

Using Testosterone To Reduce Abdominal Fat

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Yet, amid the social and political furor regarding anabolic steroids, important and relevant information is being lost to the headline-grabbing frenzy of politicians and reporters. Anabolic steroids hold the potential to treat health and quality-of-life issues of particular importance to American society and culture. Testosterone is the most widely studied anabolic steroid, being the primary androgen in men. Through media bias, testosterone has been painted as being a dangerous drug and social pariah; this contrasts sharply with the reports from responsible adult users who claim that moderate anabolic steroid use is a positive experience.

The primary benefits of testosterone use are muscle growth and increased strength; secondary benefits range from social to psychological to physical. One of the secondary benefits noted in several studies has received little attention, though it addresses one of the primary foci affecting American health and image on a cultural as well as individual basis.

Testosterone use is associated with fat loss— that is the crux of the article in a single sentence. There is more to the story and the comment is not a blanket statement promoting all levels of testosterone use in all people for fat loss, making it important to understand the basis for the statement.

It has long been observed that men lose muscle mass and gain fat as they age. The loss of muscle mass has been attributed to the age-related decline in testosterone production called andropause or ADAM (androgen deficiency in the aging male).¹ The increase in fat is rarely considered a direct consequence of ADAM, in part because obesity is widespread (no pun intended) and fat gain is multifactorial. Most research in reducing body fat has focused on behavior modification through the use of drugs that decrease appetite, thermogenic drugs that increase calorie burning or drugs that reduce calorie intake by blocking nutrient digestion. With the exception of thermogenic drugs that also have a stimulant effect, athletes and bodybuilders rarely use weight-management drugs. How then do they stay so lean?

The Cornerstone Of Success

Obviously, for all athletes, disciplined diet and regimented exercise are the cornerstones of their body composition success. Among drug-using athletes, there are numerous drugs that promote fat loss, but rarely are anabolic steroids mentioned, as other drugs are more potent in this effect. Rather than focusing on the elite athletes who practice complex polypharmacy (using multiple drugs), consider the example of the recreational athlete or bodybuilder whose use is more consistent with real-world reports. In a recent survey pending publication, researcher Jason Cohen discovered that most recreational anabolic steroid users limit their drug use to a very conservative range of 400mg-600mg testosterone ester equivalent per week for a period (cycle) of eight to 16 weeks.² This moderate dosage is still sufficient to escalate serum (blood) testosterone concentrations above the peak of normal range, also known as supraphysiologic dosing.

A moderate cycle, combined with the appropriate diet and exercise, allows adult men to achieve enviable physiques, with well-defined muscularity. Most people attribute the improvements in appearance to enhanced muscular development,

assuming that total body fat stays essentially the same unless increased cardio or a hypocaloric diet is practiced during the cycle. While results are certainly not uniform among all people, in many cases the user also experiences a minor degree of body fat loss. The pronounced muscle growth, combined with a moderate body fat reduction, can result in an apparent and impressive improvement in body composition. In gyms where moderate anabolic steroid use is prevalent, it is not uncommon to see people, even those over the age of 40, who could easily appear in a fitness magazine.

The potency of testosterone as a fat-reducing agent has never been directly studied, and the exact mechanism(s) behind this effect remains undetermined (it likely involves multiple pathways). It is known that testosterone directly interacts with receptors on and in many different organs that are involved with energy balance (fat mass, appetite, metabolism, etc).³⁻⁶ Testosterone interacts with the output of other hormones involved in regulating the metabolism, regulates genes and enzymes involved in fatty acid oxidation (calorie burning) and may increase motor activity (movement).⁷⁻¹¹ Testosterone also prohibits the formation of new fat cells at the level of the stem cell, promoting muscle cell development in its place.¹⁰

Several studies have reported on the fat-reducing effect of testosterone, including a recent multicenter study published in the *Journal of Clinical Endocrinology & Metabolism*.¹² This study looked at the effect of testosterone therapy (10 grams of 1 percent testosterone gel supplying approximately 10mg of systemic testosterone daily) on whole body and abdominal fat mass in HIV-positive men with abdominal obesity. HIV-positive men were used as the subjects because they often suffer fat gain as a result of certain drug treatments, and the use of testosterone to maintain lean mass in HIV-positive subjects is generally accepted by the medical community and the HIV-positive population; HIV is the virus that can induce AIDS.

The subjects of this study were divided into two groups, one of which received testosterone gel; the other was given a placebo gel that contained no testosterone—both groups used the respective gels for 24 weeks. The subjects were told not to change their diet or exercise, and only men who were not receiving other hormonal support were included in the study. Measurements of body fat, as well as specific regions (trunk, arms, legs, etc.) were taken before and after treatment. At the end of the first 24 weeks, all subjects were offered the opportunity to use the testosterone gel for an additional 24 weeks; most accepted.

Though both groups were similar at the beginning of the study, by the end of the first 24 weeks, significant differences were revealed. Without inducing adverse changes in lipid profile (cholesterol and triglycerides), prostate (as measured by PSA) or insulin sensitivity, testosterone treatment resulted in significantly greater decreases in overall body fat, subcutaneous abdominal fat and total fat mass while increasing lean body mass and reducing abdominal girth.¹² Subjects lost nearly one inch off the waistline, gained almost 3 pounds of lean mass and lost 7.9 percent of total fat mass, while the placebo group increased total fat mass 4.5 percent. What did not change significantly between the two groups was visceral fat mass. Visceral fat is the fat that surrounds the internal organs of the abdomen; visceral fat is associated with problems with insulin sensitivity, cardiovascular disease and the Metabolic Syndrome. In the testosterone group, visceral fat was essentially unchanged over the 24-week period (0.3 percent increase), whereas the control group experienced a 3.1 percent increase. Had testosterone been able to reduce visceral fat mass significantly, that finding could have allowed for wider acceptance of testosterone use in men suffering from the fat-gain side effects of certain HIV drug treatments and possibly others with abdominal obesity demonstrating early signs of diabetes (glucose intolerance), cardiovascular disease or the Metabolic Syndrome. It should be noted that the statistical significance was not reached, despite the trend being for increased visceral fat in the placebo group. This may reflect a failure of the study to detect a difference due to the relatively low number of subjects relative to the degree of effect. When the difference is relatively small, it takes a greater number of subjects to determine a clear and statistical significance due to the variability that

occurs between individual people within the groups.

Though the investigators were quick to point out the shortcomings of the study (failure to significantly affect visceral fat, inability to assess long-term safety, effect on cardiovascular health or prostate growth), this study certainly suggests that testosterone is effective at reducing fat mass and subcutaneous fat, without changing diet or exercise. Notably, the subjects were adult men with low to low/normal testosterone concentrations. Subjects were approved for testosterone treatment with concentrations at or below 400ng/dl—nearly twice the recommendations of the Endocrine Society. The testosterone gel treatment placed the subjects in the high/normal range, and was noted to be well-tolerated.

The results noted in this study agree with several others in which subjects treated with replacement doses of testosterone experienced a decrease in total fat mass.¹³⁻¹⁶ As testosterone is known to increase muscle size and strength in a dose-dependent manner (greater increases with higher doses), one is led to wonder what effect is experienced by bodybuilders and athletes who utilize supraphysiologic (higher than normal) doses of testosterone.¹⁷

In fact, this observation has been made in one peer-reviewed and published study. In a study of healthy young men given various doses of testosterone among the groups, a significant reduction in total, subcutaneous and visceral fat was recorded; visceral fat loss was only experienced by the groups receiving supraphysiologic doses of testosterone. The highest dose used in this particular study was 600mg of testosterone enanthate per week, the same dose practiced by many recreational anabolic steroid users. Again, adverse effects (negative side effects) were minimal and the treatment was well-tolerated.

Practical observations suggest that there is a threshold level where no further fat-loss benefit is gained and potentially a reversal is experienced. It is quite easy to pick out the smooth physiques of men using more than 1,200mg/week of a testosterone ester, much of the smoothness being due to water retention and a poor attention to diet. However, in looking at the direct effect sex steroids (testosterone, DHT and estradiol) have on fat cells, one can see a potential reason for this.

The Deal With Fat

Fat cells were long believed to be simple storage sites for fat, neither contributing nor responding to the metabolic signals of the body other than an increased energy demand due to exercise or starvation. However, it has been learned that fat tissue is an endocrine organ, sending out hormones that affect body composition, appetite and metabolism. Fat also releases a number of proteins that increase inflammation, which can reduce insulin sensitivity and negatively affect cardiovascular health. Fat also responds to hormonal signals from other tissue, much like muscle, which grows when stimulated by testosterone or insulin.

Among the many hormones that stimulate fat cells are the endogenous (natural) steroid molecules, including androgens, estrogens and glucocorticoids (cortisol). In a manner that would be very surprising to many physicians trained prior to the

1990s, fat cells alter steroid concentrations by means of steroid-metabolizing enzymes. Cortisol, the active form of the catabolic steroid that is associated with muscle loss, actually promotes fat gain. Inside the fat cell, cortisol is generated from an inactive form called cortisone. A clear association between high levels of the enzyme that activates cortisol and obesity has been reported.

Fat cells also metabolize sex steroids. Testosterone can be converted to estrogen via the enzyme aromatase, created from androstenedione via an enzyme known as AKR1C3 or deactivated via the enzyme AKR1C2. An elegant study reported in the Journal Clinical Endocrinology revealed a strong association between body mass index (BMI) and aromatase (as measured by mRNA) in subcutaneous fat cells; a weaker association between aromatase and body fat percentage was also present.¹⁸ The authors noted that the association with body fat percentage would likely have been stronger if a more precise measure was used, as opposed to the impedance method.

In clearer terms, the study showed that people were heavier and fatter if their fat cells increased estrogen content by increasing aromatization. This would decrease both testosterone (since aromatase uses testosterone to make estradiol) and the testosterone:estradiol ratio. Several papers have reported increased circulating estrogen levels in obese people, often speculating that the increased estrogen concentration is a result of an increased fat mass.¹⁹⁻²¹ From the results of this study, it appears that the estrogen-obesity cycle is self-propagating, as one leads to the other, as suggested by other researchers.²²

Estrogens act on the fat cell by activating two different classes of the estrogen receptor, α and β . The β -class of the estrogen receptor (ER β) when occupied by estradiol stimulates another class of receptor called β 2A-adrenergic receptor.²³ This is the receptor blocked by yohimbine, which is used in several fat-loss supplements. When β 2A is activated, it inhibits lipolysis, making it more difficult for the fat cell to release stored fat. Conversely, testosterone increases the density of β -adrenergic receptors, which increase lipolysis and the release of stored fat.¹⁰

To date, many studies have observed a fat-loss effect when replacement doses of testosterone are given to men— young and old, healthy or ill.¹³⁻¹⁶ One study has even documented a further benefit in giving supraphysiologic testosterone to young men, in that it reduced visceral fat, which is more strongly associated with cardiovascular disease and insulin resistance.¹⁷ Clearly, there is an interaction between sex steroids and obesity that is underappreciated.

A study recently published in the Journal of Clinical Endocrinology & Metabolism demonstrated a clear association, showing increased visceral fat in men with lower bioavailable and free testosterone and increased subcutaneous fat in men with higher estradiol levels.²⁴ The relation between obesity and sex steroids goes even further, as one looks inside the fat cell to discover that fat cells alter sex steroid concentrations by generating a high intracellular concentration of estradiol by aromatizing testosterone. In the fat cell, estradiol binds to ER β , activating a second class of receptor, β 2A, which inhibits fat loss.¹⁸

Another related finding of interest is something called CAG repeat polymorphism. This refers to a coding in a person's DNA that determines the structure of the androgen receptor. When the CAG repeat polymorphism is

longer, more androgen receptors are produced. In men, those with a greater number of repeats have greater lean mass and tend to have less body fat.²⁵

It is likely that optimizing fat loss requires a close attention to both testosterone levels and the testosterone:estrogen ratio. The gain in fat mass seen with aging is related to a drop in both testosterone and estrogen, but the drop in testosterone is much greater, allowing estrogen levels to rise in relation to testosterone.¹⁹ However, it is important to note that estrogen can't simply be eradicated from the system. Men who suffer from a genetic inability to produce estrogen due to aromatase deficiency are insulin resistant, suffer from metabolic challenges and are abdominally obese.²⁶ Of course, the opposite extreme also affects body fat, as men who are genetically prone to generate high levels of estrogen cause a female pattern of fat deposition.²⁷

These findings suggest several avenues of research in treating obesity in men. The simplest may be to provide a moderate, supraphysiologic course of testosterone. This mimics the recreational use of anabolic steroids, which has proven successful for improving body composition and appearance for a vast majority of adult male users. Another avenue may be to investigate the use of a mild aromatase inhibitor. Healthy men given a low dose of an aromatase inhibitor see a dramatic rise in circulating testosterone, as the feedback that regulates testosterone production is dependent, in part, on estradiol.²⁸ By inhibiting estrogen formation, testosterone production is maintained at a higher output. It is important not to fully suppress aromatization, as bone strength is dependent upon estrogen and men who are genetically deficient in aromatase suffer from several metabolic challenges and develop abdominal obesity.

Certainly, there is much to be learned in regard to the clinical use of testosterone or aromatase inhibitors for treating or preventing obesity. However, the findings of these studies suggest that bodybuilders and other anabolic steroid users may have been experiencing fat-loss benefits that have long been ignored by medical science.

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