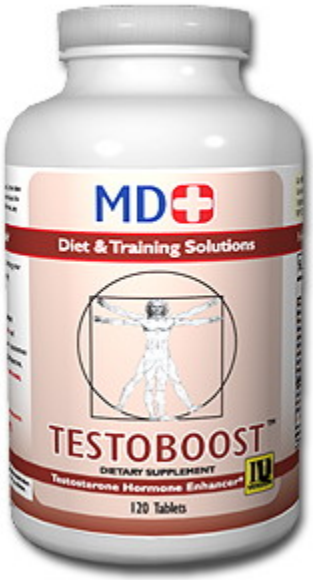
MD+ TestoBoost version IV

Natural Testosterone and Sex Drive Booster



A safe and effective way to dramatically increase your own natural testosterone production.

TestoBoost is by far the most powerful natural testosterone booster, and libido and sex drive enhancer on the market today. Version 4 is the third reformulation of the original TestoBoost, which came out in February of 2000. Each version has been improved by taking into account new research, my own clinical work, and feedback from those who used it.

http://www.mdplusstore.com

The original formula laid down the base for what I wanted to achieve for TestoBoost and that was to naturally increase testosterone levels and sex drive in both men and women. The pathways that I targeted included stimulating all the relevant areas of the hypothalamic-pituitary-testicular axis (HPTA) through several independent mechanisms, and to decrease any potential side effects from increases in estrogen and dihydrotestosterone. Subsequent versions of TestoBoost, while keeping the base intact, added several ingredients that I felt would further increase testosterone levels and sex drive, and further decrease any potential side effects.

The basis of the formula in TestoBoost, and in all the MD+ formulations, is to involve all possible pathways that lead to the desired effects, and to use multiple ingredients that work together to produce superior results. In the case of TestoBoost the desired effect was an increase in basal and elevated testosterone levels in the body, a decrease in counter productive elevations in cortisol, and a salutary effect on overall health, libido and sex drive.

All of this can be accomplished by using ingredients that are known or suspected to boost testicular steroidogenesis, increase sexual desire and performance.

For example, using a variety of ingredients to effectively act as a testosterone sink by trapping testosterone production is the most effective way to increase endogenous androgen production.

In order to do this you have to consider all the possible pathways that are involved in maximizing testosterone production, including:

- Increasing luteinizing hormone (LH) production.
- Increasing the effect of LH.
- Increasing testicular steroidogenesis directly.
- Decreasing inhibitors of steroidogenesis.
- Providing vitamins and minerals that might be frankly or marginally deficient and thus not allowing the full production of testosterone - e.g. **magnesium, zinc, B6.**
- Increasing peripheral formation of testosterone.
- Decreasing peripheral formation of dihydrotestosterone and estrogens or blocking their effects.

As well, other compounds that have been shown to have effects on sexual desire and performance can be used in the mix. On top of this TestoBoost contains **bioperine**, which significantly enhances the bioavailability of supplemented nutrients through increased absorption.

The list of ingredients that could prove useful for increasing testosterone levels is long and includes various vitamins such as **vitamin A, B6 and E, minerals such as zinc, magnesium, manganese, and other ingredients such as arginine, beta ecdysone boron, calcium-d-glucarate, catuaba bark, chasteberry (vitex agnus-castus), chrysin, co-enzyme q10 (ubiquinone), forskohlin, damiana, 5-methyl methoxy isoflavone, deer antler extract, eurycoma longifolia, genistein, GLA, glutathione, prickly pear extract, indole-3-carbinol, ipriflavone, maca root, muira puama, quercetin dihydrate, saw palmetto, schisandra chinensis, stinging nettle extract and tribulus terrestris.**

Why TestoBoost is Better than the Prohormones or Replacement therapy with Testosterone or Anabolic Steroids

Prohormones

In the past decade the big push for those who want “anabolic steroid-like” effects was the prohormones. And even though many have been taken off the over the counter market in the US and other countries, they are still available in one form or another either in North America through the black market, or internationally over the Internet. They range from androstenedione, to the more sophisticated ones that are supposed to be precursors to other anabolic steroids including nandrolone (Deca), boldenone, and even 1-testosterone compounds. The even include steroid like compounds that have various effects on either or both estrogen and testosterone.

The one exception is the legal availability of DHEA. However, while it may be useful for women, and for other purposes, it’s useless for increasing endogenous testosterone

levels in men. Most of the prohormones, including androstenedione, androstenediol Norandrostenediol, norandrostenedione, the boldenone and 1-testosterone precursors, and in fact any precursor prohormones are also relatively useless at providing real androgenic-anabolic effects, but can result in adverse effects.

The trend in the prohormones, as the manufactures get bolder and bolder, is to actually have weaker versions of the commercial anabolic steroids on the supplement market. This is the case with 1-testosterone and the 17-alpha methylated 1-testosterone, which in fact are weak anabolic steroids, with lower androgenic and anabolic effects than the more potent anabolic steroids that have been marketed over the years. Basically the ones being produced now and passed off as over the counter prohormones/steroids are the cast offs of the steroid producing drug industry.

The problem with most of these compounds on the market today is that while they have minimal androgenic and anabolic effects, they have significant side effects, both known and unknown. The prohormones that are being developed today to be used as a substitute for commercial anabolic steroids haven't been investigated or studied for their potential short term or long term side effects.

This is where the real problem with these compounds lies. We don't know what effects they have on various parts of the body including the liver, cardiovascular system, prostate, kidney, and especially on the hypothalamic-pituitary-testicular axis (HPTA). These products would never be allowed as prescription drugs because of the lack of proper animal and more importantly human trials. However, they're blatantly dumped on the market as nutritional supplements, when in fact they're drugs, or more correctly low level, and usually ineffective, anabolic steroids – with less efficacy than the commercial anabolic steroids, but with unknown, and potentially very harmful side effects.

While most of the prohormones and hormones that are available as nutritional supplements are of no use or marginally useful for body composition purposes, they can have significant side effects. One of the side effects that is most troubling is the effect they have on the hypothalamic-pituitary-testicular axis (HPTA), the pathway that's involved in endogenous testosterone production and control.

The worst-case scenario, and one that is common with the use of most of the prohormones, is that there is very little anabolic effect from the compounds themselves, but significant side effects, especially with a dampening effect on the HPTA, which in turn shuts off the production of endogenous testosterone. This HPTA shutdown also occurs with the use of exogenous testosterone and anabolic steroids, and can sometimes result in permanent dysfunction of the normal HPTA, to the point where when these compounds are discontinued (as they invariably are) endogenous testosterone levels remain in the basement, and replacement therapy is sometimes the only solution to achieving normal systemic testosterone levels. In other words they become either temporarily, or sometimes permanently eunuchoid, with a resulting need of testosterone replacement therapy.

Thus, the overall effect is a negative one in that the level of effective anabolic androgens in the body is decreased and the person thus has less anabolic hormones in

their body. On top of that there are several possible side effects including a refractory HPTA, estrogen side effects, hepatoxicity, and adverse effects on cholesterol and the cardiovascular and immune system.

And if you're a drug-tested athlete, there is the very real possibility that you will test positive for the prohormones, especially with the norsteroid and boldenone precursor prohormones. A positive test, because of the difficulty of distinguishing the metabolites of the prohormones from the metabolites of the real anabolic steroids, leads to the same severe penalties, often a two to four year ban, as a positive for anabolic steroids.

Replacement Therapy with Testosterone and Anabolic Steroids

Using testosterone and/or anabolic steroids to increase your levels of androgens in your body is also the wrong way to approach the problem of low systemic testosterone levels. For example, use of exogenous testosterone shuts down the hypothalamic-pituitary-testicular axis (HPTA) that controls testosterone production on the body.

Instead of helping stimulate testosterone production, the use of testosterone and anabolic steroids decreases the natural production of testosterone and basically shuts down your internal machinery for making testosterone. Once you go off the replacement therapy, your testosterone levels often end up lower than before you started taking the exogenous androgens. In some cases testosterone levels never even come close to recovering the pre androgen use levels, and the only alternative, if the system can't be "kick started" to produce testosterone, is to go back on replacement therapy with testosterone or anabolic steroids.

On the other hand, endogenous (developed within the body) hormone production avoids many of the problems associated with exogenous hormone use. By promoting the natural production of the hormone within the body, the regular feedback mechanisms are not by-passed and do not lead to many of the side effects associated with exogenous hormone use.

In fact the use of TestoBoost and other methods to increase endogenous testosterone production ramps up your natural testosterone producing machinery so that even if you stop taking it, your natural levels will be at least as high as before you started, and sometime higher as the body recognizes the higher level as normal and maintains that level naturally.

The bottom line is that whatever your reasons for wanting physiologically increased levels of testosterone, TestoBoost is the best way to go. Besides being more effective in increasing testosterone levels and providing an anabolic drive, the use of TestoBoost won't result in a positive drug test, as is the case with many of the prohormones, (for example the norsteroid ones), exogenous testosterone and anabolic steroids.

New Version of TestoBoost

Research in nutrient metabolism and its effects on the body's hormonal and other systems is accumulating on a daily basis. The advancements made in the scientific and medical fields far outstrips the advancements made in computer chip technologies. However, although Intel puts out a new improved chip on a regular basis, most nutritional supplement products are rarely updated.

In the nutritional supplement field, changes made to formulations are usually to remove a substance that has been either banned or found harmful. Thus the new formulations aren't done for the sake of improving the formula but simply to stay compliant and thus to avoid any legal problems. Examples of this are the new formulations of weight loss products that have removed ephedra and tried to fill the gap by introducing one or two other substances to make it look like they've actually improved the product rather than lessened it. These reworked products are a far cry from products, such as MD+ LipoFlush that are built from the ground up without even giving a thought to the use of ephedra.

My reasons for reformulating include:

1. **The use of new scientific information.**
2. **The use of information from athletes who found certain ingredients useful.**
3. **The use of information from personal clinical studies on the effectiveness of adding certain ingredients to the present formulation.**
4. **Feedback from colleagues and others who have used the product, including their subjective results and blood work.**

TestoBoost Version 4.0 contains a unique formulation that will:

- **Increase testosterone**
- **Provide a potent anabolic effect to increase muscle mass and performance**
- **Decrease body fat**
- **Increase libido and sex drive in both men and women**
- **Block excessive estrogen production**
- **Block excessive production of dihydrotestosterone**
- **Enhances prostate health in men**

TestoBoost is useful for anyone who wants to naturally increase their testosterone levels in order to increase muscle mass and strength and boost sex drive. It's also useful for those who have lower than normal endogenous testosterone levels whether due to age, overtraining, stress, sickness, or even while on or after the use of anabolic steroids and prohormones.

Changes in TestoBoost Version 4.0

A number of ingredients have been added to the TestoBoost formula. TestoBoost was already a very popular and effective supplement and very few of the original ingredients have been altered except to vary the concentrations of some of them.

As well new ingredients have been added.

New ingredients include:

Vitamin D – 400 IU
Coenzyme Q10 – 20 mg
B6 – 25 mg – as a combination now of Pyridoxine HCL and Pyridoxal-5-phosphate

The following ingredients were increased:

Vitamin C to 200 mg from 100 mg
Vitamin E to 200 IU from 100 IU
Vitamin B12 to 200 mcg from 100 mcg
Magnesium 200 mg from 150 mg
Zinc to 15 mg from 10 mg
Tribulus to 500 mg from 450 mg
Acetyl-L-Carnitine to 300 mg from 100 mg
Alpha Lipoic Acid to 150 mg from 50 mg

The Proprietary Complex ingredient amounts were rearranged with the major change being the significant increase in D-aspartate, which now is now the most abundant ingredient in the complex.

Vitamin B6 - TestoBoost version IV now has both pyridoxine (in the form of HCL) and pyridoxal-5-phosphate (P5P) in it. P5P is the metabolically active form of vitamin B6. Pyridoxine HCL, while as easily absorbed as P5P has to be converted to P5P in the body in order to be used by the enzymes involved in protein metabolism and various hormonal processes. P5P is the preferred form of vitamin B6 as it can be used directly in the body without relying on the livers conversion of other forms of vitamin B6 into P5P. As well, less is needed to achieve the same cofactor effects. As such, half of the B6 is in P5P form, and if the body needs more, it can convert the pyridoxine HCL to P5P.

Vitamin B12 (as methylcobalamin) was increased from 100mcg to 200 mcg. Methylcobalamin is the biologically active form of B12, whereas cyanacobalamin, the form used in most supplements, is the synthetic form. The body has to change the cyanacobalamin into methylcobalamin. This process may be compromised in some people so using the metabolically active form is more efficient and improves bioavailability and function. B12 helps to optimize macronutrient metabolism, maximize

muscle mass and decrease body fat. As well, it helps to decrease serum levels of homocysteine, cholesterol and C-Reactive proteins, markers of heart disease and inflammation in the body. Decreasing inflammation helps to decrease cortisol levels and thus increase the anabolic effects of TestoBoost.

Magnesium

A combination of magnesium salts (aspartate, oxide and octadecanoate) were incorporated to take the place of the magnesium oxide. While most of the magnesium is in the form of the aspartate (the form that has been shown influence testosterone levels and is the ingredient that is ZMA, along with zinc monomethione and B6, which are also in TestoBoost). Magnesium in the form of oxide and octadecanoate are also included as both sources of magnesium and to make the tablets more cohesive and less susceptible to breaking in transport.

Arginine Alpha-Ketoglutarate

Arginine alpha-ketoglutarate has both its pluses and its minuses. This compound has several important effects. There is evidence that alpha-ketoglutarate preserves muscle mass and acts as an efficient anticatabolic compound.[1,2] Addition of alpha-ketoglutarate to postoperative total parenteral nutrition prevented the decrease in muscle protein synthesis and free glutamine that usually occurs after surgery.[3] One study has found that an alpha-ketoglutarate-pyridoxine complex may have some beneficial effects on human maximal aerobic and anaerobic performance.[4]

Thus, by ingesting AKG in sufficient quantities, the demand for muscle glutamine might ultimately be spared to some degree, thereby allowing muscle protein synthesis to proceed unhindered (e.g. such as during the muscle hypertrophic response which follows a resistance training workout), and reducing the catabolism of muscle tissue.

Studies have shown that:

1. AKG reduces the decline in muscle free glutamine that is associated with reductions in protein synthesis.[5]

2. The use of AKG, instead of glutamine, prevents the decline in protein anabolism observed following surgery.[6]

AKG seems to exert anti-catabolic effects by preserving muscle glutamine.[7]
These results are not surprising in that the carbon skeleton of BCAAs can be used to synthesize glutamine after the transamination reaction of BCAAs, and ketoglutarate is the immediate carbon donor for glutamine synthesis. The utilization of arginine and ornithine as a carbon source for glutamine synthesis is also a possibility.

A recent study looked at the effects of L-arginine alpha-ketogluterate (AAKG) on measures of body composition and performance.[8] Two separate studies were conducted to assess the pharmacokinetic profile of ingesting two forms of AAKG, timed

released and non timed release, in the blood (study 1) and the effects of dietary supplementation of AAKG on training adaptations in resistance-trained men (study 2).

The study found that the 2 formulations have different pharmacokinetic patterns that may affect arginine release, uptake, and/or physiologic effect over time. As well, the L-arginine alpha-ketogluterate (AAKG) in both formulations positively influenced strength (as measured by one rep maximum bench press) and Wingate peak power performance.

Arginine also has several beneficial health effects. If used in lower doses studies have shown that it does not increase nitric oxide (NO) but still has beneficial effects on protein synthesis, the immune system, increasing growth hormone levels, increasing insulin sensitivity, and serving as substrates for other amino acids, creatine, and polyamines.

However, in higher doses, and especially if combined with nitrate/nitrite, it increases NO formation and facilitates vasodilation, improves sexual functioning, and helps keep you cool during exercise. [9],[10]

But there is an important caveat. The ability of arginine in higher doses to increase nitric oxide is one of the reasons that TestoBoost contains only 100 mg of arginine. That's because excessive production of nitric oxide, whether through the exogenous use of significant amounts of one or more of arginine, and nitrates/nitrites can result in both a decrease in muscle contraction and myotoxicity,[11],[12] and more importantly a lowering of endogenous testosterone production since nitric oxide inhibits Leydig cell steroidogenesis. (see citations and abstracts below).

So while dramatically increasing nitric oxide in the body has some benefits, and provides more of a pump when training giving the impression that it's a potent ergogenic aid, its detrimental effects on testosterone makes the use of nitric oxide supplements containing one or more of large amounts of L-arginine, L-arginine precursors, nitrates and nitrites counter productive for muscle hypertrophy, body composition and athletic performance.

Normally I simply cite the references as endnotes but in the case of nitric oxide and D-aspartate I though it would be useful for you to actually see not only the citations but the abstracts as well. As such I've included several from PubMed about both ingredients so you can see why I limit arginine and increase the amount of d-aspartate in TestoBoost version IV.

Mol Cell Endocrinol. 2002 Aug 30;194(1-2):39-50.

Nitric oxide potently inhibits the rate-limiting enzymatic step in steroidogenesis.

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Abstract

This study tested the hypothesis that nitric oxide (NO) inhibits the rate-limiting catalytic step in steroidogenesis, cytochrome P450 cholesterol side-chain cleaving enzyme (CYP11A1), independent of soluble guanylyl cyclase (GC-S) stimulation. To assess CYP11A1 activity, pregnenolone levels were quantified in murine adrenocortical Y1 cells in the presence of the 3beta-hydroxy-Delta(5)-steroid dehydrogenase inhibitor, 2alpha-cyano-17beta-hydroxy-4,4',17alpha-trimethylandrost-5-ene-3-one. The NO donor, (Z)-1-[2-(2-aminoethyl-N-(2-ammonioethyl)amino]diazen-1-ium-1,2-diolate(deta nonoate), inhibited vasoactive intestinal peptide-, forskolin- and 22alpha-hydroxycholesterol (22HC)-facilitated pregnenolonogenesis in the absence of GC-S activation and in the presence of a GC-S inhibitor, 1H-[1,2,4]oxadiazolo[4,3-a]quinoxalin-1-one (ODQ). CYP11A1 was also heterologously expressed in monkey COS7 cells. Deta nonoate inhibited 22HC-facilitated activity of the over-expressed enzyme in the absence of GC-S activation and in the presence of ODQ. The NO-independent, GC-S agonist, 1-benzyl-3-(5'-hydroxymethyl-2'-furyl)indazole did not inhibit steroidogenesis. The IC(50) for effects of free NO on CYP11A1 was potent and in the 0.4-2 microM range. **These results support the hypothesis that NO inhibits the rate-limiting enzyme in steroidogenesis independent of GC-S activation.**

Biol Reprod. 2010 Sep;83(3):434-42. Epub 2010 May 12.

Testosterone-induced modulation of nitric oxide-cGMP signaling pathway and androgenesis in the rat Leydig cells.

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Abstract

Testosterone, acting as a systemic and local factor, is one of the major regulatory molecules that initiate and maintain testicular function. In the present study, different experimental approaches were used to evaluate the role of testosterone in regulation of the nitric oxide (NO)-cGMP pathway in Leydig cells derived from normal and hypogonadotropic male rats treated with testosterone for 24 h and 2 wk. Real-time quantitative PCR and Western blot analysis revealed increased inducible NO synthase (NOS2) expression followed by increased NO secretion from Leydig cells ex vivo after continuous treatment with testosterone for 2 wk in vivo. The cGMP-specific phosphodiesterases Pde5, Pde6, and Pde9 were up-regulated, whereas PRKG1 protein was decreased after a 2-wk testosterone treatment. Induction of Nos2 and Pde5 in Leydig cells was blocked by androgen receptor antagonist. In experimental hypogonadotropic hypogonadism, expression of NOS2 was significantly reduced, and treatment with testosterone increased NOS2 expression above control levels. PDE5 protein level was unchanged in hypogonadal rats, whereas treatment of hypogonadal rats with testosterone significantly increased it. In contrast, hypogonadism and testosterone replacement reduced PRKG1 protein in Leydig cells. In vitro treatment with testosterone caused gradually increased Nos2 gene expression followed by increased nitrite and cGMP production by purified Leydig cells. **In summary, testosterone up-regulated NO signaling via increased NOS2 expression and contributed to down-regulation of cGMP signaling in Leydig cells. Thus, testosterone-induced modulation of NO-cGMP signaling may serve as a potent autocrine regulator of testicular steroidogenesis.**

Med Hypotheses. 2000 Oct;55(4):310-3.

Is steroid deficiency the cause of tolerance in nitrate therapy?

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Abstract

The award of the Nobel Prize in Physiology and Medicine for 1998 bears witness to the 'explosive' field of nitric oxide (NO), and who would have thought the explosive nitroglycerin owed its therapeutic effectiveness to this little molecule? NO is also involved in causing penile erection, which has brought sildenafil to the aid of patients with erectile dysfunction. However, emerging evidence in animals and in vitro studies indicates that NO also inhibits steroidogenesis, which may have repercussions in humans. The decrease in androgen secretion may impact on secondary sexual characteristics, including penile size. The tolerance to the nitrate therapy in

angina, characterized by volume expansion and not due to sodium retention, may also be related to steroid hormone deficiency. Decreased cortisol secretion may impair water excretion, resulting in volume expansion. Impaired aldosterone secretion would cause hyponatraemia with resultant raised renin. **I hypothesize that continuous therapy with nitrates and sildenafil will result in diminished levels of steroid hormones with predicted sequelae.**

Toxicol Appl Pharmacol. 2000 Dec 15;169(3):222-30.

Decreased steroid hormone synthesis from inorganic nitrite and nitrate: studies in vitro and in vivo.

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Abstract

Nitrites and nitrates are consumed nonchalantly in diet. Organic nitrates are also used as vasodilators in angina pectoris, but the therapy is associated with tolerance whose mechanism remains elusive. Previously, we found inorganic nitrate inhibited steroidogenesis in vitro. Because adrenocorticoids regulate water and electrolyte metabolism, tolerance may ensue from steroid deficiency. We have studied the effects of nitrite and nitrate on in vitro synthesis and in vivo blood levels of steroid hormones. In vitro, nitrite was more potent than nitrate in inhibiting human chorionic gonadotropin (hCG)-stimulated androgen synthesis by Mouse Leydig Tumor cells. At concentrations above 42 mM, nitrite completely inhibited androgen synthesis, and, unlike nitrate, the inhibition was irreversible by increasing hCG concentration. The cAMP production remained intact but reduced with both ions. The nitric oxide (NO) scavenger, 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxy-3-oxide (c-PTIO) significantly increased hCG- or cAMP-stimulated androgen synthesis in all buffers, suggesting that NO is a chemical species directly involved in the nitrite/nitrate-induced inhibition. This is further supported by c-PTIO countering the inhibitory action of methylene blue on androgen synthesis. Rats given distilled water containing 50 mg/L NaNO(2) or NaNO(3) for 4 weeks drank significantly less daily. At the end, their blood corticosterone and testosterone levels were significantly decreased. The adrenocortical histology showed bigger lipid droplets, which are pathogonomic of impaired steroidogenesis. **Nitrite and nitrate are metabolized to NO, which binds heme in cytochrome P450 enzymes, thereby inhibiting steroidogenesis. Therapeutic nitrates likewise may decrease adrenal (and gonadal) steroidogenesis.** Cortisol deficiency would impair water excretion causing volume expansion, and aldosterone deficiency would cause sodium loss and raised renin. Paradoxically, volume expansion without sodium retention and raised renin has all been reported in tolerance.

Theriogenology. 2007 Jan 15;67(2):249-54. Epub 2006 Sep 22.

D-Aspartic acid and nitric oxide as regulators of androgen production in boar testis.

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Abstract

D-Aspartic acid (D-Asp) and nitric oxide (NO) are two biologically active molecules playing important functions as neurotransmitters and neuromodulators of nerve impulse and as regulators of hormone production by endocrine organs. We studied the occurrence of D-Asp and NO as well as their effects on testosterone synthesis in the testis of boar. This model was chosen for our investigations because it contains more Leydig cells than other mammals. Indirect immunofluorescence applied to cryostat sections was used to evaluate the co-localization of D-Asp and of the enzyme nitric oxide synthase (NOS) in the same Leydig cells. D-Asp and NOS often co-existed in the same Leydig cells and were found, separately, in many other testicular cytotypes. D-Asp level was dosed by an enzymatic method performed on boar testis extracts and was 40+/-3.6 nmol/g of fresh tissue. NO measurement was carried out using a biochemical method by NOS activity determination and expressed as quantity of nitrites produced: it was 155.25+/-21.9 nmol/mg of tissue. The effects of the two molecules on steroid hormone production were evaluated by incubating testis homogenates, respectively with or without D-Asp and/or the NO-donor L-arginine (L-Arg). After incubation, the testosterone presence was measured by immunoenzymatic assay (EIA). **These in vitro experiments showed that the addition of D-Asp to incubated testicular homogenates significantly increased testosterone concentration, whereas the addition of L-Arg decreased the hormone production.** Moreover, the inclusion of L-Arg to an incubation medium of testicular homogenates with added D-Asp, completely inhibited the stimulating effects of this enantiomer. Our results suggest an autocrine action of both D-Asp and NO on the steroidogenetic activity of the Leydig cell.

D-Aspartate

D-aspartate (D-Asp) was increased to 1.2 grams per dose for two reasons. First of all aspartate has been shown to increase testosterone production in murine models.[13] A study set up to see if a relationship exists between the presence of D-Asp and the hormonal activity[14] had the following results:

1) Both D-Asp and testosterone are synthesized in rat testes in two periods of the animal's life: before birth, about the 17th day after fertilization and, after birth, at sexual maturity.

2) Immunocytochemical studies have demonstrated that this enantiomer is localized in Leydig and Sertoli cells.

3) In vivo experiments, consisting of i.p. injection of D-Asp to adult male rats, demonstrated that this amino acid accumulates in pituitary and testis (after 5 h, the accumulation was of 12 and 4-fold over basal values, respectively); simultaneously, luteinizing hormone, testosterone and progesterone significantly increased in the blood.
4) Finally, in vitro experiments, consisting of the incubation of D-Asp with isolated testes also demonstrated that this amino acid induces the synthesis of testosterone.

Recent studies have shown that D-Asp has similar effects in man in that it has a role in the regulation and physiological levels of luteinizing hormone and testosterone.[15] For the full PDF version of this study go to http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2774316/pdf/1477-7827-7-120.pdf.

D-aspartate also increase growth hormone levels and has significant metabolic effects, including AMP production, improving the salvage of ATP from in muscle cells, and also acts as an anaplerotic precursor and thus increases TCA flux and ATP formation. This aids in the synthesis of hormones, including testosterone and growth hormone.[16]

Reprod Biol Endocrinol. 2009 Oct 27;7:120.

The role and molecular mechanism of D-aspartic acid in the release and synthesis of LH and testosterone in humans and rats.

Topo E, Soricelli A, D'Aniello A, Ronsini S, D'Aniello G.

Source

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Abstract

BACKGROUND:

D-aspartic acid is an amino acid present in neuroendocrine tissues of invertebrates and vertebrates, including rats and humans. Here we investigated the effect of this amino acid on the release of LH and testosterone in the serum of humans and rats. Furthermore, we investigated the role of D-aspartate in the synthesis of LH and testosterone in the pituitary and testes of rats, and the molecular mechanisms by which this amino acid triggers its action.

METHODS:

For humans: A group of 23 men were given a daily dose of D-aspartate (DADAVIT) for 12 days, whereas another group of 20 men were given a placebo. For rats: A group of 10 rats drank a solution of either 20 mM D-aspartate or a placebo for 12 days. Then LH and testosterone accumulation was determined in the serum and D-aspartate accumulation in tissues. The effects of D-aspartate on the synthesis of LH and testosterone were gauged on isolated rat pituitary and Leydig cells. Tissues were incubated with D-aspartate, and then the concentration (synthesis) of LH and cGMP in the pituitary and of testosterone and cAMP in the Leydig cells was determined.

RESULTS:

In humans and rats, sodium D-aspartate induces an enhancement of LH and testosterone release. In the rat pituitary, sodium D-aspartate increases the release and synthesis of LH through the involvement of cGMP as a second

messenger, whereas in rat testis Leydig cells, it increases the synthesis and release of testosterone and cAMP is implicated as second messenger. In the pituitary and in testes D-Asp is synthesized by a D-aspartate racemase which convert L-Asp into D-Asp. The pituitary and testes possesses a high capacity to trapping circulating D-Asp from hexogen or endogen sources.

CONCLUSION:

D-aspartic acid is a physiological amino acid occurring principally in the pituitary gland and testes and has a role in the regulation of the release and synthesis of LH and testosterone in humans and rats.

Brain Res Rev. 2007 Feb;53(2):215-34. Epub 2006 Nov 21.

D-Aspartic acid: an endogenous amino acid with an important neuroendocrine role.

D'Aniello A.

Source

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Abstract

D-Aspartic acid (d-Asp), an endogenous amino acid present in vertebrates and invertebrates, plays an important role in the neuroendocrine system, as well as in the development of the nervous system. During the embryonic stage of birds and the early postnatal life of mammals, a transient high concentration of d-Asp takes place in the brain and in the retina. d-Asp also acts as a neurotransmitter/neuromodulator. Indeed, this amino acid has been detected in synaptosomes and in synaptic vesicles, where it is released after chemical (K(+) ion, ionomycin) or electric stimuli. Furthermore, d-Asp increases cAMP in neuronal cells and is transported from the synaptic clefts to presynaptic nerve cells through a specific transporter. In the endocrine system, instead, d-Asp is involved in the regulation of hormone synthesis and release. For example, in the rat hypothalamus, it enhances gonadotropin-releasing hormone (GnRH) release and induces oxytocin and vasopressin mRNA synthesis. In the pituitary gland**, it stimulates the secretion of the following hormones: prolactin (PRL), luteinizing hormone (LH), and growth hormone (GH) In the testes, it is present in Leydig cells and is involved in testosterone and progesterone release.** Thus, a hypothalamus-pituitary-gonads pathway, in which d-Asp is involved, has been formulated. In conclusion, the present work is a summary of previous and current research done on the role of d-Asp in the nervous and endocrine systems of invertebrates and vertebrates, including mammals.

Life Sci. 1996;59(2):97-104.

Involvement of D-aspartic acid in the synthesis of testosterone in rat testes.

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Source

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Abstract

D-Aspartic acid (D-Asp) is an endogenous amino acid which occurs in many marine and terrestrial animals. In fetal and young rats, this amino acid occurs prevalently in nervous tissue, whereas at sexual maturity it occurs in endocrine glands and above all in pituitary and testes. Here, we have studied if a relationship exists between the presence of D-Asp and the hormonal activity. The following results were obtained: 1) Both D-Asp and testosterone are synthesized in rat testes in two periods of the animal's life: before birth, about the 17th day after fertilization and, after birth, at sexual maturity. 2) Immunocytochemical studies have demonstrated that this enantiomer is localized in Leydig and Sertoli cells. 3) In vivo experiments, consisting of i.p. injection of D-Asp to adult male rats, demonstrated that this amino acid accumulates in pituitary and testis (after 5 h, the accumulation was of 12 and 4-fold over basal values, respectively); simultaneously, luteinizing hormone, testosterone and progesterone significantly increased in the blood (1.6-fold, $p < 0.05$; 3.0-fold, $p < 0.01$ and 2.9-fold, $p < 0.01$, respectively). 4) Finally, in vitro experiments, consisting of the **incubation of D-Asp with isolated testes also demonstrated that this**

amino acid induces the synthesis of testosterone. These results suggest that free D-Asp is involved in the steroidogenesis.

5-Methyl Methoxy Isoflavone

Methoxy Isoflavone has been shown to increase protein synthesis in animal models, including livestock, with no significant side effects. Anecdotal evidence over the past year has shown that it may exhibit some mild anabolic and anti cortisol effects. Similarly **ecdysone**, also known as **beta ecdysterone** and **20 beta hydroxyecdysterone**, has shown to have some anabolic effects.

Ecdysone (beta ecdysterone, ecditen, 20 beta hydroxyecdysterone)

Ecdysone was popular with Olympic lifters and other athletes in the East and several quasi scientific studies, 2 which were never published, showed that it might have significant anabolic effects.[17] Real world use has not shown dramatic effects and most of the information is available through nutritional companies that vastly overstate the anabolic properties of ecdysone. Nevertheless it makes a useful synergistic ingredient for TestoBoost even though on its own its effects are minimal.

Deer Antler Extract

Velvet deer antler extract has been used in the Orient as an aphrodisiac and a treatment for male impotence, and in Russia as an anabolic agent for athletes. Anecdotal evidence again shows that it may have some effect on increasing serum luteinizing hormone and testosterone levels and increasing sexual drive, with no significant side effects. As such, it works synergistically with other ingredients in TestoBoost that work in similar ways, especially tribulus terrestris.

Tribulus Terrestris Extract

Tribulus terrestris (TT) saponins were used successfully by Eastern European athletes to enhance body composition, strength, and performance. As with the deer antler extract tribulus is supposed to work by increasing LH and testosterone levels. It also has been shown to have some effects on sex drive and sexual function. Several recent studies have found that it does have some beneficial effects and no toxicity.

For example a recent study found that TT extract increased blood testosterone levels, strength and athletic performance.[18] Another recent study found that the use of TT does not result in a positive drug test.[19] Several studies have shown positive effects of TT extracts on rats and other animals, including increased levels of testosterone, increased libido, and positive effects on erectile dysfunction.[20],[21],[22]

Eurycoma Longifolia (Longjack)

Longjack has been used in the Orient to increase libido and male performance. While formal studies have not been done in humans, recent informal ones on murine models have shown that Longjack may increase free serum testosterone levels and sexual drive and performance.[23][24] As such, it works synergistically with other ingredients in TestoBoost that work in similar ways, including **acetyl-L-carnitine**, **catuaba bark, maca root, coleus forskohlii (forskolin), muira puama, chasteberry (vitex agnus-castus - ecdysteroids), suma root (beta ecdysone – also known as beta ecdysterone and 20 beta hydroxyecdysterone), schisandra chinensis, and avena sativa.**

For example, a recent study found that carnitines including **acetyl-L-carnitine** (ALCAR) worked as well as replacement testosterone therapy in improving sexual dysfunction, depressed mood, and fatigue in aging men.[25]

As well, ALCAR seems to have an effect on the hypothalamic-pituitary-testicular axis. Studies have shown that ALC prevented the decrease in plasma testosterone levels after chronic swimming[26], and that ALC stimulates testosterone production[27].

Genistein

Genistein (4',5,7-trihydroxyisoflavone), a major isoflavone in soybeans and a specific inhibitor of protein tyrosine kinase, acts to decrease estrogen in the body. A recent study has shown that there is a synergistic anti-estrogenic effect of **indole-3-carbinol** and genistein.[28] As well as these two ingredients, TestoBoost also contains other anti-estrogenic compounds including **calcium-d-glucarate**,[29] **chrysin** and **ipriflavone**.[30][31] The addition of **bioperine** increases the absorption of these and other ingredients increasing the biological synergistic effects of the combination of ingredients on lowering estrogen effects.

Prickly Pear Extract *

This extract is felt to have neuroprotective and antioxidant effects. Also has insulin like effects and has been shown to have a favorable effect on cholesterol in the body. There is some anecdotal evidence that the use of prickly pear acts as an adaptogen, boosting recovery via an anti-cortisol action. As such it works with other ingredients in TestoBoost to boost recovery and decrease counter productive cortisol levels.

Schisandra Chinensis

Schizandra is a woody vine with clusters of red berries that is found in northern and northeastern China and adjacent regions in Russia and Korea. It is used to treat a variety of medical conditions and is widely known as a longevity herb and aphrodisiac. Athletes have used schisandra in the belief that it will increase endurance and combat fatigue under physical stress.[32] It is also felt to have liver protective effects.

Health Benefits and Protective Effects of TestoBoost

Although TestoBoost is formulated to increase testosterone levels and enhance anabolism, it's also formulated to provide substantial health benefits. For example it has several ingredients, including **saw palmetto (serenoa repens)**, **zinc**, **quercetin**, **GLA** (in **borage oil**) and **stinging nettle** that enhance prostate health in males, and provide anti-inflammatory effects in both males and females.

Some of these ingredients also decrease the formation of dihydrotestosterone from testosterone,[33] thus maximizing testosterone levels while at the same time decreasing the adverse effects of higher systemic and tissue levels of dihydrotestosterone, which includes adverse effects on the prostate and hair loss.[34]

TestoBoost also contains several potent antioxidants, such as **alpha lipoic acid, beta carotene, vitamin C, vitamin E, Coenzyme Q10,** and **turmeric,** which act to improve pituitary and testicular/ovarian function, and decrease the adverse effects of free radicals on the hypothalamic-pituitary-testicular/ovarian axis, and the associated pathways that are responsible for maximizing endogenous testosterone production.

For example, a recent study has found that vitamin E and vitamin C protect the testes from damage secondary to oxidant damage.[35] Alpha lipoic acid (ALA), because it is a sulphur compound, can bind and help eliminate heavy metals such as copper, iron, mercury and cadmium, all of which can cause oxidant damage to the gonads (testes and ovaries).

Alpha lipoic acid (ALA) has a double antioxidant effect as it has significant antioxidant properties on its own, but also regenerates glutathione, the most important endogenous antioxidant. ALA and glutathione have been shown to have significant effects in decreasing mercury toxicity in the body.[36]

The combination of forskohlin and antioxidants in TestoBoost may also impact on Leydig cell function (these are the testicular cells that make testosterone) and result in combating the normal decrease in testosterone seen with aging and stress.[37]

TestoBoost also contains several other vitamins, minerals and nutrients that are important for optimizing testosterone levels. These include **vitamin A, vitamin B6, vitamin B12, niacin, calcium, magnesium, manganese, boron, zinc, ginger** and **Coenzyme Q10**.

The bottom line is that TestoBoost v 4.0 is the most effective testosterone booster on the market today. And although it doesn't contain any prohormones, which can have significant side effects, it surpasses any prohormone formulation in increasing testosterone levels and in providing potent anticatabolic and fat burning effects.

If increasing your anabolic drive, and maximizing muscle mass while minimizing body fat is important to you, check out NitAbol, the nighttime anabolic, fat burning combo that combines TestoBoost with GHboost and Myosin Protein.

NitAbol is also perfect for those who want to lose weight, but would prefer to maintain the muscle they have and strictly lose body fat. In this case I'd also recommend that you use LipoFlush as the ultimate fat loss supplement.

TestoBoost version IV Nutrition Panel

Supplement Facts: Serving Size: 4 Tablets
Servings Per Container: 30

	Amount Per Serving	% Daily Value
Vitamin A (as 71% Beta Carotene and 29% Palmitate)	7000 IU	140%
Vitamin C (as Ascorbic Acid and Potassium Ascorbate)	200 mg	333%
Vitamin D (as Cholecalciferol)	400 IU	100%
Vitamin E (as d-alpha tocopheryl succinate)	200 IU	667%
Niacin	10 mg	50%
Vitamin B6 (as Pyridoxine HCL & Pyridoxal-5-Phosphate)	25 mg	1250%
Vitamin B12 (as Methylcobalamin)	200 mcg	3333%
Calcium (as Calcium Phosphate)	400 mg	40%
Magnesium (as Magnesium Aspartate)	300 mg	75 %
Zinc (as Zinc Monomethionine from Optizinc®)	15 mg	100%
Manganese (as Manganese Chelate)	2 mg	100%
Boron	3 mg	*
Bioperine® *(Piper nigrum)* (fruit)	5 mg	*

	Amount Per Serving	% Daily Value
Coenzyme Q10	20 mg	*
Tribulus Terrestris extract (fruit) Saponins 200 mg	500 mg	*
Acetyl L-Carnitine HCL	300 mg	*
Nettle Extract (leaf)	250 mg	*
Alpha Lipoic Acid	150 mg	*

TestoBoost Proprietary Complex: 5113 mg *

D-Aspartate, Phosphate (as Calcium Phosphate) Catuaba (bark), Muira Puama (bark), Saw Palmetto (berry), Suma (root), Calcium D-Glucarate, Chrysin, Phosphatidylserine, Indole-3-Carbinol, Damiana (leaf), Ipriflavone, Maca (root), Eurycoma Longifolia extract (root), Prickly Pear Extract (leaf), Turmeric (root), 5-Methyl Methoxy Isoflavone, Coleus Forskohlii extract (root), Schisandra (berry), Phosphorus, Quercetin Dihydrate, L-Arginine Alpha Ketoglutarate, Passionflower (herb), Ginger extract (root), GLA (Gamma-linolenic acid from Borage seed Oil Powder), Chasteberry extract *(Vitus agnus castus)* (fruit), Deer Antler Velvet, Oat Straw (aerial parts), Genistein.

Other Ingredients: Cellulose, Stearic Acid, Croscarmellose Sodium, Magnesium Stearate, Hypromellose, Hydroxypropyl Cellulose, Polyethylene.

***Daily Value not established**

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