3.2.2.4.3 Testosterone

3.2.2.4.3.1 Do phytoestrogens make men impotent?

Phytoestrogens, together with synthetic substances released into the environment, are grouped together as environmental estrogens. While phytoestrogens have been around longer than mankind, the awareness for environmental estrogens, including phytoestrogens, is a rather new phenomenon. Attention was brought to environmental estrogens by biologists who noticed that the males of a number of species living in a highly polluted environment experienced a marked decline in fertility as well as a lack of development of the primary sex organs.

Please note that “estrogens” is a term that groups together a range of natural and artificial hormones that have a feminizing effect. The human primary estrogen is estradiol. Estrogens in placental mammals cause “estrus”, which is the scientific term for “heat”.

Androgens are so called male sex hormones. The primary androgen is testosterone, but some of its derivates, such as dihydrotestosterone, also are grouped under the androgen umbrella (please note that estradiol is also a testosterone derivate, though certainly not an androgen; by means of the enzyme aromatase, the body also converts testosterone into estradiol).

There is plenty of evidence that environmental estrogens are harming the males of some species, and some scientists suspect that they are also responsible for the declined sperm counts of human males in the Western world.

While synthetic environmental estrogens have had a massive degenerative effect on the males of species living in a highly polluted environment, the effect of phytoestrogens has been rather subtle. However, the negative impact on male sexual function is nevertheless measurable.

Red clover, for example, contains comparatively strong phytoestrogens. As cattle farmers have learned from experience, and as has been proven by science, herds that are fed on red clover fields will experience a significant decline in fertility because the pythoestrogens of the red clover interfere with the hormonal balance of the bulls.

Plant oestrogens; the cause of decreased fertility in cows
Authors: Kallela K, Heinonen K, Saloniemi H
Published in: Nord Vet Med, 36(3-4): 124 9 1984

During the stall feeding period 1982-1983, it was established that serious fertility disturbances, indicating oestrogenic stimulation, had occurred in a herd of cattle in an area supported by the College of Veterinary Medicines ambulatory clinic. Whilst investigating the cause of these disturbances it was proven that the silage administered during this period was prepared almost entirely from pure red clover aftergrowth. The oestrogenic isoflavone content of the silage (liquid chromatography examination) and the oestrogenic strength (bioassay) were considerably great. When feeding with the fodder was subsequently discontinued the disturbances ceased to occur and the cows became pregnant more easily. On the basis of the aforementioned incidences it was ascertained that plant oestrogens were almost certainly the cause of the fertility disturbances.

In nature such things don't happen accidentally but are a result of evolution and natural selection. Obviously, containing phytoestrogens is a, however slight, advantageous mutation over the absence of phytoestrogens, as phytoestrogens somehow keep the population of predators, in this case mammalian herbivores, at bay. In principle, it is the same mechanism that has made many plants outright poisonous, and the majority of the rest unfit for human consumption. Therefore, phytoestrogens are an intended (by nature) interruption to the hormonal balance of the males of herbivorous species.

Of course, the attempt of phytoestrogenous plants to disrupt the procreation of mankind has, by and large, been a failure. Male mammals, including humans, have long adapted to the fact that a large number of foods contain phytoestrogens. Evolution, after all, is a game not just of mutation, but one of mutation and adaptation.

Nevertheless, we (the human males) haven't adapted completely to the presence of pythoestrogens in plants, and they still exert some negative influence on male sexual function and male genital size.

Take, for example, a diet in which meat is replaced by ground and baked soybeans, a phytoestrogenic agricultural plant widely consumed in Asia.

Effects of replacing meat with soyabean in the diet on sex hormone concentrations in healthy adult males
Authors: Habito RC, Montalto J, Leslie E, Ball MJ
Published in: Br J Nutr, 84(4): 557-63 2000

A randomised crossover dietary intervention study was performed to evaluate the effects of replacing meat protein in the diet with a soyabean product, tofu, on blood concentrations of testosterone, dihydrotestosterone, androstanediol glucuronide, oestradiol, sex hormone-binding globulin (SHBG), and the free androgen index (total testosterone concentration / SHBG concentration x 100; FAI). Forty two healthy adult males aged 35 62 years were studied. Diets were isoenergetic, with either 150 g lean meat or 290 g tofu daily providing an equivalent amount of macronutrients, with only the source of protein differing between the two diets. Each diet lasted for 4 weeks, with a 2 week interval between interventions. Fasting blood samples were taken between 07.00 and 09.30 hours. Urinary excretion of genistein and daidzein was significantly higher after the tofu diet (P < 0.001). Blood concentrations of sex hormones did not differ after the two diets, but the mean testosterone:oestradiol value was 10 higher (P = 0.06) after the meat diet. SHBG was 3 higher (P = 0.07), whereas the FAI was 7 lower (P = 0.06), after the tofu diet compared with the meat diet. There was a significant correlation between the difference in SHBG and testosterone:oestradiol and weight change.

Adjusting for weight change revealed SHBG to be 8.8 higher on the tofu diet (mean difference 3 (95 CI 0.7, 5.2) nmol/l; P = 0.01) and testosterone:oestradiol to be significantly lower, P = 0.049). Thus, replacement of meat protein with soyabean protein, as tofu, may have a minor effect on biologically active sex hormones, which could influence prostate cancer risk. However, other factors or mechanisms may also be responsible for the different incidence rates in men on different diets.

While environmental estrogens are considered negative in almost any context, phytoestrogens have been credited with helping women when they enter menopause, and are said to have a protective effect on the heart and guard against some cancers, such as prostate cancer. They do so by lowering testosterone levels.

Estrogens and androgens compete in the male, and female, body for the same receptors. One cannot play with estrogens without messing with androgens, primarily testosterone. So, when estrogens are enhanced (in men and women), for example by the consumption of dietary supplements or a diet that includes tofu instead of meat, then automatically, androgens (testosterone) are suppressed.

Do I want to protect my heart Certainly. Do I want to guard against cancers Sure. But do I want to suppress androgen tone. Clearly not.

Pythoestrogens aren't the only answer if one intends to do something to protect one's heart. Avoiding tobacco and pursuing some physical exercise every day goes a long way in the same direction, without messing with a man's sexual function.

And there are non estrogenic micronutrients that reduce the overall incidence of cancers, such as the mineral selenium (found, for example, in nuts, most of all in Brazil nuts).

For a man who suffers from early prostate cancer, or a woman with breast cancer, it makes sense to switch to a phytoestrogenic diet. But to recommend a phytoestrogenic diet for all men because it statistically lowers the occurrence of prostate cancer is going one step to far.

Testosterone is not bad per se. Willfully lowering testosterone tone brings with it a plethora of negative side effects, such as loss of sex drive, worse erectile function, feeling less energetic overall, a loss of lean body mass, and more.

For me, the negative impact phytoestrogens have on libido is the most disturbing factor.

Sometimes, scientific, or rather: commercial, medicine doesn't make sense. On the one side, you have docs and public health officials promoting soy protein or other phytoestrogens because they are heart protective and guard against some cancers (by lowering testosterone), and on the other side (or is it the same side), you have physicians promoting testosterone replacement therapy for men who enter andropause (a phase in a man's life when testosterone levels naturally decline).

Well, I've made my choice, and it's pro testosterone.

At that point, there are two options, supplying exogenous testosterone, or increasing the body's own synthesis of testosterone.

Exogenous testosterone can be supplied through testosterone patches or testosterone cream, or through special oral testosterone preparations such as Andriol capsules. But supplying therapeutic amounts of exogenous testosterone invariably will result in a shutdown, or near shutdown of the body's own testosterone production, and this will lead to testicular and penile athrophy (a wasting away of testicular and penile tissue).

Bodybuilders who use synthetic anabolic steroids may present with an enlarged biceps or pectoralis, but they almost always pay for it with a shrinking of their penises and testicles (and that doesn't look attractive when they take off their clothes, in spite of looking attractive when exposing other parts of their bodies).

The better option is to stimulate a man's own testosterone production. There are a number of advantages to this approach. One is that it will keep a man's primary testosterone production site, the Leydig cells of the testes, busy.

This can be achieved, for example, with the Southeast Asian herbal tongkat ali, though one should be careful to obtain it from an Indonesian or Thais, not a Malaysian source.

Malaysian tongkat ali often is heavily diluted (in spite of claims of being highly concentrated), simply because tongkat ali is a protected plant in Malaysia and no longer can be obtained from forests in that country.

Tongkat ali is supposed to work on the whole hypothalamic pituitary testicular endocrine axis. This is effective because a person's testosterone levels are determined by a rather complicated negative feedback mechanism, designed by evolution to keep testosterone levels at genetically determined levels.

When the hypothalamus is signaled that testosterone levels are above the genetically determined levels, it will reduce the release of gonadotropin releasing hormone into the pituitary; the pituitary will respond be releasing less gonadotropins (luteinizing hormone and follicle stimulating hormone) and thereby put a break on Leydig cell production of testosterone.

If men with healthy testosterone levels use pharmaceutical testosterone dosages designed for men with a clinically low testosterone level, they will achieve nothing. Their overall testosterone levels will not rise, because the hypothalamus and pituitary gland, which become aware of the fact that normal testosterone levels have been achieved through exogenous testosterone, will just signal the testes to stop synthesizing testosterone. Then, testosterone levels remain normal, but the testes and penile tissue waste (atrophy) due to inactivity.

There are only two ways to raise testosterone levels beyond genetically set levels.

One is to hugely overdose exogenous testosterone. Then, plasma levels are supranormal even when the body's own testosterone production is completely shut down over a lengthy period of time.

This is the standard approach of bodybuilders who are after testosterone's anabolic effect. As mentioned, they pay for their overall muscular appearance with ridiculously small testes and a penis that has shrunk to boyhood size.

The other option is to switch one's testes into increased production, for example through tongkat ali extract (a single effective dose of tongkat ali active ingredients would be the equivalent of about 100 gram tongkat ali root; such amounts of active ingredient can realistically only be obtained through the use of an extract). This approach will not only generate testosterone levels that are above genetically set levels but also contribute to an increased size of the testes and the penis. If the aim is to raise testosterone levels for the purpose of better sexual function, then the second option is superior.

This is the case because the effect of oral tongkat ali extract supplementation is not just to raise levels of testosterone, but those of related hormones as well. This is not achieved through exogenous testosterone, which is why bodybuilders who use testosterone or synthetic anabolic steroids often not only suffer from an atrophy of the testes and penile tissue, but a loss of libido and sexual functionality as well.

While all phytoestrogens are considered disruptive for male sexual function, some phytoestrogenic herbal extracts, such as damiana, are considered aphrodisiacs for women.

The following chain of thought has been offered to explain this phenomenon: the phytoestrogens of such herbal extracts occupy estrogen receptor sites of the female body. These receptor sites are thereby closed to the woman's own estrogens. The woman's own estrogens are considered stronger than phytoestrogens. That estrogen receptor sites are occupied by weak phytoestrogens instead of the woman's own stronger estrogens tilts the balance between androgens and estrogens in favor of androgens. As the theory goes, the women will therefore feel more sexual appetite.

In men, damiana will likely have the opposite effect. Weak phytoestrogens such as damiana do not only bind to estrogen receptor sites but also to testosterone receptor sites, so that in men, they cause overall testosterone levels to decline.

3.2.2.3.3.2 Testosterone modulation

An interesting recent medical study has come to the conclusion that all successful treatments of erectile dysfunction result in increased testosterone levels.

The study under the title “Lack of sexual activity from erectile dysfunction is associated with a reversible reduction in serum testosterone” was authored by EA Jannini et al. at the Department of Experimental Medicine, University of L’Aquila, Italy. In their abstract, they reported:

The role of androgenic hormones in human sexuality, in the mechanism of erection and in the pathogenesis of impotence is under debate. While the use of testosterone is common in the clinical therapy of male erectile dysfunction, hypogonadism is a rare cause of impotence. We evaluated serum testosterone levels in men with erectile dysfunction resulting either from organic or non-organic causes before and after non-hormonal impotence therapy. Eighty-three consecutive cases of impotence (70% organic, 30% non-organic, vascular aetiology [etiology; the set of factors that contributes to the occurrence of a disease; red] being the most frequent) were subjected to hormonal screening before and after various psychological, medical (prostaglandin E1, yohimbine) or mechanical therapies (vascular surgery, penile prostheses, vacuum devices). Thirty age-matched healthy men served as a control group. Compared to controls, patients with impotence resulting from both organic and non-organic causes showed reduced serum levels of both total testosterone (11.1 +/- 2.4 vs. 17.7 +/- 5.5 nmol/L) and free testosterone (56.2 +/- 22.9 vs. 79.4 +/- 27.0 pmol/L) (both p < 0.001). Irrespective of the different aetiologies and of the various impotence therapies, a dramatic increase in serum total and free testosterone levels (15.6 +/- 4.2 nmol/L and 73.8 +/- 22.5 pmol/L, respectively) was observed in patients who achieved normal sexual activity 3 months after commencing therapy (p < 0.001). On the contrary, serum testosterone levels did not change in patients in whom therapies were ineffective.

Since the pre-therapy low testosterone levels were independent of the aetiology of impotence, we hypothesize that this hormonal pattern is related to the loss of sexual activity, as demonstrated by its normalization with the resumption of coital activity after different therapies. The corollary is that sexual activity may feed itself throughout the increase in testosterone levels."

Note: mol is the basic International System unit of amount of substance equal to the amount containing the same number of elementary units as the number of atoms in 12 grams of carbon-12. Symbol mol. Also called gram molecule; nmol = nanomol; nano- = one billionth (10 -9); pmol = picomol, pico- = one trillionth (10 -12)

What's interesting about this result is the reversed correlation. Usually, people think that they first need the testosterone, and than have more sex. But to have more sex, according to the above-cited study, is what surely leads to higher testosterone levels. What's first, the egg or the hen.

Low, or at least very low, testosterone levels clearly will lead to decreased libido and erectile dysfunction. Hypogonadal men are an obvious proof.

However, hypogonadism is definitely not the only possible cause of erectile dysfunction. More often, so says conventional medical wisdom, the cause is vascular.

The treatment of erectile dysfunction with testosterone, however, is usually not successful.

A "not successful" result is something quite common for all singular erectile dysfunction treatments… in medical, scientific studies as well as in personal experiments (not only of this author).

Isn't it quite obvious that a combination strategy should be applied?

For philosophical reasons, this bookadvocates a hypersexual lifestyle. It's the only state of being worth living in.

We advocate a frame of mind, characterized by daily being overtaken by sexual fantasies so strong that they result in spontaneous erections, even orgasms.

Pfizer's Blue alone doesn't achieve this. Yohimbine as well as some Parkinson's Disease medications such as bromocriptine come a bit closer to the desired effect.

But with Yohimbine, too, you can't force the issue (or should we say: the tissue … the corpus cavernosum tissue, this is). Or, you can, but only to a certain extend.

We would like to be as hypersexual as the perpetuators of sex crimes, minus the criminal inclination. It has been proven in numerous scientific studies that chemical castration works in suppressing criminal tendencies in sex offenders. Such chemical castration entirely works on a hormonal pathway, by suppressing testosterone.

Reported the Washington Post on March 23, 1998: "An Israeli study recently published in the New England Journal of Medicine reports that a new treatment is highly effective in men with long-standing deviant sexual behavior.

"Thirty men with paraphilia (ranging in age from 24 to 40) were treated with monthly injections of triptorelin, a long-acting drug that blocks the action of gonadotropin-releasing hormone. That is a messenger chemical from the brain that normally stimulates the pituitary gland to release other hormones that control the function of sex organs such as the testes and ovaries.

"Triptorelin treatment produced a dramatic drop in the men's testosterone levels. All of the men reported a reduction in deviant sexual fantasies and desires, from a mean of 48 per week before treatment to zero during treatment. Similarly, incidents of abnormal sexual behavior decreased from a mean of five per month to zero.

“Triptorelin did produce significant side effects. Bone-mineral density decreased in 11 of 18 men in whom it was measured. The treatment causes temporary infertility and shrinkage of the testes. Many men reported difficulty with erections and lack of sexual interest, and some had hot flashes, decreased facial hair and muscle weakness or tenderness.”

There has been ample testifying by offenders that reducing testosterone levels diminishes compulsory sexual ideas.

The following is quoted from an essay by Michael Ross, a death-row inmate convicted for the murder of several women:

“As you might expect, I have been examined by many psychiatric experts since my arrest in 1984. All of them, including the state’ s own expert psychiatric witness, diagnosed me as suffering from a paraphiliac mental disorder called ‘sexual sadism,’ which, in the experts’ words, resulted in my compulsion ‘to perpetrate violent sexual activity in a repetitive way.’

The urge to hurt women could come over me at any time, at any place. Powerful, sometimes irresistible desires would well up for no apparent reason and with no warning. Even after my arrest — while I was facing capital charges — these urges continued.

“I eventually found some relief. Almost three years after I came to death row, I started to receive weekly injections of an anti-androgen medication called Depo-Provera. Three years later, after some liver function trouble, I was switched to monthly Depo-Lupron injections, which I still receive. What these drugs did was significantly reduce my body’ s natural production of the male sex hormone — testosterone. For some reason, testosterone affects my mind differently than it does the average male. A few months after I started the treatment, my blood serum testosterone dropped below prepubescent levels. (It’ s currently 20; the normal range is 260 to 1,250.) As this happened, nothing less than a miracle occurred. My obsessive thoughts and fantasies began to diminish.”

The correlation of increased testosterone levels and sexual thoughts is clear.

So, why has testosterone replacement therapy often been shown to be not effective in restoring sexual function? There are a number of possible reasons. For example the design of the studies.

If the cause of erectile dysfunction is purely vascular, and if the measured effects are just related to cavernosal tissue, not much can indeed be expected.

3.2.2.4.3.3 Personal experience with enhancing testosterone as treatment of sexual dysfunction

I am sure that a healthy man at any age can enhance testosterone. I am not sure that it will do much for his libido and sexual function.

I have tried a good number of hormonal supplements, and, based on my own experience and the scientific studies of others, I would say that it's not the way to go for sexual enhancement. While the dopamine route is not perfect either, medications such as bromocriptine at least have a measurable effect on sexual parameters (if they are applied in the correct manner).

And then there always is yohimbe (or yohimbine, the extracted pharmaceutical), and Pfizer's Blue. Both work.

I haven't had any pro-sexual effect from testosterone undecanoate (Andriol capsules). To the contrary, the supplemental Andriol has probably weakened my own testosterone production. Testosterone supplementation really only makes sense if the body can no longer be enticed to produce own testosterone.

I have taken DHEA for years. Sites that want to sell DHEA will usually include a statement in their sales pitches that DHEA enhances libido or sexual function. I don't think DHEA is counter-effective. But from the perspective of sexual enhancement, it's probably just a strong placebo.

For the whole concept of it, I believe that matters other than supplying hormones or precursors should be emphasized. All hormone supplementation, with Testosterone undecanoate, DHEA or pregnenolone, has not shown to enhance the sexuality of basically healthy men. And enhancing sexuality would be my primary aim anyway.

On the other hand, a combination of clomiphene citrate (Clomid) and anastrozole (Arimidex) results in a character modification that I judge to be testosterone-driven. The clomiphene citrate stimulates the hypothalamus in a manner that, several steps downwards, results in higher testosterone levels. Anastrozole avoids the conversion of testosterone into estradiol.

I have also experimented with tongkat ali, both herbs that is said to increase testosterone levels on a pathway similar to clomiphene citrate.

Especially the tongkat ali has an effect similar to the combination of clomiphene citrate and anastrozole: a testosterone-driven character modification.

In both cases, I feel the testosterone by becoming aggressive more easily. Upon being provoked (for example in road traffic) I could spend minutes on imagining how I would maltreat a fellow driver who obstructed my path. I'd cut his ears, shoot his kneecaps, smash his balls, and drown him in a bathtub of human excrements.

But do I want to become more aggressive? Not really. I'm on the search for real great sex. I'm not a boxer or race driver, both of whom probably need aggressiveness to increase their chances of winning. Nor am I a body builder who would benefit from the anabolic component of an increased testosterone level.

Sadly enough, the character modification I had in mind (a general enhancement of libido and sexual performance) did not occur on any testosterone enhancement schedule.

I, for myself, have to make sense of my personal experience with testosterone-enhancing medications, especially when comparing them with scientific information on the same topic.

My hypothesis (not a theory I would have invented myself) is that in the hormonal system, testosterone levels are not the crucial factor for enhanced libido and sexual performance.

You probably have to have the appropriate receptor sites for the pro-sexual effect of testosterone, and the receptor sites have to be activated.

Apart from trying to be a good lay man, I'm also a layman. And like most laymen studying the endocrine system, I have initially strongly focused on hormones, and neglected the importance of receptors. However, hormones exert their effects not just on tissue of any kind but only on tissue that has a chemical surface which is receptive to the hormone.

I assume that the effects of testosterone on libido and sexual performance are strongly correlated to certain receptor tissue in the brain (for the libido) and the reproductive organ (for erections). If you want to enhance, you will have to enhance both, hormones and receptor tissue.

Let's compare this to a concert ensemble. To start with, I have 4 musicians. That's ok for playing some famous Mozart melodies. But if I want to play a Beethoven symphony, I will have to upgrade. How?

By hiring more musicians? Of course. But if they don't have any instruments to play, they just stand around.

It's just the same as with testosterone.

Sexual intercourse is a concert. The musicians are the testosterone, and the musical instruments are the receptor sites.

All is set and running at the concert hall. They play Mozart melodies. If I take away the musicians, there will be silence (if I totally inhibit testosterone, no sex will happen, and my libido is dead).

However, such an experiment is no proof that liberally sending more musicians onto the stage will make for a full orchestra, capable of presenting that Beethoven symphony. I have to supply the additional violins, drums, and trumpets as well.

But enhancing testosterone (by supplying it exogenously, or by stimulating the hypothalamus, or by inhibiting aromatase) is just like sending empty-handed musicians onto the stage. They can't do much if you don't supply the musical instruments.

On the same level, testosterone can only have a pro-libido and pro-sexual-performance effect if there are specific tissue receptors where it can dock on. On other tissue, testosterone has no effect.

As far as I understand it, I (and probably most other men) don't suffer from a lack of aggression receptor sites that can be influenced by increasing testosterone. With testosterone receptor sites for sexual prowess, however, I seem to have a number of receptor sites that largely correlates to my own testosterone production.

For a sexual effect from enhancing testosterone, both would have to be increased: testosterone AND receptor sites. Sadly enough, I have not come across any medication that claims to increase the number of sexual testosterone receptor sites.

3.2.2.4.3.4 The theory of enhancing testosterone for better sexual function

Testosterone and estrogens are, to a certain extent, interchangeable. Estradiol, the most powerful of the estrogens, is metabolized in the body by the enzyme aromatase from testosterone. Furthermore, estrogens can occupy testosterone receptors. And depending on receptor sites, one and the same hormone can have quite opposing effects. The body furthermore possesses a full arsenal of weapons by which certain hormones can be rendered inefficient. They can be neutralized by proteins, and up and down regulated by other hormones.

Because our knowledge on the endocrine system is far from complete, many scenarios are possible by which intended results are not achieved.

You may, for example, supply exogenous testosterone, and much of it is converted into estradiol You actually end up with a balance that is more tilted towards estrogen than it was when you started the supplementation.

Or you may overly inhibit aromatase, and then scientists find out that what is responsible for libido is not testosterone but specific hormonal receptor sites in the brain. According to such a hypothesis you would gain nothing from testosterone supplementation or aromatase inhibition. The task would have to be to properly activate those receptor sites, a task for which medications have not been developed.

The above two paragraphs are hypothetical scenarios intended to generate a feeling in the reader that the influence of hormones on libido and sexual function is sketchy, and we could be in for surprises.

The proof for the hypothesis that testosterone is the hormone of desire exists primarily as reverse evidence. If you inhibit testosterone, you can reliably kill libido. Nevertheless, the key to a sexualized lifestyle probably is hormonal.

It's just that we don't have reliable information on how hormones determine libido and sexual function. Just one thing is clear: an exogenous supply of testosterone doesn't work by the following formula: a little bit more of exogenous testosterone = a little bit more libido and sexual function; much more of exogenous testosterone = much more libido and sexual function.

That would be too nice and easy to be true.

On the other hand, if you do nothing to interfere with hormonal processes, you can be sure that your libido will likely decline heavily long before you die of natural causes. If it's already largely gone, or clearly on the decline by the time you read this, you may as well experiment with hormonal modulators at this time, even though definite information does not yet exist.

Because the theoretical knowledge about the endocrine system is far from complete, an entirely clinical approach is best. Search for information on what has worked with others, not for theories why something particular should work. (It won't work because it should.)

Currently, your best bet for improving libido and (or by) raising testosterone is probably the Southeast Asian herbal tongkat ali (eurycoma longifolia by its scientific, Latin name). This root has been used as an aphrodisiac in Southeast Asia and in Chinese medicine long before scientific studies conclusively have proven that it raises testosterone. Still, that tongkat ali is used as an aphrodisiac and that it raises testosterone does, strictly analyzed, not mean that it works as aphrodisiac because it raises testosterone. Herbal medications typically have dozens of active ingredients, many of which have not yet been studied at all. It may just be that one component of tongkat ali raises testosterone, and another one works as aphrodisiac.

Nevertheless, I think it's quite obvious that in order to have the sexuality of a 20-year-old at an age far beyond, we have to interfere with the hormonal system. An initial guideline should be that if we want the sexual health of a 20-year-old, we should implement the hormonal mix of a 20-year-old. On the face of it, this would mean: higher testosterone, lower estradiol, less sex hormone binding globulin, less aromatase activity.

Current conventional wisdom indicates that what you primarily want if you are around 50 is more free testosterone.

To achieve higher testosterone levels, a few things have to be kept in mind.

First, the supplementation of pharmaceutical testosterone by itself will often not do the trick. Their will be a tendency that exogenously supplied testosterone will not circulate long as free testosterone but will be bound to sex hormone binding globulin and hereby be rendered inefficient.

Furthermore, the body converts testosterone into estradiol, the strongest of all estrogens. ("Estrogen" is not the name of a hormone but of a group of hormones; the equivalent to estrogen is androgen, not testosterone; like estrogen, the term androgen is the name of a group of hormones; the equivalent to testosterone is estradiol). The conversion of testosterone into estradiol is made by the enzyme aromatase, and the conversion can happen in many different kinds of tissue throughout the body.

There is a definite possibility that supplying the body with exogenous testosterone will not have the desired effect, even if it remains bioavailable (not bound to sex hormone binding globulin), because the body may just convert this testosterone into estradiol. The effect may then even be the opposite of what has been wished for, with the testosterone-estradiol balance more tilted towards the estradiol than has initially been the case.

Prior to testosterone supplementation, aromatase inhibitors should be tried.

They have the power to render ineffective the main culprit in estradiol overload in men, aromatase. The most specific aromatase inhibitor is anastrozole (Arimidex).

But not all testosterone deficiencies are caused by too much aromatase activity. It could just as well be that the Leydig cells in the testicles are not sufficiently stimulated to produce enough testosterone. To stimulate the production of more endogenous testosterone may still be preferential to supplying exogenous testosterone, which often will not have the desired libido-enhancing effect.

The body's own synthesis of testosterone begins deep in the brain, in the hypothalamus. The hypothalamus secretes the gonadotropinreleasing hormone.

This hormone stimulates the pituitary gland to release yet another hormone, luteinizing hormone. This luteinizing hormone stimulates the Leydig cells in the testicles to produce testosterone. In women, it stimulates the ovaries.

The hypothalamus and pituitary gland are not gender specific. And there are a number of drugs that can be used to stimulate the hypothalamus. Clomiphene citrate (Clomid), for example. In women, the medication is used to induce ovulation, and thereby fertility. So fertile do women become on Clomid that roughly one out of 10 pregnancies is for twins.

Body builders who have shut down their own testosterone production by heavy use of anabolic steroids use Clomid to get their testosterone production started again, and, according to common claims, it works quite reliably to this effect.

Clomiphene citrate is a receptor specific hormone modulator. It doesn't only have the cited effect on the hypothalamus but also has anti-estrogenic properties in being a very weak estrogen. By binding on estrogen receptor sites, clomiphene citrate prevents the stronger estradiol from occupying these receptors.

Alas, while the above elaborations on clomiphene citrate suggest that it should work fine to raise testosterone and enhance libido, scientific studies time and again have proven that this drug does not improve libido.

While tongkat ali extract has been shown to be the only medication to have the double effect of raising testosterone and improving libido, there are also a few reliable, non-pharmaceutical methods to boost testosterone (and maybe even libido):

1. Lose weight. Fat cells produce aromatase, which converts testosterone into estradiol, thus keeping the testosterone balance low.

2. Exercise. A balanced program of daily exercise raises testosterone levels.

3. Engage in sexual activity. It has firmly been established that men who have sex more often will thereby raise their testosterone levels.

Whether the three above-mentioned steps will drastically increase libido is less certain. Or rather, it's quite obvious that it's not the grand solution we have been looking for. Enough 20-year-olds who are living a very sedate lifestyle don't have problems with their libido. And enough 50-year-olds who exercise still don't get their libido back on track.

3.2.2.4.3.5 Testosterone – a second opinion

I have recently received a series of emails from a person who apparently has more experience with the use of testosterone for sexual enhancement than I do. That he does have more experience with testosterone is a direct result of his being able to affect sexual enhancement with testosterone medications, something I have so far not been able to achieve. The reader's approach has been rather different from mine.

I read that you did not have good results with testosterone. Testosterone is better than the other things you research if used correctly. I recommend straight testosterone as it is natural and cheap. It is active orally, sublingually, or by injection, but I strongly recommend topically, either suspended in olive oil or any oil or in a gel or in alcohol. It can be rubbed on thin skin or mucous membrane in an area where it will not get rubbed off. One drop may suspend 2 to 4 mg. If you apply it about 2 to 4 times a day, in about 4 days you will notice being quite mentally horny and more spontaneous erections and more sexual fantasy and better performance. This desired state will only last a few days. Then it is desirable to stop for a few days and the whole procedure can work again. It is possible to take a couple drops per day continuously for somewhat constant more sexual arousal but with much less intense peaks. This works identically if not better for women but their dosage can be a bit less.

As you have learned of the erotic potential of jealousy, I suggest you learn the erotic potential in yourself when faced with a very horny woman (created by testosterone). There are some individual variations and other things you will notice but you can discover them all by using this information. Of course the user will also notice a sense of well-being, energy, confidence, and a little more strength and muscle.

In women after a month or two of continuous use, there will be a little more and darker hair coming in and perhaps a slight deepening of the voice and perhaps a bit of acne. At that point one may choose to continue or stop for a few weeks to let those effects subside. There will also be a noticeably bigger and more sensitive clitoris, which is perhaps the most wonderful effect of all.

Fortunately testosterone as a powder or in oil is very cheap. This sexual enhancer can be used compatibly with all your other enhancers. Unfortunately testosterone is a scheduled drug in the US but fairly easy to get by prescription for the mature man. Contrary to all the propaganda it is very safe. Do not hesitate to ask me if you need any more information or run into any problems with this. I guarantee you this will work.

I was very sad to learn from you that Amineptine does not really often create greater euphoria and sex drive and spontaneous orgasms. There is much more research to be done and I respect your effort to share your results. Experimenters in this area are fragmented and not in communication, which slows progress immensely. Briefly, I find that Yohimbine is mildly sexually enhancing but with notable side effects. Pfizer's Blue does work to promote ease of erection and staying hard much as the intracavernously injectable Papaverine and Phentolamine Mesylate.

I find that GHB is a fine euphoric and sexual enhancer when used correctly. MDMA is the best enhancer for love, but GHB is usually better for sex. MDMA interferes with erection and orgasm a lot. The trick [with] MDMA is to be erect before the effects begin and then erection will be maintained without difficulty! You may be aware that MDMA is wonderful and nearly free of side effects at first and with use becomes downright awful in after-effects. It seems the chemists have not found a good way to control these after-effects.

Arginine is a mild sexual enhancer when taken an hour or more previous to sex. DMAE is similarly weak and can feel a bit adrenergic. Lecithin works but is mild. I have not yet experientially learned Bromocriptine or Apomorphine. I do like dopamine agonism for the euphoria and the sexual enhancement.

3.2.2.4.3.6 Testosterone up-regulation, a tricky issue

There is an ample of evidence that a sufficiently high testosterone level is absolutely essential to a sexualized lifestyle. Testosterone, the hormone primarily synthesized in the testicles (but not only there), is necessary both for sexual appetite and performance (e.g. achieving a good erection). Men whose testosterone levels are low will be lacking in both.

But there are oral testosterone medications. The best-known one is Andriol. Andriol is not just testosterone but testosterone undecanoate, a chemical alteration of testosterone of which a small percentage will, in most subjects, make it past the liver into the blood stream.

Animal testicles have a reputation as aphrodisiacs, but this, is based on an overly simplistic logic. If any, there is very limited benefit on the same level on which eating liver benefits anemic patients. Of course, iron is a major mineral in blood, and often anemia is one of iron-deficiency. Eating blood products such as liver will make available to the organism some additional iron to be used in the body's own synthesis of blood, provided it's not a disturbance to this process that is at the root of the iron-deficiency anemia in the first place.

In the US, Andriol is not a licensed medication. For the serious treatment of hypogonadism, it would just be the second-best choice anywhere in the world. It's not only that it is hard to predict how much testosterone of the 40 milligram of testosterone undecanoate each capsule supplies actually makes it into the blood stream. Estimates are 2 to 3 milligram. But there will be variations from person to person, and in a single person from day to day, depending on the diet the liver has to deal with apart from the testosterone undecanoate.

But even in subjects in whom oral testosterone medications do work, such medications are not an aphrodisiac like yohimbine.

Taking for granted that testosterone undecanoate capsules do raise blood levels of free testosterone, the person taking this medication will not feel a sexualisation an hour or so after ingesting the capsules. If raising testosterone levels does work, it seldom does so immediately. There will be a good measure of unpredictability. For men on testosterone replacement therapy (several daily doses over some time), the sexualizing effect will arrive sporadic every now and then. Why it is like this, has, to the best of my knowledge, so far not been conclusively explained.

Testosterone as drug is not a medication of which one would feel much within an hour or so. Unless it is hugely overdosed. And the effect of an overdose is not just a potentiation of the desired effect of a normal dose.

The desired psychological effect of a normal dose may be increased sexual appetite, a good erection that doesn't break down, and a powerful orgasm. Tetosterone-deficient men will be lacking in all three. Testosterone supplementation in testosterone-deficient men will restore all three. But not in the manner that desire, performance, and orgasms will be enhanced an hour or so after popping an oral testosterone medication. It may well take a few days for the effects to set in in the first place. Increased desire may arrive rather surprisingly, not at a moment it was planned for. It's not as direct an effect as for aspirin or yohimbine.

Increased desire is also not a direct correlation to increased testosterone plasma levels. The endocrine system really is rather tricky. And anyway, increased plasma levels only peak a long time after an oral testosterone medication has been applied. For Andriol, they are supposed to occur some five hours after a dose has been ingested.

While the supplementation of regular or even high dosages will normally not be felt within hours after they have been ingested, it's a different story with huge overdoses. Use in women more than in men provides a clear indication to this end. Of course, Andriol, as well as other testosterone products on the market, are not intended for use in women.

But testosterone is the hormone for sexual appetite not just in men but also in women. However, normal testosterone levels in women are much, much lower than in men.

Though not intended for the use in women, women, too, are among those taking Andriol and other testosterone medications. The objective is usually to raise performance levels in sports.

Women on steroids usually exhibit an increased sexual appetite but it's again something that sets in with constant use, not as the effect of a single dose. The most likely effect of a single overdose is extreme anger. It's something more likely to happen in overdosed women, rather than men.

Contrary to the bad reputation of testosterone in this respect, high active levels of this so-called male hormone are not associated with an anger-prone personality, but rather the other way around. High plasma-levels of testosterone will usually give men a positive outlook, make them friendly, understanding, and willing to learn. Abnormally low levels go hand-in-hand with bad moods and depression. As previously mentioned, only extreme overdoses may result in anger, even rages.

Endocrine matters are rather complicated. This is the case because the endocrine system is one of multiple balances by which hormones are kept in check.

Because of this, blood testosterone levels are only an indicator of limited value if one is to judge whether there is enough testosterone activity in one's organism in order to get the best out of sex.

The bioavailability of testosterone is kept in check by a blood-stream protein called sex hormone binding protein (SHBG). Testosterone molecules that have been captured by SHBG are rendered ineffective until they are anyway discarded from the organism.

Test measure overall testosterone, both testosterone which is bound to SHBG as well as free testosterone tests are useless when it comes to assess whether a man should undergo testosterone replacement therapy or not.

The determining factor would be whether a man is sufficient in free, bio- available testosterone.

Plain logic is the smarter approach to decide on the need for testosterone replacement therapy. For all men, the process of becoming older is accompanied by a decline in the synthesis of testosterone.

But not only does the production of testosterone decline. The amount of bio-available testosterone is further reduced by the fact that older men typically "suffer" from increased levels of sex hormone binding globulin.

The decline in bio-available testosterone is largely proportional to all those other symptoms of advancing in age, such as bones becoming brittle, sacking skin, loss of lean body mass, decrease of muscle tone, obesity, and a decline in sexual desire and performance.

This all smells like a ploy of nature to get rid of older specimens of the species homo sapiens by having genetically programmed a decline in the availability of testosterone.

And indeed, there is little doubt that testosterone supplementation not only works to avoid the onset of these events of aging. Testosterone supplementation has the power to reverse these developments.

Older men undergoing testosterone replacement therapy will practically always develop stronger bones and exhibit more lean body mass while carrying less fat. They will usually have a more active and satisfying sex life, though this cannot be measured as easily as lean body mass and fat.

So, why do doctors not recommend that all men from about the middle of their 40's undergo testosterone replacement therapy?

There are a number of reasons why physicians and other health care professionals have been slow in prescribing testosterone replacement therapy.

Testosterone has substantial potential as doping drug in sports. Testosterone supplementation during training periods, long before competition, will lead to increased muscle mass, an asset in many sports. It will also enhance performance during competition, which is why doping controls for testosterone are undertaken at all major events.

The tendency among those who take testosterone supplementation, or use anabolic steroids, is for huge overdoses. And these are obviously not healthy. Therefore, sports and healthcare administrations around the world have run public awareness campaigns to the effect that anabolic steroids, or hormone supplementation sports in general, pose a serous health risk.
Testosterone is not really an anabolic steroid, but it's a steroid hormone with an anabolic effect (the more important component being testosterone's androgenic characteristics),

Sure, mice that are given huge overdoses of testosterone for most of their lives have shorter ones (lives, that is). But we are not talking huge overdoses here. We are talking about high, youthful levels as everyone healthy has them at the beginning of the 20's. And a good number of studies have show that testosterone supplementation to achieve such levels is no major health risk.

Horror stories about dreadful deaths as a consequence of the abuse of synthetic anabolic steroids are good copy but bad science. They don't apply to testosterone supplementation with the aim to achieve youthful levels of the hormone.

Apart from the doping issue. Testosterone has long been associated with two health risk: heart attacks and prostate cancer.

Women up to the age of about 50 have a much lower heart attack risk than men, primarily, so it seems, because they are protected by estrogens. Men with reduced testosterone levels have been shown to be less prone to heart attacks than men with high levels of the male hormone. In spite of this, I for myself, am in favor of a lifestyle of a high testosterone level.

But I take the scientific studies that associate high testosterone levels with heart attacks as a reminder not to expose myself to additional heart attack risks, such as smoking or obesity.

In case of a second health risk, testosterone may have been the wrong suspect for many years. Testosterone has for many years been considered the main culprit in prostate cancer. Actually, not testosterone but its more powerful metabolite dihydrotestosterone.

Testosterone is transformed by the enzyme 5-alpha-reductase into dihydrotestosterone. Actually, dihydrotestosterone is the form of testosterone, which is the major hormonal player in sex matters.

Levels of dihydrotestosterone, not testosterone itself, are what make a difference for the number of orgasms per week. Dihydrotestosterone is also the form of testosterone, which is at work in mediating erections.

And dihydrotestosterone is the form of testosterone that seems to increase the susceptibility for prostate cancer. But the truth of this assessment has already been questioned.

If on has arrived at a decision to undergo testosterone replacement therapy, one will have to decide by which means to supplement testosterone. Most people would feel most comfortable with an oral testosterone drug such as Andriol. Indeed Andriol would be the medication of choice in most countries around the world. But Andriol is not licensed in the US.

And while Andriol is convenient for the patient, there are some disadvantages, both medical and practical. Testosterone undecanoate burdens the liver, an effect that can be avoided by both testosterone injections and transdermal patches, the application of choice in the US.

In most Third World countries, all kinds of prescription medication are sold over the counter. No question asked, no prescription required.

Body builders and pharmaceutical addicts buy their drug supplies during trips to Bangkok for months ahead.

Too bad that in the case of Andriol capsules which they intend to use for testosterone's anabolic properties, what they buy is often enough just empty liver ballast. For while anybody can buy prescription drugs without prescription, it seems that, furthermore, almost anybody also can sell them. Not much of professional qualification seems to be required, and even if qualification is present, it seems to be lacking in the understanding that some medications ought to be stored in the refrigerator. Andriol is among them.

What you find in Southeast Asia are drugstores, many of them not even air-conditioned, where the Andriol lies in the cabinet, at 30 or more degrees, for months on end. The package still says "Andriol" but there is not much testosterone undecanoate left inside the capsules. That Andriol is often considered worthless as steroid among body builders may have its roots in the fact that body builders often will get their Andriol capsules not from an ordinary licensed pharmacy in an industrial country but on the black market or in countries like those in Southeast Asia where a prescription is not needed but appropriate storage is also not guaranteed.