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 web site advocates 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.

There is, furthermore, the possibility that the dosage used was just not big enough. The most common test for testosterone is one for plasma testosterone. It measures the presence of testosterone in blood.