining a healthy balance between magnesium and calcium is central to Life Extension's approach to migraine. Studies have shown that up to 50 percent of migraine patients suffer from magnesium deficiencies during an acute attack (Mauskop A et al 1998). Magnesium infusions have led to fast and continuous relief of migraine symptoms, possibly by reducing the brain's hyperexcitability (Mauskop A et al 1995; Mauskop A et al 1998). Several double-blind trials showed that oral magnesium supplementation may either reduce the frequency of migraine attacks (Mauskop A et al 1998) or decrease the number of headache days (Wang F et al 2003). These results may be due to magnesium's ability to rebalance the calcium/magnesium ratio in the brain, thus offsetting the excitability caused by excess calcium in the intracellular space.

Butterbur root. Several studies found butterbur root (*Petasites hybridus*) is an effective prophylactic agent for migraine (Diener HC et al 2004; Grossman W et al 2001; Lipton RB et al 2004). In one placebo-controlled study, 33 patients were given 25 mg of butterbur root twice a day, and 27 patients were given placebo. After three months, the patients taking butterbur experienced a reduction of 3.4 attacks per month to 1.8.

While the mechanism by which butterbur exerts its effect in migraine prophylaxis is unknown, it may work through its anti-inflammatory effects and its blockade of calcium channels in vascular smooth muscles (Scheidegger C et al 1998; Thomet OA et al 2001; Brune K et al 1993; Thomet OA et al 2001; Ko WC et al 2001; Wang GJ et al 2001).

A recent randomized, double-blind, placebo-controlled study evaluated butterbur root extract (in doses of 50 mg or 75 mg twice daily) compared with placebo. After 16 weeks of treatment, 68 percent of patients on 75 mg twice daily had a 50 percent or greater reduction in migraine attack frequency, which was significantly better than those using placebo in this study (Lipton RB et al 2004).

Feverfew. Feverfew (*Tanacetum parthenium*) preparations have been studied for migraine prophylaxis in several trials (de Weerd GJ et al 1996; Johnson ES et al 1985; Murphy JJ et al 1988; Palevitch D et al 1997; Pfaffenrath V et al 2002; Pittler MH et al 2004).

An active component of feverfew, chrysanthenyl acetate, is thought to have pain-relieving properties and to inhibit prostaglandin synthetase (Pittler MH et al 2004; Pugh WJ et al 1988). Melatonin is also present in feverfew and may contribute to overall effectiveness of this herb (Murch SJ et al 1997). Feverfew is also thought to have anti-inflammatory effects (Williams CA et al 1995) and seems to inhibit pain transmission and inflammation (Jain NK et al 1999).

Some trials have shown that use of feverfew results in decreased frequency of migraine headaches and diminishes symptoms of

nausea, vomiting, and pain, as well as light and sound sensitivity (Johnson ES et al 1985; Murphy JJ et al 1988; Palevitch D et al 1997; Pfaffenrath V et al 2002).

One of these trials aimed to test a dose-response of a new formulation of feverfew for migraine prophylaxis. A total of 147 patients participated in this randomized, double-blind, placebo-controlled study, which compared the efficacy and safety of three different doses of the new formulation and placebo. For the first 4 weeks, no treatment was given, and the participants' number of migraine attacks was measured. The active treatment or placebo was then given for 12 weeks. While overall, feverfew was not statistically more effective than placebo, the highest dose of feverfew extract administered significantly decreased the frequency of migraine episodes in patients who had at least four attacks during the initial 4-week phase (Pfaffenrath V et al 2002).

Riboflavin. Riboflavin (vitamin B 2) has been used as a prophylactic measure for migraine. An open-label, pilot study of 49 participants (45 with common migraine and 4 with classic migraine) was conducted in Liege, Belgium. Participants were given 400 mg of riboflavin as a single oral dose daily for at least three months. Treatment resulted in mean global improvement of 68.2 percent. It was concluded that high-dose riboflavin may have a role in migraine prophylaxis due to its efficacy, short-term lack of side effects, and relatively low cost (Schoenen J et al 1994).

A follow-up trial studied 55 migraine patients (Schoenen J et al 1998). Riboflavin at 400 mg daily or placebo was given for three months. Statistically significant reductions in frequency of migraine episodes and headache days were observed with riboflavin compared with placebo. The authors concluded that riboflavin was an efficacious, safe, and cost-effective option for migraine prophylaxis (Schoenen J et al 1998).

Another recently conducted, open-label study in Germany found that administration of 400 mg riboflavin daily significantly reduced frequency of migraine headaches and the use of abortive medications after three months and after six months of treatment (Boehnke C et al 2004). The authors concluded that their findings were similar to those of other investigators and that riboflavin was a well-tolerated and effective prophylactic agent for migraine.

Further studies performed in Liege, Belgium, reported that the combination of beta-blockers and riboflavin may augment their clinical efficacy without enhancing adverse events (Sandor PS et al 2000).

Coenzyme Q10. Several studies have demonstrated effectiveness of coenzyme Q10 in reducing the frequency of migraine headaches (Rozen TD et al 2002; Sandor PS et al 2005). A clinical trial of 31 patients reported a significant reduction in the average number of days with migraine after three months of treatment. Migraine frequency also fell significantly, from 4.85 attacks to 2.81. The administered dose was 150 mg daily.

A randomized, double-blind, placebo-controlled trial of 42 patients compared coenzyme Q10 at 100 mg three times a day with placebo. Participants were randomized to either placebo or coenzyme Q10 for three months. Coenzyme Q10 significantly decreased migraine attack frequency (≥ 50 percent reduction) in 47.6 percent of patients, compared with 14.4 percent of patients on placebo. In addition, coenzyme Q10 seemed to decrease headache days and days with nausea better than placebo (Sandor PS et al 2005).

S-adenosyl-L-methionine (SAME). Only one small, open clinical trial (Gatto G et al 1986) of SAME has been conducted to date. It found that long-term administration of SAME could result in pain relief in migraine sufferers. The authors speculated that this relief may be due to SAME's effect on turnover of serotonin, a target in conventional drug therapy.

LIFE EXTENSION FOUNDATION RECOMMENDATIONS

To minimize the frequency of migraine attacks, migraine triggers need to be identified and avoided (Silberstein SD et al 2003; Peatfield RC et al 1993). It is recommended that migraine sufferers stop smoking, get sufficient sleep (but not oversleep because oversleep may serve as a migraine trigger), and minimize stress. Nutrients that may be effective prophylactic agents for migraine include the following:

- **Butterbur root extract** (Petadolex)—50 milligrams (mg) up to three times daily with meals. Dose could be tapered after four to six months of oral administration, then increased again when there is an elevation in migraine incidence.
- **Feverfew extract**—100 mg daily. Formulations of feverfew standardized to 0.2 to 0.35 percent of parthenolide have been used in most studies.
- **Riboflavin** (vitamin B 2)—100 to 200 mg daily with food.
- **Melatonin**—3 to 6 mg daily before bedtime. To maximize the effect and relieve anxiety, combine with 100 to 400 mg of L-theanine.
- **SAME**—generally, between 400 mg and 1600 mg orally daily.
- **Coenzyme Q10**—100 mg up to three times daily. Avoid smoking, because smoking leads to reduction in body stores of coenzyme Q10 (Elsayed NM et al 2001).

- **Magnesium**—magnesium citrate 160-mg capsules at night before bed. Use the maximum dose tolerated without a laxative effect, usually 1 to 4 capsules. In addition, magnesium may provide some relief if taken during the early, vasoconstrictive stages of the headache.
- **Probiotics**—3.5 billion of Lactobacillus group, 1 billion of Bifidobacterium group, and 0.5 billion of Streptococcus thermophilus.

To balance the hormonal abnormalities present in most migraine patients, a complete hormone profile is strongly recommended. This will likely uncover abnormalities that can be corrected with hormone restoration therapy using bioidentical hormones. For more information on bioidentical hormones or hormone blood testing, call 1-800-544-5440, or visit www.lef.org. A complete hormone profile will check levels of major hormones, including pregnenolone, estrogen, progesterone, DHEA, and testosterone. These hormones may then be supplemented as necessary to restore youthful hormone levels and correct any imbalance that might be contributing to migraine headaches. A suggested beginning dose of DHEA is 15 to 75 mg daily.

MIGRAINE 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:

Coenzyme Q10

- See your doctor and monitor your blood glucose level frequently if you take CoQ10 and have diabetes. Several clinical reports suggest that taking CoQ10 may improve glycemic control and the function of beta cells in people who have type 2 diabetes.
- Statin drugs (such as lovastatin, simvastatin, and pravastatin) are known to decrease CoQ10 levels.

Feverfew

- Consult your doctor before taking feverfew if you take warfarin (Coumadin). Feverfew has blood-thinning properties.

Magnesium

- Do not take magnesium if you have kidney failure or myasthenia gravis.

Melatonin

- Do not take melatonin if you are depressed.
- Do not take high doses of melatonin if you are trying to conceive. High doses of melatonin have been shown to inhibit ovulation.
- Melatonin can cause morning grogginess, a feeling of having a hangover or a “heavy head,” or gastrointestinal symptoms such as nausea and diarrhea.

SAMe

- Consult your doctor before taking SAMe if you have bipolar disorder. See your doctor frequently if you take SAMe and you have bipolar disorder.
- Consult your doctor before taking SAMe if you take antidepressants. See your doctor frequently if you take SAMe in place of or in addition to antidepressants.
- Consult your doctor before taking SAMe if you have cancer. Nucleic acid methylation patterns may change in people who have cancer and take SAMe.
- Do not take SAMe if you are undergoing gene therapy.
- SAMe can cause anxiety, hyperactive muscle movement, insomnia, hypomania, and gastrointestinal symptoms such as nausea and diarrhea.

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.

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