

Erectile Dysfunction

Erectile dysfunction (ED) is a serious, life-altering problem for millions of men. A man's inability to achieve or maintain an erection is inevitably linked to complex feelings of inadequacy, frustration, and shaken confidence, which may spill over into other areas of his life. The psychological and quality-of-life consequences of ED must not be underestimated.

Since the introduction of Viagra® (sildenafil citrate) in 1998, several other drugs for the treatment of ED have been introduced; it has become clear that ED is far more prevalent than may have been suspected previously. A study conducted in the Boston area from 1987 to 1989 found that 52 percent of men between the ages of 40 and 70 suffered some degree of ED (Feldman HA et al 1994). By extrapolation, about 30 million men are affected by ED in the United States (McKay D 2004). And given men's ever increasing lifespan, it has been further estimated that the incidence of ED worldwide will more than double in the next quarter century (Goldstein I 2000).

Clearly, Viagra® and drugs like it are blockbusters for their manufacturers. They have improved the quality of life for countless millions of men and their partners who might otherwise have faced either years of continued impotence or unpleasant and possibly unsatisfactory alternatives, such as penile implants or penile injections. But these medications have their drawbacks. Despite their resounding success in the marketplace, ED drugs produce a number of side effects (some serious, such as the small risk of blindness) (Akash R et al 2005), and not all men can take them. For about 30 percent of patients, ED drugs don't work to patients' satisfaction (Sussman DO 2004). This high failure rate has prompted researchers to search for alternatives.

Over the centuries, countless products have been touted as enhancing male vigor and increasing libido. While the effectiveness of many of these substances cannot be adequately proved or disproved, several candidates have demonstrated some degree of efficacy in controlled human trials.

It may also be important for men to test their blood levels of free testosterone and estradiol (an estrogen). As men age, they often suffer from a deficiency of free testosterone while producing too much estrogen. Sexual desire and performance are strongly affected by these hormones. The good news is that there are safe ways of increasing free testosterone and reducing excess estrogen, which can lead to a significant improvement in a man's sexual satisfaction.

ANATOMY OF AN ERECTION

The penis is largely under the control of the central nervous system. The features that allow erection to occur consist of spongy columns of tissue known as the *corpus cavernosum* and *corpus spongiosum*. When these specialized tissues engorge with blood, erection is achieved. This process, however, is quite complicated, involving complex interactions among psychological and physical stimuli and chemical signals, as well as a shifting balance between inhibitory and excitatory forces.

During sexual stimulation, the brain sends signals that result in the release of nitric oxide by parasympathetic neurons in the penis. Penile endothelial cells are also stimulated to release nitric oxide. As nitric oxide diffuses into the smooth-muscle cells lining the arteries of the *corpus cavernosum* and *corpus spongiosum*, it stimulates the activation of an enzyme called guanylate cyclase. This enzyme produces cyclic guanosine monophosphate (cGMP), which prompts the smooth muscles of penile arteries to relax, allowing more blood to flow into the spongy tissues of the penis. Simultaneously, blood return via penile veins is restricted, trapping blood in the organ, resulting in engorgement and erection.

Eventually, cGMP is broken down by phosphodiesterase type 5 enzymes (PDE5). When this occurs, the erection subsides; blood flow returns to normal, and the penis resumes its normal flaccid state. The chain of events leading to erection presents several opportunities for intervention in the treatment of ED. Increasing the availability of nitric oxide is one, while decreasing the activity of PDE5 is another.

Viagra®, Cialis®, and Levitra® (vardenafil), for instance, are selective inhibitors of PDE5. By inhibiting the degradation of cGMP, which is the direct intracellular mediator of the nitric oxide pathway, these drugs promote better erections—but not without side effects and risks. For example, all the drugs in this class are contraindicated for men taking nitric oxide-donor drugs, such as organic nitrates for cardiovascular conditions. Mixing these drugs may result in dangerously low blood pressure. Viagra® may temporarily affect color vision and in rare cases may cause blindness (Akash R et al 2005). And any of these drugs may induce a sustained erection that does not subside after more than four hours—a potentially damaging condition known as priapism. Less-severe side effects commonly associated with this class of drugs include headaches, nasal congestion, and flushing (Gresser U et al 2002).

TESTOSTERONE THERAPY FOR ED

Testosterone is well known as the primary “male hormone.” In aging men with ED, returning testosterone to youthful levels appears to make perfect sense, given testosterone’s association with vigor, libido, and masculinity. However, the actual situation is more complicated.

While it is fairly well established that testosterone plays a role in libido, or sexual desire, its precise contribution to erectile function remains unclear (Martinez-Jabaloyas JM et al 2006; Mikhail N 2006; Traish AM et al 2006). ED occurs in men with normal or moderately low levels of testosterone, so it cannot be concluded that testosterone is the primary modulator of erectile function.

But among men diagnosed with hypogonadism—a condition characterized by abnormally low testosterone—erectile function does, in fact, improve after testosterone supplementation. For these men with ED, testosterone therapy is recommended to maintain secondary sex characteristics and restore erectile function (Bhasin S et al 2006). Recent evidence suggests that “a significant proportion of men [older than] 60 years of age have biochemical hypogonadism” (Caretta N et al 2005).

Testosterone replacement therapy has also been recommended as a second-line approach to treatment of ED when prescription medications alone have failed and when prostate cancer has been ruled out (Mikhail N 2006; Rosenthal BD et al 2006; Yassin AA 2006; Morales A et al 2004). As an added bonus, new data suggest that testosterone tends to reduce inflammation, a chronic condition associated with aging and degenerative processes (Maggio M 2005).

However, it is important to note that because of testosterone’s role in encouraging the growth of certain types of prostate cancer, supplementation with this hormone is not without risk. Among patients with advanced prostate cancer, current therapies are actually designed to suppress testosterone rather than boost it (Altwein J et al 2006; Berges R et al 2006). But testosterone therapy appears to be safe and beneficial for men who are free of prostate cancer.

The bottom line is this: aging men who don’t respond to other ED treatments may benefit from testosterone-boosting therapy. In these patients, restoring testosterone to youthful levels may actually be the key to restoring normal erectile function (Gooren LJ et al 2006).

Testosterone replacement should be explored with caution. It is crucial to rule out prostate cancer before considering testosterone therapy. Once prostate health is established, a physician may prescribe testosterone replacement therapy. But an alternative approach exists that doesn’t require a drug prescription. It is possible to increase circulating testosterone levels by preventing the conversion of testosterone to estrogen in the body.

TESTOSTERONE-BOOSTING SUPPLEMENTS

Certain drugs and supplements can inhibit the activity of aromatase, an enzyme that facilitates the transformation of testosterone into estrogen (Kijima I et al 2006; T’Sjoen GG et al 2005; Eng ET et al 2002, 2003). One such aromatase inhibitor is grape seed extract. It has been shown to be useful in the treatment of estrogen-dependent breast cancer, due to its ability to prevent the conversion of androgens (male hormones) to estrogens. Resveratrol, also derived from grapes, has been found to inhibit aromatase (Wang Y et al 2006). Chrysin is another plant-derived aromatase inhibitor. Although the bioavailability of chrysin by itself is problematic, the addition of piperine greatly increases the absorbability of this testosterone-boosting phytochemical (Khajuria A et al 2002). Finally, zinc may be of benefit in helping to boost testosterone through inhibition of aromatase (Kaya O et al 2006; He F et al 2005).

The hormone progesterone also acts as a 5-alpha reductase inhibitor (Tilakaratne A et al 2000; Schmidt M et al 1998).

SUPPLEMENTS TO FIGHT ED

Arginine. Arginine is an amino acid the body uses to produce nitric oxide, which relaxes smooth muscle, thus allowing for increased blood flow in many parts of the body. This action may explain why ED is more common in men with forms of vascular disease in which disorders of nitric oxide play a role, such as ischemic heart disease and stroke. For example, 75 percent of men with ischemic heart disease suffer from ED (Kloner RA et al 2003).

Risk factors for ED include conditions such as high blood pressure, abnormally high blood lipids (i.e., elevated low-density lipoprotein cholesterol and triglycerides), obesity, diabetes, and smoking (McKay D 2004). Recently, some US scientists reported that it may be necessary to add aging itself to the list of risk factors that produce vascular dysfunction of the kind associated with ED. “The normal aging process may induce significant global vascular dysfunction (involving the endothelium and the vascular smooth muscle),” wrote scientists published in the *International Journal of Cardiology*. This age-associated dysfunction was judged to occur even in the absence of clinically diagnosed atherosclerosis and was related to alterations in the production of endothelial nitric oxide (Al-Shaer MH et al 2006).

The link between ED and vascular disease is so strong that physicians are advised to consider men who present with ED but no diagnosis of heart disease as undiagnosed cardiovascular patients until proven otherwise (Jackson G et al 2006). It is believed that alterations in the availability of vascular endothelial nitric oxide represent the common thread linking these interrelated pathologies (Sullivan ME et al 1999).

Arginine has been shown to improve ED and other nitric oxide–dependent conditions, including atherosclerosis (Napoli C et al 2006). One recent study of healthy people who took sustained-release arginine showed that a moderate dose improved endothelial function and blood pressure. Blood pressure reductions, especially in patients with borderline or frank hypertension, occurred after just one week of L-arginine therapy (Miller AL 2006).

In a small human trial, 40 percent of men who received 2.8 g arginine daily for two weeks experienced improvement in their ED symptoms, compared with no improvement among patients receiving placebo (Zorgniotti AW et al 1994). In a larger, double-blind, placebo-controlled trial using high-dose arginine (5 g daily) for six weeks, 31 percent of patients taking arginine reported significant improvements in sexual performance, compared with only 12 percent of controls. Researchers noted that all patients who responded to treatment had initially been shown to have low urinary nitrite and nitrate levels. Among ED patients who responded to arginine treatment, levels of these vascular nitric oxide indicators doubled by the end of the study (Chen J et al 1999).

Arginine and pycnogenol. Several studies have examined the effects of arginine combined with a botanical remedy, such as yohimbine or pycnogenol. Derived from the bark of the French maritime pine, pycnogenol is actually a group of compounds that have been shown to stimulate the activity of the enzyme responsible for converting arginine to nitric oxide. The combination of pycnogenol and arginine is believed to work synergistically to improve vascular endothelial function and presumably erection dynamics.

A Bulgarian study of 40 men appears to have demonstrated this effect. Participants received 1.7 g L-arginine daily for the first month of the three-month study. During the second month, 40 mg pycnogenol was added to this regimen. The pycnogenol dose was increased to 120 mg daily during the third month. After the first month, just five percent of participants experienced normal erection. This increase was statistically insignificant, but after the second month, with the addition of pycnogenol, a remarkable 80 percent of men experienced normal erection. This success rate improved further, to 92.5 percent, after the increase in pycnogenol dose during the third month (Stanislavov R et al 2003).

Yohimbine. Yohimbine (also known as yohimbe) has been used for the treatment of ED for more than 70 years. Derived from the bark of an African evergreen tree, yohimbine is regulated as a drug in some countries, where it is pharmacologically classified as an alpha-2-adrenergic receptor antagonist. As such, it blocks brain receptors involved in releasing norepinephrine in the genitals. Since norepinephrine is the principal neurotransmitter involved in the vascular smooth muscle contraction that reduces penile blood flow, ending an erection, blocking norepinephrine receptors may help prolong tumescence (swelling or enlarging) (Andersson KE et al 2001).

Animal studies have indicated that yohimbine enhances sexual performance (Clark JT et al 1985; Morales A et al 1982), while human studies have demonstrated that yohimbine is more effective at relieving ED than placebo (Vogt HJ et al 1997; Mann K et al 1996; Morales A et al 1987; Reid K et al 1987). In the late 1990s, British researchers conducted a comprehensive review of all controlled clinical trials of yohimbine for the treatment of ED in humans. They evaluated only studies that were randomized, double-blind, and placebo-controlled, with sufficient statistical analysis. This meta-analysis considered seven such trials, and its authors concluded, “Yohimbine is believed to be a reasonable therapeutic option for ED that should be considered as initial pharmacological intervention” (Ernst E et al 1998).

The researchers also noted that yohimbine is well tolerated. Serious adverse events were rare and reversible. Side effects included elevated blood pressure and induction of anxiety at higher doses, but, wrote the British scientists, “The benefit of yohimbine medication for ED seems to outweigh its risks.”

More recently, researchers have investigated the merits of combining yohimbine with a nitric oxide enhancer. One such trial combined yohimbine and arginine. The double-blind, placebo-controlled, three-way crossover, randomized clinical trial enrolled 45 men who received either 6 g arginine hydrochloride and 6 mg yohimbine, or 6 mg yohimbine alone, or placebo, one to two hours before intended intercourse. After drug washout, participants were switched to another regimen, and efficacy was assessed. The “on-demand” combination of 6 g arginine and 6 mg yohimbine was found to be significantly better than placebo at improving erections in men with mild to moderate ED. “It appears to be a promising addition to first-line therapy for ED,” wrote the researchers (Lebret T et al 2002).

In 2005, Scottish researchers investigated the safety of this combination when coadministered with intravenous nitroglycerine in healthy men. One of the major drawbacks of current pharmaceuticals (e.g., Viagra® or Levitra®) for the treatment of ED is their potentially dangerous interaction with nitrate drugs, which are commonly prescribed for heart patients. The research team wondered if the yohimbine/arginine combination avoided this pitfall. Participants received 7.7 mg yohimbine tartrate and 6 g L-arginine glutamate, followed by increasing doses of nitroglycerine. Blood pressure and pulse were carefully monitored. The

yohimbine/arginine combination was found to be “well tolerated and bioavailable in healthy male subjects,” wrote the researchers. “No significant [low blood pressure] interaction with intravenous nitroglycerine was detected at the doses investigated” (Kernohan AF et al 2005).

Panax ginseng. For more than two millennia, *Panax ginseng* has been used by Chinese healers for its tonic and restorative properties. Sometimes called Korean red ginseng, this venerable folk herb is believed to enhance physical performance and promote health and longevity (McKay D 2004). Modern scientists have identified active constituents, especially saponin glycosides known as ginsenosides, which may be responsible for some of ginseng’s antioxidant and health preserving properties. Ginsenosides have been shown to increase the release of nitric oxide in the erectile tissue of laboratory animals, thus demonstrating a potential mechanism of action for this folk remedy in the treatment of organic ED (Choi YD et al 1998, 1999; Kim HJ et al 1998; Chen X et al 1995).

Human clinical trials have demonstrated ginseng’s potential usefulness as a treatment for ED. Korean researchers divided 90 men with ED into three equal groups. Participants received *Panax ginseng*, placebo, or trazodone, an oral antidepressant drug that has occasionally been used to enhance sexual function. Afterwards, participants were evaluated for indicators of healthy erectile function, such as frequency of intercourse, premature ejaculation, and morning erections. None of these parameters changed among the three groups. But participants who received *Panax ginseng* demonstrated a significant improvement in objective and subjective erection parameters such as penile rigidity, girth, duration of erection, improved libido, and patient satisfaction. Serum testosterone levels were unaffected. Overall, the therapeutic efficacy of ginseng on ED was 60 percent, while participants taking trazodone or placebo experienced only 30 percent efficacy (Choi HK et al 1995).

Korean researchers conducted a double-blind, placebo-controlled, crossover study of *Panax ginseng* in 45 men with confirmed ED. Efficacy was evaluated using a number of objective parameters, and hormone levels were monitored for any changes. Participants received either 900 mg ginseng three times daily for eight weeks or placebo. After a two-week washout period, participants were switched to the opposite regimen. Scores on a standardized index of erectile function were significantly higher in patients treated with ginseng than in those receiving placebo. Participant reports regarding penetration and maintenance of erection indicated that these parameters were significantly better among patients taking ginseng. Overall, 60 percent of ginseng patients rated ginseng as having improved erections (Hong B et al 2002).

Another Korean research team investigated the effects of *Panax ginseng* in laboratory rodents with diabetes similar to human type 2 diabetes. Knowing that oxidative stress plays a role in ED among diabetics, the researchers wondered if the antioxidant effects of ginseng might improve vascular function and ameliorate ED. Eighty-four rats were divided into two groups. One group received ginseng for one month, and the other group received none. Erectile function was measured objectively by electrical stimulation of the nerve innervating the animals’ erectile tissue. Blood work determined the oxidative stress status of the animals’ tissue. Results of these tests demonstrated two things: the erectile tissue of diabetic rats on normal feed exhibited significant oxidative stress and responded poorly to sexual stimulation. Diabetic rats eating ginseng, however, experienced significantly reduced oxidative stress and enjoyed unimpaired erectile function. From these results, researchers concluded that oxidative stress may play a direct role in ED among diabetics. But, they noted, ginseng may “preserve potency” through its antioxidant activity (Ryu JK et al 2005).

Given these results and similar positive results obtained in human and animal studies (Choi YD et al 1999), one researcher remarked, “Although *Panax ginseng* activity is modest in comparison to the current treatments of choice for ED, the possibility of increased erectile capacity, if used in concert with other mediators of nitric oxide production, should be further investigated” (McKay D 2004).

Maca. Maca (*Lepidium meyenii*) is a root vegetable that has been cultivated and consumed in the Peruvian Andes for centuries (Chung F et al 2005). The dried root of this Brassica-family vegetable is a rich source of amino acids and mineral nutrients, such as iodine, iron, and magnesium. Indigenous people have traditionally used maca root for its presumed aphrodisiac properties and its purported ability to enhance fertility (Rowland DL et al 2003).

Animal studies suggest that maca may enhance sexual behavior and other parameters associated with sexual health. In fact, some studies have shown that Maca increases litter size, boosts sperm production, and even protects sperm from damage by stressors such as high altitude, pesticides, and lead (Rubio J et al 2006; Bustos-Obregon E et al 2005; Chung F et al 2005; Ruiz-Luna AC et al 2005; Gonzales GF et al 2004; Cicero AF et al 2001, 2002; Zheng BL et al 2000).

Peruvian researchers conducted a trial that sought to determine whether Maca’s effects on sexual response were attributable to changes in mood or elevations in testosterone. The double-blind, placebo-controlled, randomized, parallel trial lasted for three months. Men ranging in age from 21 to 56 years received either 1.5 g or 3.0 g maca or placebo. They were subsequently evaluated for self-perception of sexual desire. Changes in mood (depression or anxiety) were monitored, in conjunction with changes in testosterone levels.

Improvement in sexual desire was noted after eight weeks of maca therapy. Sex hormones such as testosterone and estradiol did not change. There was no correlation between enhanced sexual desire and either hormone levels or changes in depression or

anxiety ratings. "Maca has an independent effect on sexual desire at 8 and 12 weeks of treatment," wrote researchers. "This effect is not because of changes in either . . . scores for depression or anxiety or serum testosterone and estradiol levels" (Gonzales GF et al 2002).

The same research team followed up this study by more closely examining maca's effects, if any, on reproductive hormones (Gonzales GF et al 2003). Fifty-six healthy men ranging from 21 to 56 years of age received either 1.5 g or 3.0 g maca or placebo daily for three months. Levels of luteinizing hormone, prolactin, testosterone, follicle-stimulating hormone, estradiol, and 17-alpha hydroxyprogesterone were measured at 2, 4, 8, and 12 weeks. Compared to placebo, maca had no significant effect on any hormone. Researchers discussed the significance of these findings: "Treatment with maca may be an interesting alternative, since it improves sexual desire . . . without affecting serum testosterone levels, as has been demonstrated in the present study."

Ginkgo biloba. Ginkgo has also emerged as a possible treatment for ED, especially ED associated with the use of modern antidepressant medications. The newer selective serotonin reuptake inhibitor (SSRI) class of antidepressants, in particular, has been associated with a relatively high degree of sexual dysfunction. While depression itself is often associated with decreased libido, modern treatments for depression may add to the problem. For instance, a recent study conducted in Europe estimated that about one third of all patients taking SSRI antidepressants suffered from some degree of drug-induced sexual dysfunction (Williams VS et al 2006). It has been proposed that this effect is mediated in men by drug-induced increases in the amount of serotonin in the central nervous system, which in turn inhibits physiological mechanisms involved in penile erection (McKenna K 1999).

An open trial of ginkgo for the treatment of antidepressant-induced sexual dysfunction concluded that an impressive 76 percent of men experienced improved sexual function after taking 120 to 240 mg ginkgo extract daily for one month. “*Ginkgo biloba* generally had a positive effect on all four phases of the sexual response cycle: desire, excitement (erection and lubrication), orgasm, and resolution (afterglow),” wrote the authors. They speculated that this effect might be caused by ginkgo’s ability to improve dilation of peripheral blood vessels or to modulate central serotonin receptor factors (Cohen AJ et al 1998).

Icariin. This glycoside is found in horny goat weed. It has been studied for its ability to enhance erections and improve sexual performance by affecting nitric oxide synthesis. In one animal study, icariin administered to castrated rats improved a number of measures that are connected to erectile health, including nitric oxide levels and intracavernosal pressure (Liu WJ et al 2005). These results have been supported by additional studies that have found that icariin can improve intracavernosal pressure and thus enhance erectile quality (Tian L et al 2004).

Testofen. Testofen is a standardized extract of fenugreek. A number of animal studies conducted by Gencor Pacific have shown that Testofen can raise testosterone levels more than placebo can and to a degree comparable to the prescription drug Viagra®. In another animal study conducted by Gencor, Testofen produced activity almost equivalent to testosterone in castrated rats. The product is under investigation in human beings.

Tribulus terrestris and DHEA. ED has been associated with declining levels of dehydroepiandrosterone (DHEA), an important hormone that tends to be depleted steadily with age (Basar NM et al 2005; Feldman HA et al 1994). Studies have shown that supplemental DHEA may be helpful in relieving symptoms of ED in patients with initially low DHEA levels (Reiter WJ et al 1999, 2001).

An increase in bioavailable DHEA may underlie the efficacy of another herbal remedy for impotence, *Tribulus terrestris*. Also known as puncture vine, *Tribulus* contains the active ingredient protodioscin, which is reportedly converted to DHEA in the body (Adimoelja A 2000). This DHEA-boosting activity may account for puncture vine’s reputation as an aphrodisiac in its native Europe and Asia. While some animal studies appear to confirm the ability of *Tribulus* to improve sexual functioning, no reliable human trials have taken place (Gauthaman K et al 2002, 2003; Adaikan PG et al 2000).

WEIGHT LOSS: RESTORING FUNCTION

Lifestyle changes may be sufficient to reverse ED, especially when ED is associated with obesity. Epidemiological studies indicate that physical activity and leanness are associated with a reduced risk for ED. A study published in the prestigious Journal of the American Medical Association found that about one-third of obese men with confirmed ED were able to improve their sexual function after losing weight and increasing physical activity over the course of two years (Esposito K et al 2004).

In a randomized trial involving 110 obese Italian men, test participants lost more than 10 percent of their initial body weight and roughly quadrupled the amount of time they spent exercising each week. Serum markers of inflammation, including C-reactive protein and interleukin-6, also decreased significantly. After two years, about one third of these initially obese participants reported significantly better sexual function than did control participants, whose weight and exercise levels remained roughly constant (Esposito K et al 2004).

Of course, this study also shows that lifestyle changes may not be feasible, or adequate, to reverse established ED in all cases. Fortunately, some safe, natural alternatives are available whose clinical data support their benefits for sex and health.

A WORD OF CAUTION

Some so-called aphrodisiacs may actually do more harm than good. “Spanish fly” is a case in point. Derived not from a fly but from the blister beetle, Spanish fly, or *cantharidin*, is far more likely to cause painful poisoning than pleasurable erection. It should be avoided (Sandroni P 2001; Karras DJ et al 1996).

LIFE EXTENSION FOUNDATION RECOMMENDATIONS

ED is a very serious condition that can gravely affect a man's quality of life. In many cases, however, ED has physical causes that can be remedied through lifestyle changes and supplementation. Men who are overweight may find some relief through weight loss. For more information on male weight loss, please see the chapter titled Obesity.

In addition, a number of supplements may help:

- **L-arginine**—2 to 9 grams (g) daily
- **Yohimbine**—6 mg daily
- **Ginseng**—900 to 2700 mg daily
- **Maca extract**—3 g daily
- **Ginkgo biloba**—120 mg daily
- **Zinc**—30 to 50 mg daily
- **Grape seed extract**—100 mg daily
- **Chrysin**—1400 to 2800 mg daily
- **Icariin**—80 mg daily
- **Testofen**—500 mg daily
- **Saw palmetto**—320 mg daily
- **Nettle extract**—240 mg daily
- **DHEA**—50 to 75 mg daily. A DHEA blood test after 3 to 6 weeks can enable one to optimize individual dosing.

It is also recommended that men test their hormone levels to see if an underlying hormonal deficiency may be contributing to their ED. If testing is conducted, it is important to note that so-called normal levels of testosterone for older men reflect simply averages in the current population. Life Extension believes that most aging men would not prefer to accept the loss of youthful vigor as “normal.” Instead, the Foundation suggests that a more valid “optimal” range for all men would be in the upper one-third of the range for men aged 21 to 49 and that any supplementation treatment should aim to restore hormone levels to that range. In addition, testing for estrogen levels is also recommended, with an effort to reduce estradiol to below 30 pg/mL. It is important, however, that men not begin testosterone therapy unless prostate cancer has been ruled out. For more information on male hormone therapy, please see the chapter titled Male Hormone Modulation.

Because of the close association between ED and heart disease, men with ED who have not been diagnosed with heart disease are encouraged to seek diagnostic testing to detect the possible presence of heart disease.

PRODUCT AVAILABILITY

All the nutrients and supplements discussed in this section are available through the Life Extension Foundation Buyers Club, Inc. For ordering information, call anytime toll-free 1-800-544-4440, or visit us online at www.LifeExtension.com.

The blood tests discussed in this section are available through Life Extension National Diagnostics, Inc. For ordering information, call anytime toll-free 1-800-208-3444, or visit us online at www.LifeExtension.com.

Erectile Dysfunction 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:

Chrysin

- Do not take chrysin if you have prostate cancer.
- Chrysin can increase the effects of aromatase inhibitors such as aminoglutethimide, anastrozole and letrozole.

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.

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.

Ginseng

- Consult your doctor before taking ginseng if you have high blood pressure. Overuse of ginseng can increase blood pressure.
- Consult your doctor before taking ginseng if you take nonsteroidal anti-inflammatory drugs (NSAIDs) and/or warfarin (Coumadin). Taking NSAIDs or warfarin with ginseng can increase the risk of bleeding.
- Consult your doctor before taking ginseng if you have diabetes. Taking ginseng can cause an extreme drop in your blood glucose level.
- Ginseng can cause breast pain, vaginal bleeding after menopause, insomnia, headaches, and nosebleeds.

L-Arginine

- Do not take L-arginine if you have the rare genetic disorder argininemia.
- Consult your doctor before taking L-arginine if you have cancer. L-arginine can stimulate growth hormone.
- Consult your doctor before taking L-arginine if you have kidney failure or liver failure.
- Consult your doctor before taking L-arginine if you have herpes simplex. L-arginine may increase the possibility of recurrence.

Saw Palmetto

- Consult your doctor before taking saw palmetto if you have any form of cancer that is stimulated by hormones.

Zinc

- High doses of zinc (above 30 milligrams daily) can cause adverse reactions.
- Zinc can cause a metallic taste, headache, drowsiness, and gastrointestinal symptoms such as nausea and diarrhea.
- High doses of zinc can lead to copper deficiency and hypochromic microcytic anemia secondary to zinc-induced copper deficiency.
- High doses of zinc may suppress the immune system.

Yohimbe

- Do not take yohimbe if you have heart disease, kidney or liver problems, or a history of ulcers.
- Do not take yohimbe if you have panic disorder, posttraumatic stress disorder, or Parkinson's disease, since yohimbe tends to increase anxiety and trigger panic attacks.
- Yohimbe can cause agitation, anxiety, sleeplessness, tremors, dizziness, headache, queasiness, vomiting, elevated blood pressure, and fast heartbeat.

For more information see the Safety Appendix

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