ANABOLICS 2005

by William Llewellyn

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FOREWORD

By Rick Collins, J.D.

In my extensive legal work involving anabolic steroid cases, I've corresponded with countless self-professed steroid authorities from the medical, scientific and academic fields. These are individuals with impressive credentials and doctoral degrees. You'd think they'd be fountains of knowledge. But they are mostly armchair experts or professional alarmists far more interested in pushing their own agenda than in engaging in any objective discussion. Bill Llewellyn is different.

Bill and I were both raised on Long Island, where a robust gym culture has flourished since the 1980s. Bill was part of that culture, and his experiences in the trenches and as a purely self-taught scientist have shaped his perspectives and influenced his writings as an author and columnist. By his own straightforward admission, Bill has taken a great deal of steroids. "I couldn't imagine writing so much about something I had little firsthand experience with," he said in a 2003 *Muscular Development* interview. What he has learned through the years he now passes down, in the belief that lack of reliable information presents far greater dangers than open discussion. He has little patience for trendy terms like "guru," instead describing himself as "a researcher with a reverence for the truth."

Bill's approach neither demonizes nor glamorizes anabolic steroids. From his monthly columns in *Muscular Development* to his original ANABOLICS 2000, Bill has striven to tell the truth as he sees it about a topic he knows a great deal about. Of course, his choice for frankness rather than dogmatic condemnation has made him a controversial figure. The cover of *ESPN The Magazine* once pictured him as a mysterious robed renegade, with a story that portrayed him as the representative of all those who would undermine the movement to eradicate performance-enhancing substances from sports. In actuality, Bill has little interest in the ethics issue concerning steroids in competitive athletics. His motivation has always been to study the "cosmetic" application of steroids for bodybuilding – the same application that attracts the great majority of steroid users.

When I was researching my book on steroids and the law, Bill was one of my invaluable resources on complex issues of steroid chemistry, real-world pharmacology, and product history/contents/counterfeiting. In my day-to-day practice, I have often found the previous editions of his book to be an indispensable reference. I can't imagine anyone involved with steroids in almost any capacity not having the latest edition of Bill's book.

Rick Collins, J.D.
Author, LEGAL MUSCLE: ANABOLICS IN AMERICA

Anabolic Overview

An Introduction to Testosterone

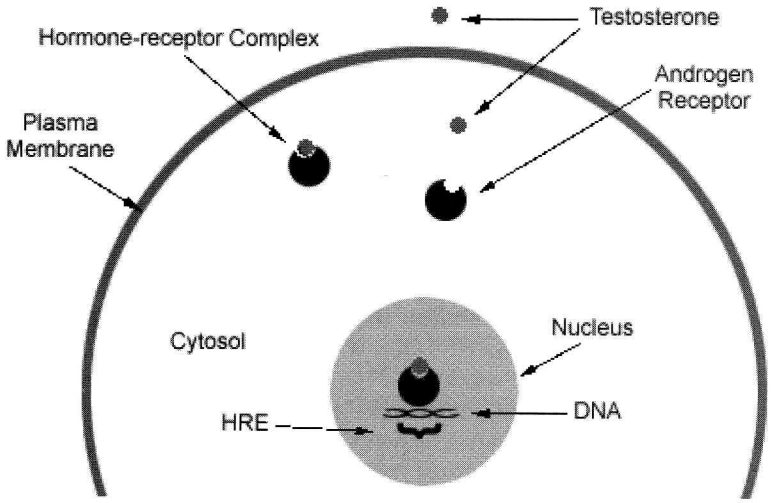
Anabolic steroids are a class of medications that contain a synthetically manufactured form of the hormone testosterone, or a related compound that is derived from (or similar in structure and action to) this hormone. In order to fully grasp how anabolic steroids work, it is, therefore, important to understand the basic functioning of testosterone.

Testosterone is the primary male sex hormone. It is manufactured by the Leydig's cells in the testes at varying amounts throughout a person's life span. The effects of this hormone become most evident during the time of puberty, when an increased output of testosterone will elicit dramatic physiological changes in the male body. This includes the onset of secondary male characteristics such as a deepened voice, body and facial hair growth, increased oil output by the sebaceous glands, development of sexual organs, maturation of sperm, and an increased libido. Indeed the male reproductive system will not function properly if testosterone levels are not significant. All such effects are considered the masculinizing or "androgenic" properties of this hormone.

Increased testosterone production will also cause growth promoting or "anabolic" changes in the body, including an enhanced rate of protein synthesis (leading to muscle accumulation). Testosterone is the reason males carry more muscle mass than women, as the two sexes have vastly contrasting amounts of this hormone. More specifically, the adult male body will manufacture between 2.5 and 11mg per day[1] while females only produce about ¼ mg. The dominant sex hormone for women is estrogen, which has a significantly different effect on the body. Among other things, a lower androgen and high estrogen level will cause women to store more body fat, accumulate less muscle tissue, have a shorter stature, and become more apt to bone weakening with age (osteoporosis).

The actual mechanism in which testosterone elicits these changes is somewhat complex. When free in the blood stream, the testosterone molecule is available to interact with various cells in the body. This includes skeletal muscle cells, as well as skin, scalp, kidney, bone, central nervous system, and prostate tissues. Testosterone binds with a cellular target in order to exert its activity, and will, therefore, effect only those body cells that posses the proper hormone receptor site (specifically the androgen receptor). This process can be likened to a lock and key system, with each receptor (lock) only being activated by a particular type of hormone (key). During this interaction, the testosterone molecule will become bound to the intracellular receptor site (located in the cytosol, not on the membrane surface), forming a new "receptor complex". This complex (hormone + receptor site) will then migrate to the cell's nucleus where it will attach to a specific section of the cell's DNA, referred to as the hormone response element. This will activate the transcription of specific genes, which in the case of a skeletal muscle cell will ultimately cause (among other things) an increase in the synthesis of the two primary contractile proteins, actin and myosin (muscular growth). Carbohydrate storage in muscle tissue may be increased due to androgen action as well.

Once this messaging process is completed, the complex will be released, and the receptor and hormone will disassociate. Both are then free to migrate back into the cytosol for further activity. The testosterone molecule is also free to diffuse back into circulation to interact with other cells. The entire receptor cycle, including hormone binding, receptor-hormone complex migration, gene transcription and subsequent return to cytosol is a slow process, taking hours, not minutes, to complete. For example, in studies using a single injection of nandrolone, it is measured to be 4 to 6 hours before free androgen receptors migrate back to the cytosol after activation. It is also suggested that this cycle includes the splitting and formation of new androgen receptors once returned to cytosol, a possible explanation for the many observations that androgens are integral in the formation of their own receptor sites[2].



Cellular Diagram: Testosterone freely diffuses through the plasma membrane and binds with an intracellular androgen receptor. The hormone-receptor complex then enters the cell nucleus to bind with a specific segment of DNA (the Hormone Response Element), activating the transcription of specific genes.

In the kidneys, this same process works to allow androgens to augment erythropoiesis (red blood cell production)[3]. It is this effect that leads to an increase in red blood cell concentrations, and possibly increased oxygen transport capacity, during anabolic/androgenic steroid therapy. Many athletes mistakenly assume that oxymetholone and boldenone are unique in this ability, due to specific uses or mentions of this effect in drug literature. In fact, stimulation of erythropoiesis occurs with nearly all anabolic/androgenic steroids, as this effect is simply tied with activation of the androgen receptor in kidney cells. The only real exceptions might be compounds such as dihydrotestosterone and some of its derivatives[4], which are rapidly broken down upon interaction with the 3alpha-hydroxysteroid dehydrogenase enzymes (kidney tissue has a similar enzyme distribution to muscle tissue, see "anabolic/androgenic dissociation" section), and therefore display low activity in these tissues.

Adipose (fat) tissues are also androgen responsive, and here these hormones support the lipolytic (fat mobilizing) capacity of cells[5]. This may be accomplished by an androgen-tied regulation of beta-adrenergenic receptor concentrations or general cellular activity (through adenylate cyclase)[6]. We also note that the level of androgens in the body will closely correlate (inversely) with the level of stored body fat. As the level of androgenic hormones drops, typically the deposition of body fat will increase[7]. Likewise as we enhance the androgen level, body fat may be depleted at a more active rate. The ratio of androgen to estrogen action is in fact most important, as estrogen plays a counter role by acting to increase the storage of body fat in many sites of action[8]. Likewise, if one wished to lose fat during steroid use, estrogen levels should be kept low. This is clearly evidenced by the fact that non-aromatizing steroids have always been favored by bodybuilders looking to increase the look of definition and muscularity while aromatizing compounds are typically relegated to bulking phases of training due to their tendency to increase body fat storage. Aromatization is discussed in more detail in a following section (see: Estrogen Aromatization).

As mentioned, testosterone also elicits androgenic activity, which occurs by its activating receptors in what are considered to be androgen responsive tissues (often through prior conversion to dihydrotestosterone See: DHT Conversion). This includes the sebaceous glands, which are responsible for the secretion of oils in the skin. As the androgen level rises, so does the release of oils. As oil output increases, so does the chance for pores becoming clogged (we can see why acne is such a common side effect of steroid use). The production of body, and facial hair is also linked to androgen receptor activation in skin and scalp tissues. This becomes most noticeable as boys mature into puberty, a period when testosterone levels rise rapidly, and androgen activity begins to stimulate the growth of hair on the body and face. Some time later in life, and with the contribution of a genetic predisposition, androgen activity in the scalp may also help to initiate male-pattern hair loss. It is a misconception that dihydrotestosterone is an isolated culprit in the promotion of hair loss, however; as in actuality it is the general activation of the androgen receptor that is to blame (see: DHT Conversion). The functioning of sex glands and libido are also tied to the activity of androgens, as are numerous other regions of the central nervous/neuromuscular system.

Direct and Indirect Anabolic Effects

Although testosterone had been isolated, synthesized, and actively experimented with for many decades now, there is still some debate today as to exactly how steroids effect muscle mass. At this point in time, the primary mode of anabolic action with all anabolic/androgenic steroids is understood to be direct activation of the cellular androgen receptor and increases in protein synthesis. As follows, if we are able to increase our androgen level from an external source by supplementing testosterone or a similar anabolic steroid, we can greatly enhance the rate in which protein is retained by the muscles. This is clearly the primary cause for muscle growth with all anabolic/androgenic steroids. As our hormone levels increase, so does androgen receptor activation, and ultimately the rate of protein synthesis.

But other indirect mechanisms could possibly affect muscle growth outside of the normally understood androgen action on protein synthesis. An indirect mechanism is one that is not brought about by activation of the androgen receptor, but the affect androgens might have on other hormones, or even the release of locally acting hormones or growth promoters inside cells (perhaps mediated by other membrane bound receptors). We must remember also that muscle mass disposition involves not only protein synthesis, but also other factors such as tissue nutrient transport and protein breakdown. We need to look at androgenic interaction with these factors as well to get a compete picture. Concerning the first possibility, we note that studies with testosterone suggest that this hormone does not increase tissue amino acid transport[9]. This fact probably explains the profound synergy bodybuilders have noted in recent years with insulin, a hormone that strongly increases transport of nutrients into muscle cells. But regarding protein breakdown, we do see a second important pathway in which androgens might affect muscle growth.

Anti-Glucocorticoid Effect of Testosterone

Testosterone (and synthetic anabolic/androgenic steroids) may help to increase mass and strength by having an anti-catabolic effect on muscle cells. Considered one of the most important indirect mechanisms of androgen action, these hormones are shown to effect the actions of another type of steroid hormone in the body, glucocorticoids (cortisol is the primary representative of this group)[10]. Glucocorticoid hormones actually have the exact opposite effect on the muscle cell than androgens, namely sending an order to release stored protein. This process is referred to as catabolism, and represents a breaking down of muscle tissue. Muscle growth is achieved when the anabolic effects of testosterone are more pronounced overall than the degenerative effects of cortisol. With intense training and a proper diet, the body will typically store more protein than it removes, but this underlying battle is always constant.

When administering anabolic steroids however, a much higher androgen level can place glucocorticoids at a notable disadvantage. With their effect reduced, fewer cells will be given a message to release protein, and more will be accumulated in the long run. The primarily mechanism believed to bring this effect out is androgen displacement of glucocorticoids bound to the glucocorticoid receptor. In fact, in-vitro studies have supported this notion by demonstrating that testosterone has a very high affinity for this receptor[11], and further suggesting that some of its anabolic activity is directly mediated through this action[12]. It is also suggested that androgens may indirectly interfere with DNA binding to the glucocorticoid response element[13]. Although the exact underlying mechanism is still in debate, what is clear is that steroid administration inhibits protein breakdown, even in the fasted state, which seems clearly indicative of an anti-catabolic effect.

Testosterone and Creatine

In addition to protein synthesis, a rise in androgen levels should also enhance the synthesis of creatine in skeletal muscle tissues[14]. Creatine, as creatine phosphate (CP), plays a crucial role in the manufacture of ATP (adenosine triphosphate), which is a main store of energy for the muscles. As the muscle cells are stimulated to contract, ATP molecules are broken down into ADP (adenosine diphosphate), which releases energy. The cells will then undergo a process using creatine phosphate to rapidly restore ADP to its original structure, in order to replenish ATP concentrations. During periods of intense activity however, this process will not be fast enough to compensate and ATP levels will become lowered. This will cause the muscles to become fatigued and less able to effort a strenuous contraction. With increased levels of CP available to the cells, ATP is replenished at an enhanced rate and the muscle is both stronger and more enduring. This effect will account for some portion of the early strength increases seen during steroid therapy. Although perhaps not technically considered an anabolic effect as tissue

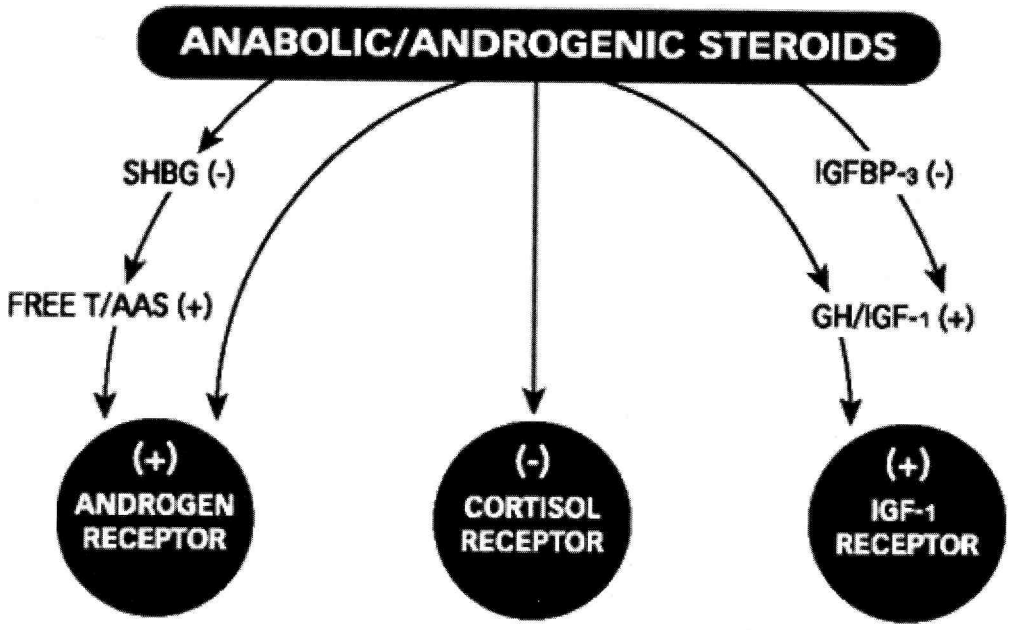
hypertrophy is not a direct result, androgen support of creatine synthesis is certainly still looked at as a positive and growth supporting result in the mind of the bodybuilder.

Testosterone and IGF-1

It has also been suggested that there is an indirect mechanism of testosterone action on muscle mass mediated by Insulin-Like Growth Factor. To be more specific, studies note a clear link between androgens and tissue release of[15], and responsiveness to, this anabolic hormone. For example, it has been demonstrated that increases in IGF-1 receptor concentrations in skeletal muscle are noted when elderly men are given replacement doses of testosterone[16]. In essence, the cells are becoming primed for the actions of IGF-1, by testosterone. Alternately we see marked decreases in IGF-1 receptor protein levels with androgen deficiency in young men. It also appears that androgens are necessary for the local production and function of IGF-1 in skeletal muscle cells, independent of circulating growth hormone, and IGF-1 levels[17]. Since we do know for certain that IGF-1 is at least a minor anabolic hormone in muscle tissue, it seems reasonable to conclude that this factor, at least at some level, is involved in the muscle growth noted with steroid therapy.

Direct and Indirect Steroids?

In looking over the proposed indirect effects of testosterone, and pondering the effectiveness of the synthetic anabolic/androgenic steroids, we must resist the temptation to believe we can categorize steroids as those which directly, and those which indirectly, promote muscle growth. The belief that there are two dichotomous groups or classes of steroids ignores the fact that all commercial steroids promote not only muscle growth but also androgenic effects. There is no complete separation of these traits at this time, making clear that all activate the cellular androgen receptor. I believe the theory behind direct and indirect steroid classifications originated when some noted the low receptor binding affinity of seemingly strong anabolic steroids like oxymetholone and methandrostenolone[18]. If they bind poorly, yet work well, something else must be at work. This type of thinking fails to recognize other factors in the potency of these compounds, such as their long half-lives, estrogenic activity and weak interaction with restrictive binding proteins (see: Free vs. Bound Testosterone). While there may possibly be differences in the way various compounds could foster growth indirectly, such that advantages might even be found with certain synergistic drug combinations, the primary mode of action with all of these compounds is the androgen receptor. The notion that steroid X and Y must never be stacked together because they both compete for the same receptor when stimulating growth, while X and Z should be combined because they work via different mechanisms, should likewise not be taken too seriously. Such classifications are based on speculation only, and upon reasonable investigation are clearly invalid.



MECHANISM OF ACTION DIAGRAM: The mechanism of anabolic action due to the administation of anabolic/androgenic steroids. AAS causes not only direct stimulation of the androgen receptor, but also supports muscle growth by increasing the levels of free androgens, increasing androgen receptor density, inhibiting corticosteroid action, increasing GH/IGF-1, and suppressing IGF-1 binding proteins.

Free vs. Bound Testosterone

A very small amount of testosterone actually exists in a free state, where interaction with cellular receptors is possible. The majority will be bound to the proteins SHBG (sex hormone binding globulin, also referred to as sex steroid binding globulin and testosterone-estradiol binding globulin) and albumin, which temporarily prevent the hormone from exerting activity. Steroid hormones actually bind much more avidly to SHBG than albumin (with approximately 1,000 times greater affinity), however albumin is present in a level 1,000 times greater than SHBG. Therefore, the activity of both binding proteins in the body is relatively equal. The distribution of testosterone in men is typically 45% of testosterone bound to SHBG, and about 53% bound to albumin. The remaining 2% of the average blood concentration exists in a free, unbound state. In women, the percentage of free testosterone is lower, measured to be approximately 1%. A binding protein called ABP (androgen binding protein) also helps to mediate androgen activity in the reproductive system, although since it is found exclusively in these tissues, it is not relevant to muscle growth.

The level of free testosterone available in the blood is likewise an important factor mediating its activity, as only a small percentage is really active at any given time. It must also be noted that as we alter testosterone to form new anabolic/androgenic steroids, we also typically alter the affinity in which the steroid will bind to plasma proteins. This is an important consideration, as the higher percentage we have of free hormone, the more active the compound should be on a milligram for milligram basis. And the variance can be substantial between different compounds. For example, Proviron® (1-methyl dihydrotestosterone) binds with SHBG many times more avidly than testosterone[19], while mibolerone (7,17 dimethyl-nandrolone) and bolasterone (7,17 dimethyl-testosterone) show virtually no affinity for this protein at all (clearly the reason these steroids are such potent androgens).

The level of SHBG present in the body is also variable, and can be altered by a number of factors. The most prominent seems to be the concentration of estrogen and thyroid hormones present in the blood. We generally see a reduction in the amount of this plasma binding protein as estrogen and thyroid content decreases, and a rise in SHBG as they increase. A heightened androgen level due to the administration of anabolic/androgenic steroids has also been shown to lower levels of this protein considerably. This is clearly supported by a 1989 German study, which noted a strong tendency for SHBG reduction with the oral anabolic steroid stanozolol (Winstrol®)[20]. After only 3 days of administering a daily dose of .2mg/kg body-weight (about 18mg for a 200lb man), SHBG was lowered nearly 50% in normal subjects. Similar results have been obtained with the use of injectable testosterone enanthate; however, milligram for milligram, the effect of stanozolol was much greater in comparison. The form of administration may have been important in reaching this level of response. Although the injectable was not tried in the German study, we can refer to others comparing the effect of oral vs. transdermal estrogen[21]. These show a much greater response in SHBG levels when the drug is given orally. This is perhaps explained by the fact that SHBG is produced in the liver. Therefore, we cannot assume that injectable Winstrol® (or injectable steroids in general) will display the same level of potency in this regard.

Lowering the level of plasma binding proteins is also not the only mechanism that allows for an increased level of free testosterone. Steroids that display a high affinity for these proteins may also increase the level of free testosterone by competing with it for binding. Obviously if testosterone finds it more difficult to locate available plasma proteins in the presence of the additional compound, more will be left in an unbound state. A number of steroids including dihydrotestosterone, Proviron®, and Oral-Turinabol (chlorodehydromethyltestosterone) display a strong tendency for this effect. If the level of free-testosterone can be altered by the use of different anabolic/androgenic steroids, the possibility also exists that one steroid can increase the potency of another through these same mechanisms. For example, Proviron® is a poor anabolic, but its extremely high affinity for SHBG might make it useful by allowing the displacement of other steroids that are more active in these tissues.

We must not let this discussion lead us into thinking that binding proteins serve no valuable function. In fact they play a vital role in the transport and functioning of endogenous androgens. Binding proteins act to protect the steroid against rapid metabolism, ensure a more stable blood hormone concentration, and facilitate an even distribution of hormone to various body organs. The recent discovery of a specific receptor for Sex Hormone-Binding Globulin (SHBG-R) located on the membrane surface of steroid responsive body cells also suggests a much more complicated role for this protein than soley hormone transport. However, it remains clear that manipulating the tendency of a hormone to exist in an unbound state is an effective way to alter drug potency.

Estrogen Aromatization

Testosterone is the primary substrate used in the male body for the synthesis of estrogen (estradiol), the principal female sex hormone. Although the presence of estrogen may seem quite unusual in men, it is structurally very similar to testosterone. With a slight alteration by the enzyme aromatase, estrogen is produced in the male body. Aromatase activity occurs in various regions of the male body, including adipose[22], liver[23], gonadal[24], central nervous system[25], and skeletal muscle[26] tissues. In the context of the average healthy male, the amount of estrogen produced is generally not very significant to one's body disposition, and may even be beneficial in terms of cholesterol values (See Side Effects: Cardiovascular Disease). However, in larger amounts it does have potential to cause many unwanted effects including water retention, female breast tissue development (gynecomastia), and body fat accumulation. For these reasons, many focus on minimizing the build-up or activity of estrogen in the body with aromatase inhibitors such as Arimidex and Cytadren, or anti-estrogens such as Clomid or Nolvadex, particularly at times when gynecomastia is a worry or the athlete is attempting to increase muscle definition.

We must, however, not be led into thinking that estrogen serves no benefit. It is actually a desirable hormone in many regards. Athletes have known for years that estrogenic steroids are the best mass builders, but it is only recently that are we finally coming to understand the underlying mechanisms why. It appears that reasons go beyond the simple size, weight, and strength increases that one would attribute to estrogen-related water retention, with this hormone actually having a direct effect on the process of anabolism. This is manifest through increases in glucose utilization, growth hormone secretion, and androgen receptor proliferation.

Glucose Utilization and Estrogen

Estrogen may play a very important role in the promotion of an anabolic state by affecting glucose utilization in muscle tissue. This occurs via an altering of the level of available glucose 6-phosphate dehydrogenase, an enzyme directly tied to the use of glucose for muscle tissue growth and recuperation[27 28]. More specifically, G6PD is a vital part of the pentose phosphate pathway, which is integral in determining the rate nucleic acids and lipids are to be synthesized in cells for tissue repair. During the period of regeneration after skeletal muscle damage levels of G6PD are shown to rise dramatically, which is believed to represent a mechanism for the body to enhance recovery when needed. Surprisingly, we find that estrogen is directly tied to the level of G6PD that is to be made available to cells in this recovery window.

The link between estrogen and G6PD was established in a study demonstrating levels of this dehydrogenase enzyme to rise after administration of testosterone propionate. The investigation further showed that the aromatization of testosterone to estradiol was directly responsible for this increase, and not the androgenic action of this steroid[29]. The non-aromatizable steroids dihydrotestosterone and fluoxymesterone were tested alongside testosterone propionate, but failed to duplicate the effect of testosterone. Furthermore, the positive effect of testosterone propionate was blocked when the aromatase inhibitor 4-hydroxyandrostenedione (formestane) was added, while 17-beta estradiol administration alone caused a similar increase in G6PD to testosterone propionate. The inactive estrogen isomer 17-alpha estradiol, which is unable to bind the estrogen receptor, failed to do anything. Further tests using testosterone propionate and the anti-androgen flutamide showed that this drug also did nothing to block the positive action of testosterone, establishing it as an effect independent of the androgen receptor.

Estrogen and GH/IGF-1

Estrogen may also play an important role in the production of growth hormone and IGF-1. IGF-1 (insulin-like growth factor) is an anabolic hormone released in the liver and various peripheral tissues via the stimulus of growth hormone (See Drug Profiles: Growth Hormone). IGF-1 is responsible for the anabolic activity of growth hormone such as increased nitrogen retention/protein synthesis and cell hyperplasia (proliferation). One of the first studies to bring this issue to our attention looked at the effects of the anti-estrogen tamoxifen on IGF-1 levels, demonstrating it to have a suppressive effect[30]. A second, perhaps more noteworthy, study took place in 1993, which looked at the effects of testosterone replacement therapy on GH and IGF-1 levels alone, and compared them to the effects of testosterone combined again with tamoxifen[31]. When tamoxifen was given, GH and IGF-1 levels were notably suppressed, while both values were elevated with the administration of testosterone enanthate alone. Another study has shown 300mg of testosterone enanthate weekly to cause a slight IGF-1 increase in

normal men. Here the 300mg of testosterone ester caused an elevation of estradiol levels, which would be expected at such a dose. This was compared to the effect of the same dosage of nandrolone decanoate; however, this steroid failed to produce the same increase. This result is quite interesting, especially when we note that estrogen levels were actually lowered[32] when this steroid was given. Yet another demonstrated that GH and IGF-1 secretion is increased with testosterone administration on males with delayed puberty, while dihydrotestosterone (non-aromatizable) seems to suppress GH and IGF-1 secretion[33].

Estrogen and the Androgen Receptor

It has also been demonstrated that estrogen can increase the concentration of androgen receptors in certain tissues. This was shown in studies with rats, which looked at the effects of estrogen on cellular androgen receptors in animals that underwent orchiectomy (removal of testes, often done to diminish endogenous androgen production). According to the study, administration of estrogen resulted in a striking 480% increase in methyltrienolone (a potent oral androgen often used to reference receptor binding in studies) binding in the levator ani muscle[34]. The suggested explanation is that estrogen must either be directly stimulating androgen receptor production, or perhaps diminishing the rate of receptor breakdown. Although the growth of the levator ani muscle is commonly used as a reference for the anabolic activity of steroid compounds, it is admittedly a sex organ muscle, and different from skeletal muscle tissue in that it possesses a much higher concentration of androgen receptors. However, this study did look at the effect of estrogen in fast-twitch skeletal muscle tissues (tibialis anterior and extensor digitorum longus) as well, but did not note the same dramatic increase as the levator ani. Although discouraging at first glance, the fact the estrogen can increase androgen receptor binding in any tissue remains an extremely significant finding, especially in light of the fact that we now know androgens to have some positive effects outside of muscle tissue.

Anti-Estrogens and the Athlete

So what does this all mean to the bodybuilder looking to gain optimal size? Basically I think it calls for a cautious approach to the use of estrogen maintenance drugs if mass is the objective. Obviously, anti-estrogens should be used if there is a clear need for them due to the onset of estrogenic side effects, or at the very least, the drugs being administered should be substituted for non-estrogenic compounds. Gynecomastia is certainly an unwanted problem for the steroid user, as are noticeable fat mass gains. But if these problems have not presented themselves, the added estrogen due to a cycle of testosterone or Dianabol, for example, might indeed be aiding in the buildup of muscle mass. An individual confident they will notice, or are not prone to getting, these side effects, may therefore want to hold off using estrogen maintenance drugs so as to achieve the maximum gains in tissue mass as possible.

DHT Conversion

As we see from our discussion with estrogen, in considering the physiological effects of any steroid, we must look at all of its active metabolites, and not just the initial compound. This includes not only estrogenic products, but androgenic metabolites as well. With this in mind, it is important to note that the potency of testosterone is considerably increased in many androgen responsive tissues when it converts to dihydrotestosterone. More commonly referred to by the three-letter abbreviation DHT, this hormone is, in fact, measured to be approximately three to four times stronger than testosterone. It is the most potent steroid found naturally in the human body, and important to discuss if we are to understand the full activity of testosterone, as well as other anabolic/androgenic steroids that undergo a similar conversion.

Testosterone is converted to dihydrotestosterone upon interaction with the 5-alpha reductase enzyme. More specifically, this enzyme removes the C4-5 double-bond of testosterone by the addition of two hydrogen atoms to its structure (hence the name di-hydro testosterone). The removal of this bond is important, as in this case it creates a steroid that binds to the androgen receptor much more avidly than its parent steroid. 5-alpha reductase is present in high amounts in tissues of the prostate, skin, scalp, liver and various regions of the central nervous system, and as such represents a mechanism for the body to increase the potency of testosterone specifically where strong androgenic action is needed. In these areas of the body little testosterone will actually make its way to the receptor without being converted to dihydrotestosterone, making DHT by far the active form of androgen here.

DHT and Androgenic Side Effects

In some regards this local potentiation of testosterone's activity may be unwelcome, as higher androgenic activity in certain tissues may produce a number of undesireable side effects. Acne, for example, is often triggered by dihydrotestosterone activity in the sebaceous glands, and the local formation of dihydrotestosterone in the scalp is typically blamed for triggering male pattern hair loss. You should know that it is a terrible misconception among bodybuilders that dihydrotestosterone is an isolated culprit when it comes to these side effects. All anabolic/androgenic steroids exert their activities, both anabolic and androgenic, through the same cellular androgen receptor. Dihydrotestosterone is no different than any other steroid except that it is a more potent activator of this receptor than most, and can be formed locally in certain androgen-sensitive tissues. All steroids can cause androgenic side effects in direct relation to their affinity for this receptor, and DHT has no known unique ability in this regard.

Benefits of DHT

While a lot of attention is being paid to the negative side effects of the androgen dihydrotestosterone, you should know that there are some known benefits to the strong androgenic activity brought about by this hormone as well. For example, DHT plays an important role in the organization and functioning of the central nervous system. Many neural cells contain active androgen receptors, and it is thought that there may even be a specific importance of dihydrotestosterone in this area of the body. Studies have shown DHT to have a profoundly greater impact in these cells compared to testosterone. More specifically, animal models demonstrated that both testosterone and DHT would result in increased androgen receptor proliferation in neural cells three and seven hours after being administered, however only DHT was able to sustain this increase at the twenty-one hour mark[35]. Although some might contend that this difference is simply due to DHT forming a more stable and lasting complex with the androgen receptor, others suggest that DHT and testosterone might even be affecting neural cells differently, such that the dihydrotestosterone-receptor complex and testosterone-receptor complex might be activating the transcription of different target genes.

The strong interaction between the central nervous system and skeletal muscles, collectively referred to as the neuromuscular system, is of key importance to the athlete. There appears to be little doubt that the ability of the body to adapt to training, and to activate nerve endings in muscle tissue, is reliant on the interactions of the neuromuscular system. Inhibiting the formation of DHT during a testosterone cycle may therefore inadvertently interfere with strength and muscle mass gains. This would explain why bodybuilders commonly report a drop in steroid potency when they add the 5-alpha reductase inhibitor finasteride to a testosterone cycle. Many complain strength and even muscle mass gains slow significantly when this medication is added, which would not make

sense if testosterone and androgen receptor activation in muscle tissue were solely responsible for growth. Clearly more is involved, and we cannot look at dihydrotestosterone simply as a side-effect hormone.

A Brief History of Anabolic/Androgenic Steroids

While it had been clear for many centuries that the testicles were crucial for the male body to properly develop, it was not until modern times that an understanding of testosterone began to form. The first solid scientific experiments in this area, which eventually led to the discovery and replication of testosterone (and related androgens), were undertaken in the 1800's. During this century a number of animal experiments were published, most of which involved the removal and/or implantation of testicular material from/in a subject. Although very crude in design by today's standards, these studies certainly laid the foundation for the modern field of endocrinology (the study of hormones). By the turn of the century, scientists were able to produce the first experimental androgen injections. These were actualized either through the filtering of large quantities of urine (for active hormones), or by extracting testosterone from animal testicles. Again, the methods were rough but the final results proved to be very enlightening.

Chemists finally synthesized the structure of testosterone in the mid 1930's, sparking a new wave of interest in this hormone. With the medical community paying a tremendous amount of attention to this achievement, the possible therapeutic uses for a readily available synthetic testosterone quickly became an extremely popular focus. Many believed the applications for this type of a medication would be extremely far reaching, with uses ranging from the maintenance of an androgen deficiency, to that of a good health and well being treatment for the sickly or elderly. During the infancy of such experimentation, many believed they had crossed paths with a true "fountain of youth".

Dihydrotestosterone and nandrolone, two other naturally occurring steroids, were also isolated and synthesized in the early years of steroid development. To make things even more interesting, scientists soon realized that the androgenic, estrogenic and anabolic activity of steroid hormones could be adjusted by altering their molecular structure. The goal of many researchers thereafter became to manufacture a steroid with extremely strong anabolic activity, but will display little or no androgenic/estrogenic properties. This could be very beneficial, because side effects will often become pronounced when steroid hormones are administered in supraphysiological amounts. A "pure" anabolic would theoretically allow the patient to receive only the beneficial effects of androgens (lean muscle mass gain, increased energy and recuperation etc.), regardless of the dosage. Some early success with the creation of new structures convinced many scientists that they were on the right track. Unfortunately none of this progress led researchers the their ultimate goal. By the mid 1950's, well over one thousand testosterone, nandrolone, and dihydrotestosterone analogues had been produced, but none proved to be purely anabolic compounds.

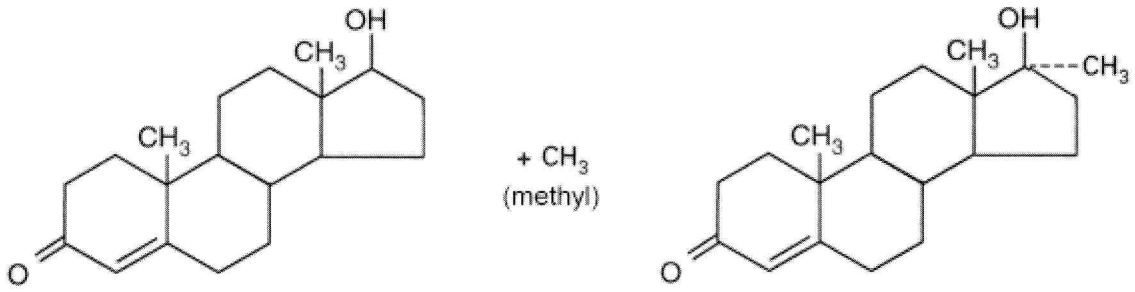
The failure to reach this goal was primarily due to an initial flawed understanding of testosterone's action. Scientists had noticed high levels of DHT in certain tissues, and believed this indicated an unusual receptor affinity for this hormone. This led to the belief that the human body had two different androgen receptors. According to this theory, one receptor site would respond only to testosterone (eliciting the beneficial anabolic effects), while the other is activated specifically by the metabolite, dihydrotestosterone. With this understanding, eliminating the conversion of testosterone to DHT was thought capable of solving the problem of androgenic side effects, as these receptors would have little or none of this hormone available for binding. More recently however, scientists have come to understand that only one type of androgen receptor exists in the human body. It is also accepted that no anabolic/androgenic steroid can possibly be synthesized that would participate only with receptors in tissues related to anabolism. DHT, which was once thought not to bind to the same receptor as testosterone, is now known to do so at approximately three to four times the affinity of its parent, and the unusual recovery of DHT from androgen responsive tissues is now attributed to the distribution characteristics of the 5a-reductase enzyme.

Synthetic AAS Development

In order to develop products that would be effective therapeutically, chemists needed to solve a number of problems with using natural steroid hormones for treatment. For example, oral dosing was a problem, as our basic steroids testosterone, nandrolone and dihydrotestosterone are ineffective when administered this way. The liver would efficiently break down their structure before reaching circulation, so some form of alteration was required in order for a tablet or capsule to be produced. Our natural steroid hormones also have very short half-lives in the body, so when administered by injection, an extremely frequent and uncomfortable dosing schedule is required if a steady blood level is to be achieved. Therefore, extending steroid activity was a major goal for many chemists during the early years of synthetic AAS development. Scientists also focused on the nagging problems of possible excess estrogenic buildup in the blood, particularly with testosterone, which can become very uncomfortable for patients undergoing therapy.

Methylated Compounds and Oral Dosing

Chemists realized that by replacing the hydrogen atom at the steroid's 17th alpha position with a carbon atom (a process referred to as *alkylation*), its structure would be notably resistant to breakdown by the liver. The carbon atom is typically added in the form of a methyl group (CH3), although we see oral steroids with an added ethyl (C2H5) grouping as well. A steroid with this alteration is commonly described as a C-17 alpha alkylated oral, although the terms methylated or ethylated oral steroid are also used. The alkyl group cannot be removed metabolically, and therefore inhibits reduction of the steroid to its inactive 17-ketosteroid form by occupying one of the necessary carbon bonds. Before long, pharmaceutical companies had utilized this advance (and others) to manufacture an array of effective oral steroids including methyltestosterone, Dianabol, Winstrol®, Anadrol 50®, Halotestin®, Nilevar, Orabolin and Anavar. The principle drawback to these compounds is that they place a notable amount of stress on the liver, which in some instances can lead to actual damage to this organ.



Testosterone Methyltestosterone

Because the alkyl group cannot be removed, it mediates the action of the steroid in the body. Methyltestosterone, for example, is not simply an oral equivalent of testosterone, as the added alkylation changes the activity of this steroid considerably. One major change we see is an increased tendency for the steroid to produce estrogenic side effects, to spite the fact that it actually lowers the ability of the hormone to interact with aromatase[36]. Apparently with 17-alkylation present on a steroid, aromatization (when possible) produces a more active form of estrogen (typically 17alpha-methyl or 17alpha-ethyl estradiol). These estrogens are more biologically active than estradiol due to their longer half-life and weaker tendency to bind with serum proteins. In some instances, 17alpha-alkylation will also enhance the ability of the initial steroid compound to bind with and activate the estrogen or progesterone receptor[37]. An enhancement of estrogenic properties is also obvious when we look at methandrostenolone, which is an alkylated form of boldenone (Equipoise®), and Nilevar, which is an alkylated form of the mild anabolic nandrolone. Dianabol is clearly more estrogenic than Equipoise®, a drug not noted for producing strong side effects of this nature. The same holds true for the comparison of Nilevar to Deca, a compound that we also know to be extremely mild in this regard.

C17 alpha alkylation also typically lowers the affinity in which the steroid binds to the androgen receptor, as is noted with the weak relative binding affinity of such popular agents as Dianabol and Winstrol (stanozolol). However, since this alteration also greatly prolongs the half-life of a steroid, as well as increases the tendency for it to exist in an unbound state, it creates a more potent anabolic/androgenic agent in both cases. This explains why Dianabol and stanozolol are notably effective in relatively lower weekly doses (often 140mg weekly will produce

notable growth) compared to injectables such as testosterone and nandrolone, which often need to reach doses of 300-400mg weekly for a similar level of effect.

Non-Alkylated Orals

In an attempt to solve the mentioned problems with liver toxicity we see with c17-alpha alkylated compounds, a number of other orals with different chemical alterations (such as Primobolan®, Proviron®, Andriol® and Anabolicum Vister) were created. Primobolan® and Proviron® are alkylated at the one position (methyl), a trait which also slows ketosteroid reduction. Andriol® uses a 17beta carboxylic acid ester (used with injectable compounds, discussed below), however, here the oil-dissolved steroid is sealed in a capsule and is intended for oral administration. This is supposed to promote steroid absorption through intestinal lymphatic ducts, bypassing the first pass through the liver. In addition to 1 methylation, Primobolan® also utilizes a 17 beta ester (acetate) to further protect against reduction to inactive form (here there is no lymphatic system absorption). Anabolicum Vister uses 17beta enol ether linkage to protect the steroid, which is very similar to esterification as the ether breaks off to release a steroid base (boldenone in this case). While all of these types of compounds do not place the same stress on the liver, they are also much less resistant to breakdown than 17 alkylated orals, and are ultimately less active milligram for milligram.

Esters and Injectable Compounds

You may notice that many injectable steroids will list long chemical names like testosterone cypionate and testosterone enanthate, instead of just testosterone. In these cases, the cypionate and enanthate are esters (carboxylic acids) that have been attached to the 17-beta hydroxyl group of the testosterone molecule, which increase the active life span of the steroid preparation. Such alterations will reduce the steroid's level of water solubility, and increase its oil solubility. Once an esterified compound has been injected, it will form a deposit in the muscle tissue (depot) from which it will slowly enter circulation. Generally the larger the ester chain, the more oil soluble the steroid compound will be, and the longer it will take for the full dosage to be released. Once free in circulation, enzymes will quickly remove the ester chain and the parent hormone will be free to exert its activity (while the ester is present the steroid is inert).

There are a wide number of esters, which can provide varying release times, used in medicine today. To compare, an ester like decanoate can extend the release of active parent drug into the blood stream for three to four weeks, while it may only be a extended for a few days with an acetate or propionate ester. The use of an ester allows for a much less frequent injection schedule than if using a water based (straight) testosterone, which is much more comfortable for the patient. We must remember when calculating dosages, that the ester is figured into the steroids measured weight. 100mg of testosterone enanthate, therefore, contains much less base hormone than 100mg of a straight testosterone suspension (in this case it equals 72mg of testosterone). In some instances, an ester may account for roughly 40% or more of the total steroid weight, but the typical measure is somewhere around 15% to 35%. Below are the free base equivalents for several popular steroid compounds.

100 mg of steroid as:	Approximate Free Equivalent:
Trenbolone acetate	87mg
Testosterone propionate	83mg
Testosterone enanthate	72mg
Testosterone cypionate	70mg
Testosterone undecanoate	63mg
Nandrolone phenylpropionate	67mg
Nandrolone decanoate	64mg

It is also important to stress the fact that esters do not alter the activity of the parent steroid in any way. They work only to slow its release. It is quite common to hear people speak about the properties of different esters, almost as if they can magically alter a steroid's effectiveness. This is really nonsense. Enanthate is not more powerful than cypionate (perhaps a few extra milligrams of testosterone released per injection, but nothing to note), nor is Sustanon some type of incredible testosterone blend. Personally, I have always considered Sustanon a very poor buy in the face of cheaper 250mg enanthate ampules. Your muscle cells see only testosterone; ultimately there is no difference. Reports of varying levels of muscle gain, androgenic side effects, water retention etc. are only issues of timing. Faster releasing testosterone esters will produce estrogen buildup faster simply because there is more testosterone free in the blood from the start of the cycle. The same is true when we state that Durabolin® is

a milder nandrolone for women compared to Deca. It is simply easier to control the blood level with a faster acting drug. Were virilization symptoms to become apparent, hormone levels will drop much faster once we stop administeration. This should not be confused with the notion that the nandrolone in Durabolin® acts differently in the body than that released from a shot of Deca-Durabolin®.

It is also worth noting that while the ester is typically hydrolyzed in general circulation, some will be hydrolyzed at the injection site where the steroid depot first contacts blood. This will cause a slightly higher concentration of both free steroid and ester in the muscle where the drug had been administered. On the plus side, this may equate to slightly better growth in this muscle, as more hormone is made available to nearby cells. Many bodybuilders have come to swear by the use of injection sites such as the deltoids, biceps and triceps, truly believing better growth can be achieved if the steroid is injected directly into these muscles. The negative to this is that the ester itself may be irritating to the tissues at the site of injection once it is broken free. In some instances it can be so caustic that the muscle itself will become swollen and sore due to the presence of the ester, and the user may even suffer a low-grade fever as the body fights off the irritant (the onset of such symptoms typically occurs 24-72 hours after injection). This effect is more common with small chain esters such as propionate and acetate, and can actually make a popular steroid such as Sustanon (which contains testosterone propionate) off-limits for some users who experience too much discomfort to justify using the drug. Longer chain esters such as decanoate and cypionate are typically much less irritating at the site of injection, and therefore are preferred by sensitive individuals.

Anabolic/Androgenic Dissociation

Although never complete, scientists had some success in their quest to separate the androgenic and anabolic properties of testosterone. A number of synthetic anabolic steroids had been developed as a result, with many being notably weaker and stronger than our base androgen. In order to first assess the anabolic and androgenic potential of each newly developed steroid, scientists had generally used rats as a model. To judge androgenic potency the typical procedure involved the post-administration measure (% growth) of the seminal vesicles and ventral prostate. These two tissues will often respond unequally to a given steroid, however, so an average of the two figures is used. Anabolic activity was most commonly determined by measuring the growth of the levator ani, a sex organ (not skeletal) muscle. This tissue may not be the most ideal one to use though, as it contains more androgen receptor than most skeletal muscles (the AR is still less abundant here than in target tissues such as the ventral prostate)[38 39]. In integrating both measures, the anabolic index is used, which relates the ratio of anabolic to androgenic response for a given steroid. An anabolic index greater than one indicates a higher tendency for anabolic effect, and therefore classifies the drug as an anabolic steroid. A measure lower than one in turn assesses the steroid as androgenic. There is some variance between experimental results and the actual real world experiences with humans, but (with a few exceptions) designations based on the anabolic index are generally accepted. Below are discussed a few factors that greatly effect anabolic/androgenic dissociation.

Nandrolone and 19-norandrogens

The section of this book dealing with DHT conversion is important, because it helps us understand the anabolic steroid nandrolone and many of its derivatives. Nandrolone is identical to testosterone except it lacks a carbon atom in the 19th position, hence its other given name 19-nortestosterone. Nandrolone is very interesting because it offers the greatest ratio of anabolic to androgenic effect of the three natural steroids (see: Synthetic AAS Chemistry). This is because it is metabolized into a less potent structure (dihydronandrolone) in androgen target tissues with high concentrations of the 5-alpha reductase enzyme, which is the exact opposite of what happens with testosterone. Apparently the removal of the c4-5 double bond, which normally increases the androgen receptor binding capability of testosterone, causes an unusual lowering of this ability with nandrolone. Instead of becoming three to four times more potent, it becomes several times weaker. This is a very desirable trait if you want to target anabolic effects over androgenic. This characteristic also carries over to most synthetic steroids derived from nandrolone, making this an attractive base steroid to use in the synthesis of new, primarily anabolic, steroids.

5-alpha Irreducible Steroids

When we look at the other mild anabolic steroids Primobolan®, Winstrol® and Anavar, none of which are derived from nandrolone, we see another interesting commonality. These steroids are DHT derivatives that are unaffected by 5alpha-reductase, and therefore neither become weaker or stronger in androgen responsive target tissues with high concentrations of this enzyme. In essence, they have a very balanced effect between muscle and androgen tissues, making them outwardly less androgenic than testosterone.

This is why these steroids are technically classified as anabolics, and are undeniably less troublesome than many other steroids in terms of promoting androgenic side effects. However, if we wanted to look for the absolute least androgenic steroid, the title would still go to nandrolone (or perhaps one of its derivatives). Female bodybuilders should likewise take note that to spite the recommendations of others, steroids like Anavar, Winstrol and Primo are not the least risky steroids to use. This is of great importance, as male sex hormones can produce many undesirable and permanent side effects when incorrectly taken by females (See: Side Effects, Virilization).

3-alpha Hydroxysteroid Dehydrogenase

The 3-alpha hydroxysteroid dehydrogenase enzyme is also important, because it can work to reduce the anabolic potency of certain steroids considerably. As follows, not all potent binders of the androgen receptor are, as a rule, great muscle-building drugs, and this enzyme is an important factor. Dihydrotestosterone is a clear example. Just as the body converts testosterone to DHT as a way to potentiate its action in certain tissues (skin, scalp, prostate etc.), it also has ways of countering the strong activity of DHT, in other tissues where it is unneeded. This is accomplished by the rapid reduction of DHT to its inactive active metabolites, namely androstanediol, before it reaches the androgen receptor. This activity occurs via interaction with the 3-alpha hydroxysteroid dehydrogenase enzyme. This enzyme is present in high concentrations in certain tissues, including skeletal muscle, and DHT is much more open to alteration by it than other steroids that possess a c4-5 double-bond (like testosterone)[40]. This causes dihydrotestosterone to be an extremely poor anabolic, to spite the fact that it actually exhibits a much higher affinity for the cellular androgen receptor than most other steroids. Were it able to reach the cellular androgen receptor without first being metabolized by 3a-HSD, it certainly would be a formidable muscle-building steroid. Unfortunately this is not the case, explaining why injectable dihydrotestosterone preparations (no longer commercially produced) were never favorite drugs among athletes looking to build mass. This trait is also shared by the currently popular oral androgen Proviron®, which is, in essence, just an oral form of DHT (1-methyl dihydrotestosterone to be specific) and known to be an extremely poor tissue builder.

Anabolics and Potency

One must remember that being classified as an anabolic just means that the steroid is more inclined to produce muscle growth than androgenic side effects. Since both effects are mediated through the same receptor, and growth is not produced by androgen receptor activation in muscle tissue alone (other CNS tissues, for example, are integral to this process as well), we find that a reduction in the androgenic activity of a compound will often coincide with a similar lowering of its muscle-building effectiveness. If we are just looking at overall muscle growth, androgenic steroids (usually potent due to their displaying a high affinity to bind with the androgen receptor in all tissues) are typically much more productive muscle-builders than anabolics, which usually bind with lower affinity in many tissues. In fact, with all of the analogues produced throughout the years, the base androgen testosterone is still considered to be one of the most effective bulking agents. The user must simply endure more side effects when acquiring his or her new muscle with this type of drug. Individuals wishing to avoid the stronger steroids will, therefore, make a trade-off, accepting less overall muscle gain in order to run a more comfortable cycle.

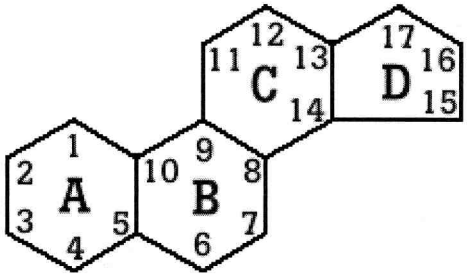
RBA Assay:

Another way of evaluating the potential ratio of anabolic to androgenic activity is the more recent practice of simply comparing the relative binding affinity (RBA) of various steroids for the androgen receptor in rat skeletal muscle versus prostate. When we look at the detailed study published in 1984, we see a clear trend of uniformity. Aside from dihydrotestosterone and Proviron® (mesterolone) which undergo rapid enzymatic reduction in muscle tissue, the remaining anabolic/androgenic steroids seem to bind with near equal affinity to receptors in both tissues. This study also discusses the unique activity of testosterone and nandrolone compounds, which are good substrates for the 5a-reductase enzyme found in androgen target tissues (such as the prostate), and seem to provide the most notable variance between anabolic and androgenic effect in humans. When it comes to real-world use, these particular substrates tend to behave differently (somewhat more so that other steroids) in regards to their anabolic and androgenic profiles than the animal models would suggest.

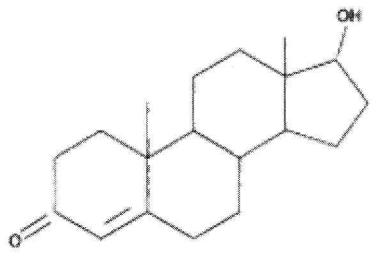
Compound	Human SHBG	Rabbit Muscle	Rat Muscle	Rat Prostate	Ratio M vs. P
methyltrienolone	<.01	1	1	1	1
dihydrotestosterone	1	.07	<.01	.46	.03
mesterolone	4.4	.21	.08	.25	.32
testosterone	.19	.07	.23	.15	1.53
nandrolone	.01	.20	.24	.60	.4
methyltestosterone	.05	.1	.11	.13	.85
methenolone	.03	.09	.24	.14	1.67
stanozolol	.01	.03	.02	.03	.6
methandrostenolone	.02	.02	.02	.03	.75
fluoxymesterone	<.01	.02	.01	.02	.77
oxymetholone	<.01	<.01	<.01	<.01	1.54
ethylestrenol	<.01	.01	<.01	<.01	2

RBA of various anabolic/androgenic steroids as competitors for human SHBG binding of DHT, and for receptor binding of methyltrienolone in cytosol from rabbit, rat skeletal muscle and prostate. Source: Endocrinology 114(6):2100-06 1984 June, "Relative Binding Affinity of Anabolic-Androgenic Steroids...", Saartok T; Dahlberg E; Gustafsson JA.

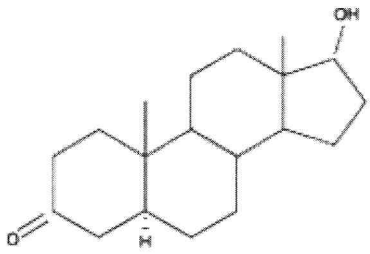
Synthetic AAS Chemistry



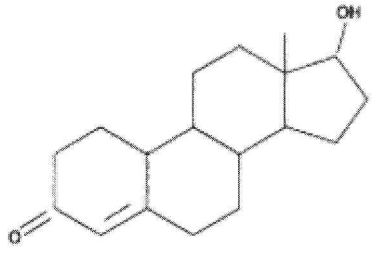
Steran Nucleus
(All natural and synthetic AAS hormones share this base structure)



Testosterone



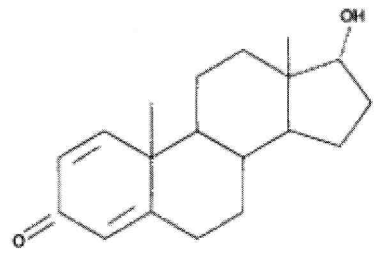
Dihydrotestosterone



Nandrolone

All anabolic/androgenic steroids are preparations containing one of the above three natural steroid hormones, or chemically altered derivatives thereof. In creating new synthetic compounds, one of the three natural hormones is selected as a starting point, typically due to the possession of particular traits that may be beneficial for the new compound. For instance, of the three natural steroids above, dihydrotestosterone is the only steroid devoid of the possibility of aromatization and 5-alpha reduction. It was likewise a very popular choice in the creation of synthetics that lack estrogenic activity and/or exhibit a more balanced androgenic to anabolic activity ratio. Nandrolone was typically used when even lower androgenic action is desired, due to its weakening upon interaction with the 5-alpha reductase enzyme. Nandrolone also aromatizes much more slowly than testosterone. Testosterone is our most powerful muscle-building hormone, and also exhibits strong androgenic activity due to its conversion to a more potent steroid (dihydrotestosterone) via 5-alpha reductase.

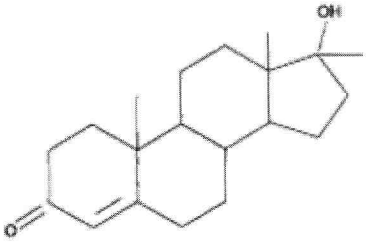
Testosterone derivatives



Boldenone (+c1-2 double bond)

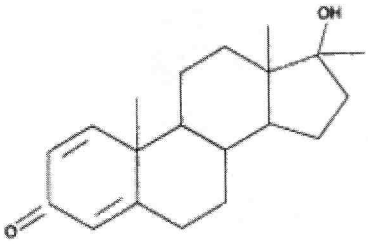
Boldenone is testosterone with an added double-bond between carbon atoms one and two. However, this bond changes the activity of the steroid considerably. First, it dramatically slows aromatization, such that boldenone converts to estradiol at about half the rate of testosterone. Secondly, this bond causes the steroid to be a very poor substrate for the 5-alpha reductase enzyme. The more active 5-alpha reduced metabolite 5alpha-dihydroboldenone is produced only in very small amounts in humans. The hormone instead tends to convert via 5-

beta reductase to 5beta-dihydroboldenone (a virtually inactive androgen). This makes it lean towards being an anabolic instead of an androgen, although both traits are still notably apparent with this steroid. The c1-2 double bond also slows the hepatic breakdown of the structure, increasing its resistance to 17-ketosteroid deactivation and its functional half-life and oral bioavailability.



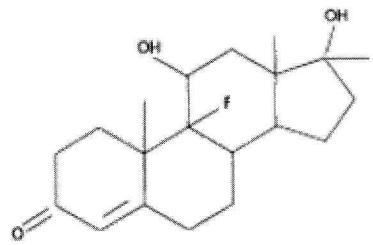
Methyltestosterone (+ 17alpha methyl)

This is the most basic derivative of testosterone, differing only by the added 17-alpha methylation that makes the steroid orally active. Conversion to 17-alpha methylestradiol makes this steroid extremely estrogenic, to spite the fact that this alteration actually reduces interaction with the aromatase enzyme.



Methandrostenolone (+c 1-2 double bond; 17-alpha methyl)

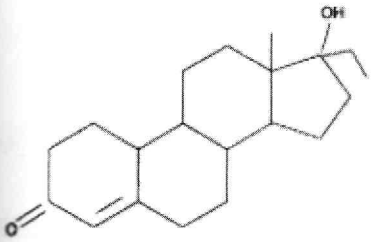
In many regards, methandrostenolone is very similar to boldenone, as it too exhibits reduced estrogenic and androgenic activity due to the c1-2 double-bond. However, this steroid does have a reputation of being somewhat estrogenic though, owing to the fact that it converts to a highly active form of estrogen (17alpha-methylestradiol See: Methylated Compounds and Oral Dosing). Methandrostenolone is also much more active milligram for milligram, as the 17-alpha methyl group also gives it a longer half-life and allows it to exist in a more free state than its cousin boldenone.



Fluoxymesterone (+11-beta hydroxyl; 9-fluoro; 17-alpha methyl)

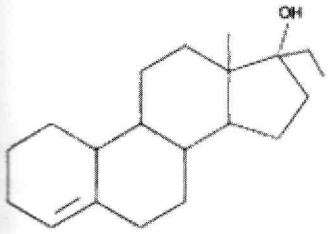
Halotestin is a c-17alpha alkylated oral derivative of testosterone. The 11-beta group functions to inhibit aromatization, so there is no estrogen conversion at all with this steroid. It also works to lower the affinity of this steroid toward restrictive serum binding proteins. I have no explanation for the function of the 9-fluoro group at this time, however, can say that it neither blocks aromatization nor 5-alpha reduction. This is supported by the fact that other 9-fluoro steroids have been shown to aromatize, as well as studies showing fluoxymesterone to be an active substrate for the 5-alpha reductase enzyme.

Nandrolone derivatives



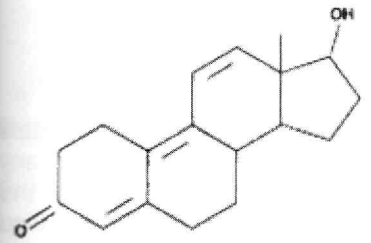
Norethandrolone (+ 17-alpha ethyl)

Norethandrolone is simply nandrolone with an added 17-alpha ethyl group. This alteration is rarely used with anabolic/androgenic steroids, and is much more commonly found with synthetic estrogens and progestins. Although 17-ethylation inhibits 17-ketosteroid reduction just as well as 17-methylation, and therefore allows this steroid to exhibit a similarly high level of oral activity, this group also tends to increase progesterone receptor binding. Norethandrolone is clearly a "troublesome" hormone in terms of water retention, fat gain and gynecomastia, which may in part be due to its heightened binding to this receptor.



Ethylestrenol (+17-alpha ethyl; - 3 Keto)

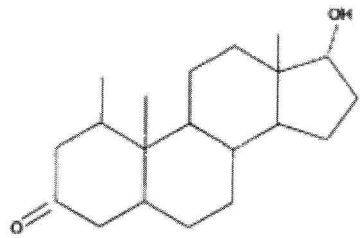
Ethylestrenol is an oral derivative of nandrolone, very similar in structure to norethandrolone. In fact, it differs from this steroid only by the removal of the 3-keto group, which is vital to androgen receptor binding. As such, ethylestrenol is possibly the weakest steroids milligram for milligram ever sold commercially. Any activity this steroid does exhibit is likely from its conversion to norethandrolone, which does seem to occur with some affinity (apparently the 3 oxygen group is metabolically added to this compound without much trouble). This is probably the most interesting trait of ethylestrenol, which is an undistinguished compound otherwise.



Trenbolone (+ c9-10 double bond; c11-12 double bond)

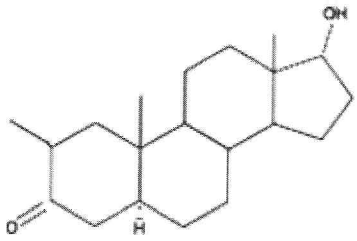
Although a derivative of nandrolone, the two additional double-bonds present on trenbolone make any similarities to its parent hormone extremely difficult to see. First, the 9-10 bond inhibits aromatization. Nandrolone is very slowly aromatized, however, some estrogen is still produced from this steroid. Not so with trenbolone. The 11-12 bond additionally increases androgen receptor binding. This steroid also does not undergo 5-alpha reduction like nandrolone, and as such does not share the same dissociation between anabolic and androgenic effects (trenbolone is much more androgenic in comparison).

Dihydrotestosterone derivatives



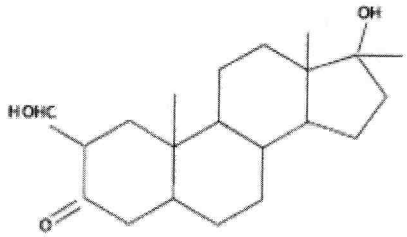
Mesterolone (+ 1-methyl)

Mesterolone is a potent orally active derivative of dihydrotestosterone. Similar to methenolone, it possesses a non-toxic 1-methyl group, which increases its resistance to hepatic breakdown. This alteration does not increase the stability of the 3-keto group however, and as such, this steroid is a poor anabolic like its parent.



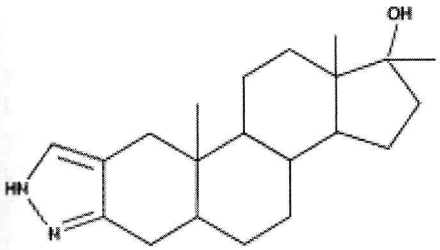
Drostanolone (+ 2-methyl)

Drostanolone is simply dihydrotestosterone with an added 2-methyl group. This addition greatly increases the stability of the 3-keto group, vital to androgen binding. As such, the activity of this steroid in muscle tissue is greatly enhanced (see: Anabolic/Androgenic Dissociation).



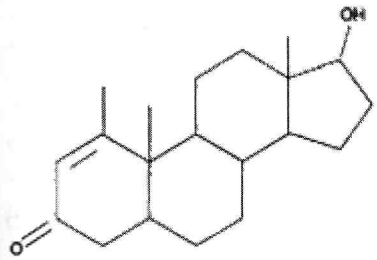
Oxymetholone (+2 hydroxymethylene; 17alpha-methyl)

Oxymetholone is an orally active derivative of dihydrotestosterone. The 17-methyl group is well understood at this point as we have discussed it with many steroids, however, the 2-hydroxymethylene group is not seen on any other commercial steroid. We do know that this group greatly enhances anabolic potency by increasing the stability of the 3-keto group, and that the configuration of this substituent also appears to allow this steroid to bind and activate the estrogen receptor.



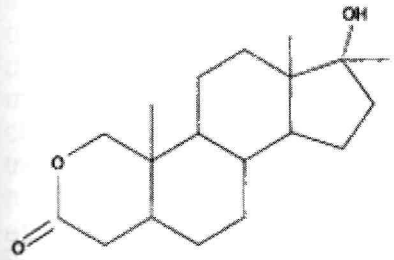
Stanozolol (+ 3,2 pyrazol; 17-alpha methyl)

Stanozolol is a potent anabolic steroid, owing to the fact that the 3-2 pyrazol group creates a stable configuration off the A-ring that allows for androgen receptor binding (this steroid is one of the few that does not possess an actual 3-keto group). As such, it is highly active in muscle tissue, unlike dihydrotestosterone.



Methenolone (+ 1-methyl; 1-2 double bond).

Methenolone also is a potent anabolic steroid, due to the fact that the c1-2 double bond increases the stability of the 3-keto group. The 1-methyl group works to increase its oral bioavailability, making methenolone (as methenolone acetate) one of the few orally active non-17-alkylated orals. The c 1-2 bond may also help increase hepatic resistance (slightly) to 17-ketosteroid deactivation as well.



Oxandrolone (2-oxygen substitution; 17-alpha methyl)

Oxandrolone is an orally active derivative of dihydrotestosterone, due to its 17-methylation. It also differs from DHT by the substitution of its 2-carbon molecule with oxygen. This is the only commercial steroid to carry this group, and further, the only to have a modification to the base carbon structure of the Steran nucleus. The 2-oxo group increases resistance of the 3-keto group to metabolism considerably, making oxandrolone a potent anabolic.

Steroid Nomenclature

Perhaps not obvious at first glance, there is a naming convention in place that was used to create identities for the various anabolic/androgenic steroid hormones. This typically involves forming a root word to convey the structural base of the steroid, and signifying other unique structural characteristics by including appropriate prefixes or suffixes. Below, we will look at the common roots, prefixes and suffixes used in steroid nomenclature, and identify them, as they are used in the various commercial compound names. As you will see, the adoption of names like nandrolone, methandrostenolone and ethylestrenol were not as arbitrary as one might imagine. This section is also helpful if you wish to understand the deeper chemical designations for the various substances that one might find in the medical literature, which involve the exclusive use of this terminology (such as is the representation of methandrostenolone as 17b-hydroxy-17a-methylandrosta-1,4-dien-3-one).

Common prefixes and suffixes used in steroid naming:

Structural Property	Prefix	Suffix
Carbonyl (C=O)	oxo-; keto-	-one
Hydroxyl	hydroxy-	-ol
Double Bond (C=C)		-ene; -en
Methyl	meth-; methyl-	
Ethyl	eth-; ethyl-	

Common roots used in steroid naming:

Androstane	Base carbon structure of dihydrotestosterone (no double-bond)
Androstene	Base carbon structure of or similar to testosterone (one double-bond)
Androstadiene	Base carbon structure similar to methandrostenolone (two double-bonds; di-ene)
Estren; Estra *also: Norandrostene*	Base structure of nandrolone (19-norandrostene) and estrogen

Common Commercial Compound Names:

Name	***Taken From***	***Incorporated Into Name As***
Boldenone	[17b-ol, androstadiene, 3-one]	BOL DEN ONE
Ethylestrenol	[17a ethyl, estren, 17b-ol]	ETHYL ESTREN OL
Fluoxymesterone	[9-fluoro, 11b-hydroxyl, 17a-methyl, testosterone, 3-one]	FLU OXY ME STER ONE
Mesterolone	[1-methyl, dihydrotestosterone, 17b-ol, 3-one]	ME STER OL ONE
Methandienone	[1a-methyl, androstadiene, 3-one]	METH ANDIEN ONE
Methandrostenolone	[17a-methyl, androstadiene, 17b-ol, 3-one]	METH ANDROSTEN OL ONE
Methenolone	[1-methyl, c1-2 double bond (en), 17bol, 3-one]	METH EN OL ONE
Nandrolone	[norandrostene, 17b-ol, 3-one]	NANDR OL ONE
Norethandrolone	[19-nor, 17a-ethyl, (nor)androstene, 17b-ol, 3-one]	NOR ETH ANDR OL ONE
Oxandrolone	[2-oxy, androstane, 17b-ol, 2-one]	OX ANDR OL ONE
Oxymetholone	[2-hydroxymethylene, 17a-Methyl, 17b-ol, 3-one]	OXY METH OL ONE
Stanozolol	[Stanolone (androstanolone, DHT), 2-pyrazol, 17b-ol]	STANO ZOL OL
Trenbolone	[tri-en, 17b-ol, 3-one]	TREN BOL ONE

Steroid Side Effects

The action of testosterone can be both beneficial and detrimental to the body. On the plus side, this hormone has a direct impact on the growth of muscle tissues, the production of red blood cells and overall well being of the organism. But it may also negatively effect (among other things) the production of skin oils, growth of body, facial and scalp hair, and the level of both "good" and "bad" cholesterol in the body. In fact, men have a shorter average life span than women, which is believed to be largely due to the cardiovascular defects that this hormone may help bring about. Testosterone will also naturally convert to estrogen in the male body, a hormone with its own unique set of effects. As we have discussed earlier, raising the level of estrogen in men can increase the tendency to notice water retention, fat accumulation, and the development of female tissues in the breast (gynecomastia). Clearly we see that most of the "bad" side effects from steroids are simply those actions of testosterone that we are not looking for when taking a steroid. Raising the level of testosterone in the body will simply enhance both its good and bad properties, but for the most part we are not having "toxic" reactions to these drugs. A notable exception to this is the possibility of liver damage, which is a worry isolated to the use of c17-alpha alkylated oral steroids. Unless the athlete is taking anabolic/androgenic steroids abusively for a very long duration, side effects rarely amount to little more than a nuisance.

One could make a case that periodic steroid use might even be a healthy practice. Clearly a person's physical shape can relate closely to one's overall health and well being. Provided some common sense is paid to health checkups, drug choice, dosage and off-time, how can we say for certain that the user is worse off for doing so? This position is, of course, very difficult to publicly justify with steroid use being so deeply stigmatized. Since this can be a very lengthy discussion, I will save the full health, moral and legal arguments for another time. For now I would like to run down the list of popularly discussed side effects, and include any current treatment/avoidance advice where possible.

Acne

Rampant acne is one of the more obvious indicators of steroid use. As you know, teenage boys generally endure periods of irritating acne as their testosterone levels begin to peak, but this generally subsides with age. But when taking anabolic/androgenic steroids, an adult will commonly be confronted with this same problem. This is because the sebaceous glands, which secrete oils in the skin, are stimulated by androgens. Increasing the level of such hormones in the skin may therefore enhance the output of oils, often causing acne to develop on the back, shoulders, and face. The use of strongly androgenic steroids in particular can be very troublesome, in some instances resulting in very unsightly blemishes all over the skin. To treat acne, the athlete has a number of options. The most obvious is to be very diligent with washing and topical treatments, so as to remove much of the dirt and oil before the pores become clogged. If this proves insufficient, the prescription acne drug Accutaine® might be a good option. This is a very effective medication that acts on the sebaceous glands, reducing the level of oil secreted. The athlete could also take the ancillary drug Proscar®/Propecia® (finasteride) during steroid treatment, which reduces the conversion of testosterone into DHT, lowering the tendency for androgenic side effects with this hormone. It is of note however that this drug is more effective at warding off hair loss than acne, as it more specifically effects DHT conversion in the prostate and hair follicles. It is also important to note that testosterone is the only steroid that really converts to dihydrotestosterone, and only a few others actually convert to more potent steroids via the 5a-reductase enzyme at all. Many steroids are also potent androgens in their own right, such as Anadrol 50® and Dianabol. As such, they can exert strong androgenic activity in target tissues without 5a-reduction to a more potent compound, which makes Propecia® useless. One can also simply opt take primarily "anabolic" compoiunds, which impart comparable less androgenic activity. For sensitive individuals attempting to build mass, nandrolone would, therefore, be a much better option than testosterone.

Aggression

Aggressive behavior can be one of the scarier sides to steroid use. Men are typically more aggressive than women because of testosterone, and likewise the use of steroids (especially androgens) can increase a person's aggressive tendencies. In some instances this can be a benefit, helping the athlete hit the weights more intensely or perform better in a competition. Many professional powerlifters and bodybuilders take a particular liking to this effect. But on the other hand, there is nothing more unsettling than a grown man, bloated with muscle mass, who cannot control his temper. A steroid user who displays an uncontrollable rage is clearly a danger to himself and others. If an athlete is finding himself getting agitated at minor things during a steroid cycle, he should certainly find

a means to keep this from getting out of hand. Remembering to take a couple of deep breaths at such times can be very helpful. If such attempts prove to be ineffective, the offending steroids should be discontinued. The bottom line is that if you lack the maturity and self-control to keep your anger in check, you should not be using steroids.

Anaphylactic Shock

Anaphylactic shock is an allergic reaction to the presence of a foreign protein in the body. It most commonly occurs when an individual has an allergy to things like a specific medication (e.g. penicillin), insect bites, industrial/household chemicals, foods (commonly nuts, shellfish, fruits) and food additives/preservatives (particularly sulfur). With this sometimes-fatal disorder the smooth muscles are stimulated to contract, which may restrict a person's breathing. Symptoms include wheezing, swelling, rash or hives, fever, a notable drop in blood pressure, dizziness, unconsciousness, convulsions or death. This reaction is not really seen with hormonal products like anabolic/androgenic steroids, but this may change with the rampant manufacture of counterfeit pharmaceuticals. Being that there are no quality controls for black market producers, toxins might indeed find their way into some preparations (particularly injectable compounds). My only advice would be to make every attempt to use only legitimately produced drug products, preferably of First World origin. When anaphylactic shock occurs, it is most commonly treated with an injection of epinephrine. Individuals very sensitive to certain insect bites are familiar with this procedure, many of whom keep an allergy kit (for the self administration of epinephrine) close at hand.

Birth Defects

Anabolic/androgenic steroids can have a very pronounced impact on the development of an unborn fetus. Adrenal Genital Syndrome in particular is a very disturbing occurrence, in which a female fetus can develop male-like reproductive organs. Women who are, or plan to become pregnant soon, should never consider the use of anabolic steroids. It would also be the best advice to stay away from these drugs completely for a number of months prior to attempting the conception of a child, so as to ensure the mother has normal hormonal chemistry. Although anabolic/androgenic steroids can reduce sperm count and male fertility, they are not linked to birth defects what taken by someone fathering a child.

Blood Clotting Changes

The use of anabolic/androgenic steroids is shown to increase prothrombin time, or the duration it will take for a blood clot to form. This basically means that while an individual is taking steroids, he/she may notice that it takes slightly longer than usual for a small cut or nosebleed to stop seeping blood. During the course of a normal day this is hardly cause for alarm, but it can lead to more serious trouble if a severe accident occurred, or an unexpected surgery was needed. Realistically, the changes in clotting time are not extremely dramatic, so athletes are usually only concerned with this side effect if planning for a surgery. The clotting changes brought about by anabolic steroids are amplified with the use of medications like Aspirin, Tylenol and especially anticoagulants, so your doctor should be informed of their use (steroids) if undergoing any notable treatment with these types of drugs.

Cancer

Although it is a popular belief that steroids can give you cancer, this is actually a very rare phenomenon. Since anabolic/androgenic steroids are synthetic version of a natural hormone that your body can metabolize quite easily, they usually place a very low level of stress on the organs. In fact, many steroidal compounds are safe to administer to individuals with a diagnosed liver condition, with little adverse effect. The only real exception to this is with the use of c17 alpha alkylated compounds, which due to their chemical alteration are somewhat liver toxic. In a small number of cases (primarily with Anadrol 50®), this toxicity has lead to severe liver damage and subsequently cancer. But we are speaking of a statistically insignificant number in the face millions of athletes who use steroids. These cases also tended to be very ill patients, not athletes, who were using extremely large dosages for prolonged periods of time. Steroid opponents will sometimes point out the additional possibility of developing Wilm's Tumor from steroid abuse, which is a very serious form of kidney cancer. Such cases are so rare however, that no direct link between anabolic/androgenic steroid use and this disease has been conclusively established. Provided the athlete is not abusing methylated oral substances, and is visiting a doctor during heavier cycles, cancer should not be much of a concern.

Cardiovascular Disease

As mentioned earlier, the use of anabolic/androgenic steroids may have an impact on the level of LDL (low density lipoprotein), HDL (high density lipoprotein) and total cholesterol values. As you probably know, HDL is considered the "good" cholesterol since it can act to remove cholesterol deposits from the arteries. LDL has the opposite effect, aiding in the buildup of cholesterol on the artery walls. The general pattern seen with steroid use is a lowering of HDL concentrations, while total and LDL cholesterol numbers increase. The ratio of HDL to LDL values is usually more important than one's total cholesterol count, as these two substances seem to balance each other in the body. If these changes are exacerbated by the long-term use of steroidal compounds, it can clearly be detrimental to the cardiovascular system. This may be additionally heightened by a rise in blood pressure, which is common with the use of strongly aromatizable compounds.

It is also important to note that due to their structure and form of administration, most 17alpha alkylated oral steroids have a much stronger negative impact on these levels compared to injectable steroids. Using a milder drug like Winstrol® (stanozolol), in hopes HDL level changes will also be mild, may therefore not turn out to be the best option. One study comparing the effect of a weekly injection of 200mg testosterone enanthate vs. only a 6mg daily oral dose of Winstrol® demonstrates this well[41]. After only six weeks, stanozolol was shown to reduce HDL and HDL-2 (good) cholesterol by an average of 33% and 71% respectively. The HDL reduction (HDL-3 subfraction) with the testosterone group was only an average of 9%. LDL (bad) cholesterol also rose 29% with stanozolol, while it actually dropped 16% with the use of testosterone. Those concerned with cholesterol changes during steroid use may likewise wish to avoid oral steroids, and opt for the use of injectable compounds exclusively.

We must also note that estrogens generally have a favorable impact on cholesterol profiles. For example, estrogen replacement therapy in postmenopausal women is regularly linked to a rise in HDL cholesterol and a reduction in LDL values. Likewise the aromatization of testosterone to estradiol may be beneficial in preventing a more dramatic change in serum cholesterol due to the presence of the hormone. A recent study investigated just this question by comparing the effects of testosterone alone (280 mg testosterone enanthate weekly), vs. the same dose combined with an aromatase inhibitor (250mg testolactone 4 times daily)[42]. Methyltestosterone was also tested in a third group, at a dose of 20mg daily. The results were quite enlightening. The group using only testosterone enanthate showed no significant decrease in HDL cholesterol values over the course of the 12-week study. After only four weeks, the group using testosterone plus an aromatase inhibitor displayed a reduction on average of 25%. The methyltestosterone group noted an HDL reduction of 35% by this point, and also noted an unfavorable rise in LDL cholesterol. This clearly should make us think a little more closely about estrogen maintenance during steroid therapy. Aside from deciding whether or not it is actually necessary in any given circumstance, drug choice may also be an important consideration. For example, the estrogen receptor antagonist Nolvadex® does not seem to exhibit antiestrogenic effects on cholesterol values, and in fact often raises HDL levels. Using this to combat the side effects of estrogen instead of an aromatase inhibitor such as Arimidex® or Cytadren® may therefore be a good idea, particularly for those who are using steroids for longer periods of time.

Since heart disease is one of the top killers worldwide, steroid using athletes (particularly older individuals) should not ignore these risks. If nothing else it is a very good idea to have your blood pressure and cholesterol values measured during each heavy cycle, making sure to discontinue the drugs should a problem become evident. It is also advisable to limit the intake of foods high in saturated fats and cholesterol, which should help minimize the impact of steroid treatment. Since blood pressure and cholesterol levels will usually revert back to their pre-treated norms soon after steroids are withdrawn, long-term damage is not a common worry.

Depression

Obviously steroid use will have an impact on hormone levels in the body, which in turn may result in a change in one's general disposition or mood. On the one hand, we might see very aggressive behavior. But for some people there is also, at times, the other extreme side, depression. This can occur in certain individuals, whom are psychologically sensitive to an imbalance in androgen and estrogen levels. This is most common with male bodybuilders, at times when anabolic/androgenic steroids are discontinued. Given a deeply suppressed endogenous testosterone level, it may take time for one's normal hormonal balance to return. During this period, estrogen levels may be more stable than testosterone, as our bodies can produce it from adrenal hormones. The result may be a protracted window of time where estrogen seems to be the more dominant sex hormone. For some, this window can be filled with feelings of emotional sensitivity, sadness, and lack of motivation (symptoms of depression).

Depression may also occur during the course of a steroid cycle, particularly with the sole use of anabolics. Although these compounds are mild in comparison to androgens, many can still suppress the endogenous production of testosterone. If the testosterone level drops significantly during treatment, the administered anabolics may not provide enough of an androgen level to compensate, and a marked loss of motivation and sense of well-being may result. The best advice when looking to avoid cycle or post-cycle depression is to closely monitor drug intake and withdrawal. The use of a small weekly testosterone dose might prove very effective if added to a mild dieting/anabolic cycle, warding off feelings of boredom and apathy to training. Of course a strong steroid cycle should always be discontinued with the proper use of ancillary drugs (Nolvadex®, Arimidex®, HCG, Clomid® etc.). Although tapering schedules are very common, they are not an effective way to restore endogenous testosterone levels.

Gynecomastia

Gynecomastia is the medical term for the development of female breast tissues in the male body. This occurs when the male is presented with an unusually high level of estrogen, particularly with the use of strong aromatizing androgens such as testosterone and Dianabol. The excess estrogen can act upon receptors in the breast and stimulate the growth of mammary tissues. If left unchecked, this can lead to an actual obvious and unsightly tissue growth under the nipple area, in many cases taking on a very feminine appearance. To fight this side effect during steroid therapy, many find it necessary the use some form of estrogen maintenance medication. This includes an estrogen antagonist such as Clomid® or Nolvadex®, which blocks estrogen from attaching to and activating receptors in the breast and other tissues, or an aromatase inhibitor such as Femara® or Arimidex®, which blocks the enzyme responsible for the conversion of androgens to estrogens. Aromatase inhibitors like this are currently the most effective options, but also the most costly.

It is worth noting however, that many believe a slightly elevated estrogen level may help the athlete achieve a more pronounced muscle mass gain during a cycle (see: Estrogen Aromatization). With this in mind many athletes decide to use anti-estrogens only when it is necessary to block gynecomastia. It is of course still a good idea to always keep an anti-estrogen on-hand when administering an aromatizable steroid, so that it is readily accessible should trouble become evident. Puffiness or swelling under the nipple is one of the first signs of pending gynecomastia, often accompanied by pain or soreness in this region (an effect termed gynecodynea). This is a clear indicator that some type of anti-estrogen is needed. If the swelling progresses into small, marble like lumps, action absolutely must be taken immediately to treat it. Otherwise, if the steroids are continued at this point without ancillary drug use, the user will likely be stuck with unsightly tissue growth that can only be removed with a surgical procedure.

It is also important to mention that progestins seem to augment the stimulatory effect of estrogens on mammary tissue growth. There appears to be a strong synergy between these two hormones here, such that gynecomastia might even be able to occur with the help of progestins, without excessive estrogen levels being necessary. Since many anabolic steroids, particularly those derived from nandrolone, are known to have progestational activity, we must not be lulled into a false sense of security. Even a low estrogen producer like Deca can potentially cause gyno in certain cases, again fostering the need to keep anti-estrogens close at hand if you are very sensitive to this side effect.

Hair loss

The use of highly androgenic steroids can negatively impact the growth of scalp hair. In fact, the most common form of male pattern hair loss is directly linked to the level of androgens in such tissues, most specifically the stronger DHT metabolite of testosterone. The technical term for this type of hair loss is androgenetic alopecia, which refers to the interplay of both the male androgenic hormones and a genetic predisposition in bringing about this condition. Those who suffer from this disorder are shown to posses finer hair follicles and higher levels of DHT in comparison to a normal, hairy scalp. But since there is a genetic factor involved, many individuals will not ever see signs of this side-effect, even with heavy steroid use. Clearly those individuals who are suffering from (or have a familial predisposition for) this type of hair loss should be very cautious when using the stronger drugs like testosterone, Anadrol 50®, Halotestin® and Dianabol.

In many instances, the renewal of lost hair can be very difficult, so avoiding this side-effect before it occurs is the best advice. For those who need to worry, the decision should probably be made to either stick with milder substances (Deca-Durabolin® most favored), or use the ancillary drug Propecia®/Proscar® (finasteride) when taking testosterone, methyltestosterone or Halotestin. Propecia® is a very effective hair loss medication, which inhibits the 5-alpha reductase enzyme specifically in the hair follicles and prostate. However, it offers us little benefit with drugs that are highly androgenic without 5alpha reduction, the most notable offenders being Anadrol

50® and Dianabol. We must also remember that all anabolic/androgenic steroids activate the androgen receptor, and can, likewise, all promote hair loss given the right dosage and conditions.

Headaches

Athletes sometimes report an increased frequency of headaches when using anabolic/androgenic steroids. This seems to be most common during heavier bulking cycles, when an individual is utilizing strongly estrogenic compounds. One should not simply take an aspirin and ignore this problem, as it is may indicate a more troubling side effect of steroid use, high blood pressure. Since high blood pressure invites a number of unwanted health risks, monitoring it on a regular schedule is important during heavy steroid use, especially if the individual is experiencing headaches. Some athletes choose to lower their blood pressure in such cases with a prescription medication like Catapres, but most find this an appropriate time to discontinue steroid use. Milder anabolics, which generally display little or no ability to convert to estrogen, are also more acceptable options for individuals sensitive to blood pressure increases. Less seriously, many headaches are due to simple strain on the neck and scalp muscles. The athlete may be lifting with much more intensity during a steroid cycle, and as a result may place added strain on these muscles. In this case, a short break from training, and some general rest, will often take care of the problem. Of course if anyone is experiencing a very serious or persistent headache, a visit to the doctor may be in order.

High Blood Pressure/Hypertension

Athletes using anabolic/androgenic steroids will commonly notice a rise in blood pressure during treatment. High blood pressure is most often associated with the use of steroids that have a high affinity for estrogen conversion, such as testosterone and Dianabol. As estrogen builds in the body, the level of water and salt retention will typically elevate and lead to increased blood pressure. This may be further amplified by the added stress of intense weight training and rapid weight gain. Since hypertension (high blood pressure) can place a great deal of stress on the body, this side effect should not be ignored. If it is left untreated, high blood pressure can increase the likelihood for heart disease, stroke or kidney failure. Warning signs that one may be suffering from hypertension include an increased tendency to develop headaches, insomnia or breathing difficulties. In many instances these symptoms do not become evident until BP is seriously elevated, so a lack of these signs is no guarantee that the user is safe. Obtaining your blood pressure reading is a very quick and easy procedure (either at a doctors office, pharmacy or home); steroid-using athletes should certainly be monitoring BP values during stronger cycles so as to avoid potential problems.

If an individual's blood pressure values are becoming notably elevated, some action should/must be taken to control it. The most obvious is to avoid the continued use of the offending steroids, or at least to substitute them with milder, non-aromatizing compounds. It is also of note that although aromatizing steroids are typically involved, nonaromatizing androgens like Halotestin® or trenbolone are occasionally also linked to high blood pressure, so these are perhaps not the ideal alternatives in such a situation. The athlete also has the option of seeking the benefit of high blood pressure medications such as diuretics, which can dramatically lower water and salt retention. Catapres (clonidine HCL) is also a popular medication among athletes, because in addition to its blood pressure lowering properties, it has also been documented to raise the body's output of growth hormone.

Immune System Changes

The use of anabolic/androgenic steroids has been shown to produce changes in the body that may impact an individual's immune system. These changes can be both good and bad for the user. For instance, during steroid treatment, many athletes find they are less susceptible to viral illnesses. New studies involving the use of compounds like oxandrolone and Deca-Durabolin® with HIV+ patients seem to support this claim, clearly showing that these drugs can have a beneficial effect on the immune system. Such therapies are, in fact, catching on in recent years, and many doctors are now less reluctant to prescribe these drugs to their ill patients. But just as a person may be less apt to notice illness during steroid treatment, the discontinuance of steroids can produce a rebound effect in which the immune system is less able to fight off pathogens. This most likely coincides with the rebound activity/production of cortisol, a catabolic hormone in the body, which may act to suppress immune system functioning. When the administered steroids are withdrawn, an androgen deficient state is often endured until the body is able to rebalance hormone production. Since testosterone and cortisol seem counter each other's activity in many ways, the absence of a normal androgen level may place cortisol in an unusually active state. During this period of imbalance, cortisol will not only be stripping the body of muscle mass, but may also cause the athlete to be more susceptible to colds, flu, etc. The proper use of ancillary drugs (anti-estrogens, testosterone

stimulating drugs) is the most common suggestion for helping to avoid this problem, which will hopefully allow the user to restore a proper balance of hormones once the steroids are removed.

We also cannot ignore the other possibility that steroids could actually increase cortisol levels in the body during treatment. Termed hypercortisolemia, this effect is a common occurrence with anabolic/androgenic steroid therapy. This is because anabolic/androgenic steroids may interfere with the ability for the body to clear corticosteroids from circulation, due to the fact that in their respective pathways of metabolism these hormones share certain enzymes. When overloaded with androgens competing for the same enzymes, cortisol may be broken down at a slower rate, and levels of this hormone will in turn begin to build. Due to their strong tendency to inhibit the activity of the 3beta hydroxysteroid dehydrogenase enzyme, oral c17 alpha alkylated orals may be particularly troublesome in regards to elevated cortisol levels, as again this is a common pathway for corticosteroid metabolism. Though an elevated cortisol level is not a common concern during typical steroid cycles, problems can certainly become evident when these drugs are used at very high doses or for prolonged periods of time. This, of course, may lead to the athlete becoming "run-down" and more susceptible to illness, as well as foster a more over-trained and static (less anabolic) state of metabolism.

Kidney Stress/Damage

Since your kidneys are involved in the filtration and removal of byproducts from the body, the administration of steroidal compounds (which are largely excreted in the urine) may cause them some strain. Actual kidney damage is most likely to occur when the steroid user is suffering from severe high blood pressure, as this state can place an undue amount of stress on these organs. There is actually evidence to suggest that steroid use can be linked to the onset of Wilm's Tumor in adults, which is a rapidly growing kidney tumor normally seen in children and infants. However, such cases are so rare that no conclusive link has been established. Obviously the kidneys are vital to one's heath, so the possibility of any kind of damage (although low) should not be ignored during heavy steroid treatment. If the user is noticing a darkening of color (in some cases a distinguishable amount of blood), or pain/difficulty when urinating, kidneys strain might be a legitimate concern. Other warning signs include pain in the lower back (particularly in the kidney areas), fever and edema (swelling). If organ damage is feared, the administered steroidal compounds should be discontinued immediately, and the doctor paid a visit to rule out any serious trouble.

Since kidney stress/damage is generally associated with the use of stronger aromatizing compounds such as testosterone and Dianabol (which often raise blood pressure), individuals sensitive to high blood pressure/kidney stress should avoid such compounds until health concerns are safely addressed. If steroid use is still necessitated by the individual, it may be a good idea to avoid the stronger compounds and opt for one of the milder anabolics. Primobolan®, Anavar and Winstrol®, for example, do not convert to estrogen at all, and may be acceptable options. Also favorable drugs in this regard are Deca-Durabolin® and Equipoise®, which have only a low tendency to convert to estrogen.

Liver Stress/Damage

Liver stress/damage is not a side-effect of steroid use in general, but is specifically associated with the use of c17 alpha alkylated compounds. As mentioned earlier, these structures contain chemical alterations that enable them to be administered orally. In surviving a first pass by the liver, these compounds place some level of stress on the organ. In some instances, this has led to severe damage, even fatal liver cancer. The disease peliosis hepatitis is one worry, which is an often life-threatening condition where the liver develops blood-filled cysts. Liver cancer (hepatic carcinoma) has also been noted in certain cases. While these very serious complications have occurred on certain occasions where liver-toxic compounds were prescribed for extended periods, it is important to stress that this is not very common with steroid using athletes. Most of the documented cases of liver cancer have in fact been in clinical situations, particularly with the use of the powerful oral androgen Anadrol 50® (oxymetholone). This may be directly related to the high dosage of this preparation, as Anadrol 50® contains a whopping 50mg of active steroid per tablet. This is a considerable jump from other oral preparations, most of which contain 5mg or less of a substance. With one Anadrol 50® tablet, the liver will therefore have to process (roughly) the equivalent of 10 Dianabol tablets. This obvious stress is further amplified when we look at the unusually high dosage schedule for ill patients receiving this medication. With Anadrol 50®, the manufacturer's recommendations may call for the use of as many as 8 or 10 tablets daily. This is a far greater amount than most athletes would ever think of consuming, with three or four tablets per day being considered the upper limit of safety. It is also important to note that the actual number of cases involving liver damage have been few, and have not been a significant enough of a problem to warrant discontinuing this compound. Methyltestosterone, the first steroid shown to cause

liver trouble, is also still available as a prescription drug in this country. The average recreational steroid user who takes toxic orals at moderate dosages for relatively short periods is therefore unlikely to face devastating liver damage.

Although severe liver damage may occur before the onset of noticeable symptoms, it is common to notice jaundice during the early stages of such injury. Jaundice is characterized by the buildup of bilirubin in the body, which in this case will usually result from the obstruction of bile ducts in the liver. The individual will typically notice a yellowing of the skin and eye whites as this colored substance builds in the body tissues, a clear sign to terminate the use of any c17 alpha alkylated steroids. In most instances, the immediate withdrawal of these compounds is sufficient to reverse and prevent any further damage. Of course, the athlete should avoid using orals for an extended period of time, if not indefinitely, should jaundice occur repeatedly during treatment. It is also a good idea to visit your physician during oral treatment in order to monitor liver enzyme values. Since liver stress will be reflected in your enzyme counts well before jaundice is noticed, this can remove much of the worry with oral steroid treatment.

Prostate Enlargement

Prostate cancer is currently one of the most common forms of cancer in males. Benign prostate enlargement (a swelling of prostate tissues often interfering with urine flow) can precede/coincide this cancer, and is clearly an important medical concern for men who are aging. Prostate complications are believed to be primarily dependent on androgenic hormones; particularly the strong testosterone metabolite DHT in normal situations, much in the same way estrogen is linked to breast cancer in women. Although the connection between prostate enlargement/cancer and steroid use is not fully established, the use of steroids may theoretically aggravate such conditions by raising the level of androgens in the body. It is, therefore, a good idea for older athletes to limit/avoid the intake of strong 5-alpha reducible androgens like testosterone, methyltestosterone and Halotestin, or otherwise use Proscar® (finasteride), which was specifically designed to inhibit the 5-alpha reductase enzyme in scalp and prostate tissues. This may be an effective preventative measure for older athletes who insist on using these compounds. However, drugs like Dianabol, Anadrol 50® and Proviron, which do not convert to DHT yet are still potent androgens, are not effected by its use. It is also important to mention that not only androgens, but also estrogens, are necessary for the advancement of this condition. It appears that the two work synergistically to stimulate benign prostatic growth, such that one without the other would not be enough to cause it. It has, therefore, been suggested that a non-aromatizable compound like DHT may be a safer option for older men looking for androgen replacement therapy than testosterone. Anti-estrogens might even turn out to be more effective at treating BPH than a drug like finasteride, which is used to lower androgenic activity in the prostate. Estrogen suppression is easier to accomplish in males, and should be accompanied with less side effects. It would also be very sound advice, regardless of steroid use, for individuals over 40 to have a physician check the prostate on a regular basis.

Sexual Dysfunction

The functioning of the male reproductive system depends greatly on the level of androgenic hormones in the body. Therefore, the use of synthetic male hormones may have a dramatic impact on an individual's sexual wellness. On one extreme, we may see a man's libido and erection frequency become significantly heightened. This is most commonly seen with the use of strongly androgenic steroids, which seem to have the most dramatic stimulating impact on this system. In some instances, this can reach the point of becoming problematic, although more often than not, the athlete is simply much more active and sexually aggressive during the intake of steroids.

On the other extreme, we may also see a lack of sexual interest, possibly to the point of impotency. This occurs mainly when androgenic hormones are very low. This will often happen after a steroid cycle is discontinued, as the endogenous production of testosterone is commonly suppressed during the cycle. Removing the androgen (from an outside source) leaves the body with little natural testosterone until this imbalance is corrected. The loss of its' metabolite DHT is particularly troubling, as this hormone may have a strong affect on the reproductive system that may not be apparent with other less androgenic hormones. Therefore, it is a very good idea to use testosterone-stimulating drugs like HCG and/or Clomid®/Nolvadex® when coming off of a strong cycle, so as to reduce the impact of steroid withdrawal. Impotency/sexual apathy may also occur during the course of a steroid cycle, particularly when it is based strictly on anabolic compounds. Since all "anabolics" can suppress the manufacture of testosterone in the body, the administered drugs may not be androgenic enough to properly compensate for the testosterone loss. In such a case, the user might opt to include a small androgen dosage (perhaps a weekly testosterone injection), or again reverse/prevent the androgen suppression with the use of a medication like HCG.

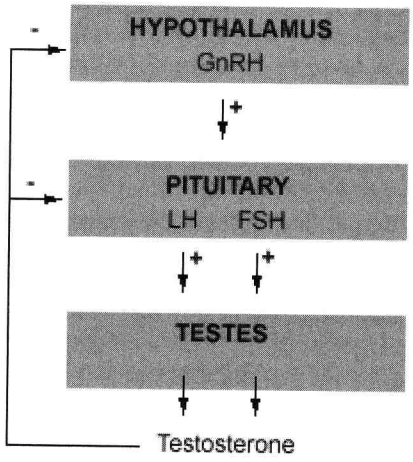
It is also interesting to note that it is not always simply an androgen vs. anabolic issue. People will often respond very differently to an equal dose of the same drug. While one individual may notice sexual disinterest or impotency, another may become extremely aggressive. It is, therefore, difficult to predict how someone will react to a particular drug before having used it.

Stunted Growth

Many anabolic/androgenic steroids have the potential to impact an individual's stature if taken during adolescence. Specifically, steroids can stunt growth by stimulating the epiphyseal plates in a person's long bones to prematurely fuse. Once these plates are fused, future linear growth is not possible. Even if the individual avoids steroid use subsequently, the damage is irreversible and he/she can be stuck at the same height forever. Not even the use of growth hormone can reverse this, as this powerful hormone can only thicken bones when used during adulthood. Interestingly enough, it is not the steroids themselves, but the buildup of estrogen that causes the epiphyseal plates to fuse. Women are shorter than men on average because of this effect of estrogen, and likewise the use of steroids that readily convert to estrogen can prematurely suppress/halt a person's growth. In fact, the use of steroids like Anavar, Winstrol® and Primobolan® (which do not convert to estrogen) can actually increase one's height if taken during adolescence, as their anabolic effects will promote the retention of calcium in the bones. This would also hold true for non-aromatizing androgens such as trenbolone, Proviron® and Halotestin®. It is still good common sense to advise adolescents to avoid steroid use, at least until their bodies are fully mature and steroid use will have a less dramatic impact.

Testicular Atrophy

The human body always prefers to remain in a very balanced hormonal state, a tendency known as homeostasis. When the administration of androgens from an outside source causes a surplus of hormone, it will cause the body to stop manufacturing its own testosterone. Specifically, this happens via a feedback mechanism where the hypothalamus detects a high level of sex steroids (including androgens, progestins and estrogens) and shuts off the release of GnRH (Gonadotropin Releasing Hormone, formerly referred to as luteinizing hormone releasing hormone). This, in turn, causes the pituitary to stop releasing luteinizing hormone and FSH (follicle stimulating hormone), the two hormones (primarily LH) that stimulate the Leydig's cells in the testes to release testosterone (negative feedback inhibition has been demonstrated at the pituitary level as well). Without stimulation by LH and FSH, the testes will be in a state of production limbo, and may shrink from inactivity. In extreme cases the steroid user can notice testicles that are unusually and frighteningly small. However, this effect is temporary, and once the drugs are removed (and hormone levels rebalanced) the testicles should return to their original size. Many regular steroid users find this side-effect quite troubling, and use HCG during a steroid cycle in order to try to maintain testicular activity (and size) during treatment. The more estrogenic androgens (testosterone, Anadrol 50® and Dianabol) are most dramatic in this regard, and are therefore poor choices for individuals who seriously want to avoid testicle shrinkage. Non-aromatizing anabolics would be a better option, however be warned that all steroids will have an impact on the production of testosterone if taken at an anabolically effective dosage (yes, even Anavar and Primobolan®).



The Hypothalamic-Pituitary-Testicular Axis: The hypothalamus releases Gonadotropin Releasing Hormone, which stimulates the pituitary to release luteinizing hormone and follicle stimulating hormone. This promotes the release of testosterone from the testes. Testosterone, as well as estrogens and progestins, in turn cause negative feedback inhibition at the hypothalamus (and to some extent the pituitary).

Water and Salt Retention

Many anabolic/androgenic steroids can increase the amount of water and sodium stored in body tissues. In some instances, steroid induced water retention can bring about a very bloated appearance to the body (hands, arms, face etc.), which will also reduce the visibility of muscle features (loss of definition). Athletes often ignore this side effect, particularly during bulking cycles when the excess water stored in the muscles, joints and connective tissues will help to improve an individual's overall strength. With the use of many strong androgens, water retention can account for much of the initial strength and body weight gain during steroid treatment, with "water-weight" sometimes amounting to ten or more pounds.

Although water retention may not be the most unwelcome side effect during a bulking cycle (greater strength and mass), it can lead to dangerous problems such as high blood pressure and kidney damage. The body is clearly under more strain when dealing with an unusually high level of water, so athletes should not simply ignore this. Water retention is most specifically associated with the presence of estrogen in the body, and is therefore common with the use of aromatizing compounds (such as testosterone and Dianabol). If water retention becomes an obvious problem during a cycle, the use of an anti-estrogen (Nolvadex®, Proviron®) may help minimize it. An antiaromatase like Arimidex® is, in fact, the most effective option, a drug that inhibits the conversion of testosterone to estrogen. Sometimes the athlete will alternately opt to use a diuretic, which can rapidly shed the water so as to achieve a more comfortable/attractive physique in a short period of time. This is a common practice when preparing for a competition, as diuretic use allows the user a great level of control over water stores. Of course, discontinuing the offending compounds, or substituting them with a milder anabolic would be the simplest option for recreational steroid users.

Virilization

Since anabolic/androgenic steroids are synthetic male hormones, they can produce a number of undesirable changes when introduced into the female body. This includes the possibility of "virilization", which refers to the tendency for women to develop masculine characteristics when taking these drugs. Virilization symptoms include a deepening or hoarsening of the voice, changes in skin texture, acne, menstrual irregularities, increased libido, hair loss (scalp), body/facial/pubic hair growth and an enlargement of the clitoris. In extreme cases the female genitalia can become very disfigured, and may actually take on a penis-like appearance. Clearly, women must be very careful when considering the use of steroids, especially since most virilization symptoms are irreversible. The stronger androgenic compounds should be off-limits, with cautious female athletes restricting themselves to the use of only mild anabolics such as Winstrol®, Primobolan®, Anavar and Durabolin® (the shorter acting nandrolone). Nandrolone is actually the preferred hormone, as it displays the lowest level of androgenic to anabolic activity. Since even these milder anabolics have the potential to cause problems, users should additionally remember to be conservative with drug dosages and duration of intake. After each cycle a notable break from treatment would be a good idea as well, so that the body has sufficient time to reestablish a hormonal balance.

Steroid Safety: Studies with Real-World Dosages

If you so much as mention anabolic steroids to the average person, you usually get some cross looks in response. State that you are actually considering a cycle, and you are likely to be lectured about the tremendous heath risks you are about to undertake; how your hair might fall out and testicles disappear, or your body eaten away by cancer. Or maybe you will just lose you mind to uncontrolled fits of psychotic rage, or suffer a life-threatening heart attack. You'll probably hear something like, "Is all that really worth it... to build a little more muscle?" Clearly, the American public has been given a very strong message about steroids: stay far away from them, they are DEADLY! You can't convince too many people that smoking a joint will *REALLY* cause a 16-year-old kid to pull out his dad's gun and shoot his friend in the face, but, for some reason, the "over the top" anti-drug message with steroids seems to have worked. Most people are terrified of them.

Those actually taking anabolic steroids usually see things very differently. They believe the dangers are terribly exaggerated in the media. In fact, these athletes will routinely point out that the medical literature for the past 50 years fails to make much note of any serious consequences of steroid use, with most clinical studies looking quite favorably on these drugs. Steroid opponents, on the other hand, will still make sure you know that bodybuilders take much larger doses of steroids than those used in medical situations; therefore, in much greater danger than the patients using them. Who is right? Is that occasional cycle really a serious health risk? This month I would like to touch on this debate by looking closely at three medical studies that were published recently. They concern not small clinical doses, but a level of steroid usage that any recreational bodybuilder would recognize as sufficient for building muscle. Many markers of safety are assessed in these papers, giving us a fairly good indication of what dangers, realistically, are presented.

600mg/wk of Testosterone

The first is a testosterone dose-response study published in the American Journal of Physiology Endocrinology and Metabolism in July of 2001, which looked at the effects of various doses of testosterone enanthate on body composition, muscle size, strength, power, sexual and cognitive functions, and various markers of health[43]. 61 normal men, ages 18-35, participated in this investigation. They were divided into five groups, with each receiving weekly injections of 25, 50, 125, 300 or 600 milligrams for a period of 20 weeks. This treatment period was preceded by a control (no drug) period of 4 weeks, and followed by a recovery period of 16 weeks. Markers of strength and lean body mass gains were the greatest with larger doses of testosterone, with the 600mg group gaining slightly over 17 pounds of fat-free mass on average over the 20 weeks of steroid therapy. There were no significant changes in prostate-specific antigen (PSA), liver enzymes (liver stress), sexual activity, or cognitive functioning at any dose. The only negative trait noted was a slight HDL (good) cholesterol reduction in all groups except those taking 25mg. The worst reduction of 9 points was noted in the 600mg group, which still averaged 34 points after 20 weeks of treatment. All groups, except this one, remained in the normal reference range for males (40-59 points).

600mg/wk of Nandrolone

Next we look at a study conducted with HIV+ men, which charted the lean-mass-building effects of nandrolone decanoate[44]. 30 people participated in this investigation, with each given the same (high) weekly dose of this drug. Half underwent resistance training so that two groups (trained and untrained) were formed. The dosing schedule was quite formidable, beginning with 200mg on the first week, 400mg on the second, and 600mg for the remaining 10 weeks of peak therapy. Doses were slowly reduced from weeks 13 to 16 to withdraw patients slowly from the drug. Potential negative metabolic changes were looked at closely including cholesterol and lipid levels (including subfractions of HDL and LDL), triglycerides, insulin sensitivity and fasting glucose levels. Even with the high dosages used here, no negative changes were noted in total or LDL cholesterol, triglycerides, or insulin sensitivity. In fact, the group also undergoing resistance exercise noticed significant improvements in LDL particle size distribution, lipoprotein(a) levels, and triglyceride values, which all indicate improved cardiovascular disease risk. Carbohydrate metabolism was also significantly improved in this group. The only negative impact noted during this study was a reduction in HDL (good) cholesterol values similar to that noted with the testosterone study, with an 8-10 point reduction noted between both groups.

100mg/day of Anadrol

Lastly, we find a study looking at the potent oral steroid oxymetholone (Anadrol)[45]. This steroid is thought by bodybuilders to be one of the most dangerous ones around, who as a group seem to treat it with both a lot of respect and caution. It is not common to find them exceeding the doses and intake durations of this investigation, making it a very good representation of real-world Anadrol usage. This study involves 31 elderly men, between the ages of 65 and 80. The men were divided into three groups, with each taking 50mg, 100mg or placebo daily for a 12-week period. Changes in lean body mass and strength were measured, as well as common markers of safety including total, LDL and HDL cholesterol levels, serum triglycerides, PSA (prostate-specific antigen) and liver enzymes. Muscle mass and strength gains were again relative to the dosage taken, with the end results being similar to those noted with 20 weeks of testosterone enanthate therapy at 125mg or 300mg per week (about 6.4 and 12 lb of lean body mass gained for the 50mg and 100mg doses respectively). There were no significant changes in PSA, total or LDL cholesterol values, or fasting triglycerides; however, there was a significant reduction in HDL cholesterol values (reduced 19 and 23 points for the 50mg and 100mg groups respectively). Liver enzymes (transaminases AST and ALT) increased only in the 100mg group, but the changes were not dramatic, and were not accompanied by hepatic enlargement or the development of any serious liver condition.

Adding It All Up

One hundred and twenty one men participated in these three studies, which involved the use of high doses of steroids for periods of three to five months. It may be shocking to most of the staunch opponents of steroid use, but all of the men participating were still alive at the conclusion of their respective investigations. An unbiased assessment of the metabolic changes and health risks does not seem to reveal any short-term significant dangers. The main negative impact of steroid use in all three cases was a reduction in good (HDL) cholesterol values, which is a legitimate concern when it comes to assessing one's risk for developing cardiovascular disease. However, it is uncertain if a short-lived increase in this particular risk factor will relate to any tangible damage to one's health. It is also unknown how much, if any of this is offset by the other positive metabolic changes that were seen to accompany combined steroid use and exercise. Logic would seem to suggest that the very periodic use of steroids, under parameters similar to these studies, should entail relatively minimal risks to ones health overall. At the very least, it is extremely difficult to argue that an isolated cycle with a moderate drug dose, such as those used here, is tantamount to playing Russian roulette with your body, as most media campaigns against the use of these drugs would seem to suggest.