There you go hydro, now drop some links!

Anabolic pharmacology by Bill Roberts. Drug profiles and articles.

Info provided by "fighter guy"

Oral steroids Drug Active half-life
Anadrol / Anapolan50 (oxymetholone) - 8 to 9 hours
Anavar (oxandrolone) - 9 hours
Dianabol (methandrostenolone, methandienone) - 4.5 to 6 hours
Methyltestosterone - 4 days
Winstrol (stanozolol)
(tables or depot taken orally) - 9 hours

Depot steroids Drug Active half-life
Deca-durabolin (Nandrolone decanate) - 15 days
Equipoise - 14 days
Finaject (trenbolone acetate) - 3 days
Primobolan (methenolone enanthate) - 10.5 days
Sustanon or Omnadren - 15 to 18 days
Testosterone Cypionate - 12 days
Testosterone Enanthate - 10.5 days
Testosterone Propionate - 4.5 days
Testosterone Suspension - 1 day
* Winstrol (stanozolol) - 1 day

*Winstrol depot does not actually possess a classical half-life because it is unesterified. Instead, the microcrystals dissolve slowly. Once they have all...
dissolved levels of the drug fall very rapidly. It is still an important consideration, and we have included it with a half-life of one day.

Ancillaries Drug Active half-life
Arimidex - 3 days
Clenbuterol - 1.5 days
Clomid - 5 days
Cytadren - 6 hours
Ephedrine - 6 hours
T3 - 10 hours

Detection times

Anavar - 3 weeks
Anadrol - 2 months
Andriol - 1 week
Boldenone Undecylenate - 4-5 months
Clen - 4-5 Days
DBol - 5 weeks
Deca - 18 months
Ephedrin - 4-5 Days
Halo - 2 months
Nandrolon Phenylprop - 12 months
Nilevar - 5-6 weeks
Parabolan - 4-5 weeks
Proviron - 5 weeks
Primo Depot - 4-5 weeks
Sustanon - 3 months
Spiropent - 4-5 days
Test cyp - 3 months
Test enat - 3 months
Test Prop - 2-3 weeks
Trenbolon Acet - 4-5 weeks
*Test supspension No metabolites. t/e should be back to normal in days
Equipoise - 4-5months
Winny oral - 3 weeks
Winny inj - 2 months

Title: Re: AAS Studies/Links/Literature

Test esters: By Bill Llewellyn

Here is an article I got some time back and pasted into notepad, I do not remember the source, but it is a pretty good source of info for understanding esters. I decided to post it because I have answered a few questions regarding esters over the last few days and thought it would be of help.

One of the most misunderstood subjects in the world of steroids is the ester--the mechanism by which injectable esterified steroids like testosterone cypionate, testosterone enanthate, and Sustanon work. If you take a quick look around the Internet you will probably find countless articles that consider one
form of a steroid far more effective than another. Arguments over the superiority of cypionate to enanthate, or Sustanon to all other testosterones are of course very common. Such arguments are in all practicality, baseless. In this report we'll take an authoritative look at the ester and what specifically it does to a steroid.

WHAT AN ESTER IS, AND HOW IT WORKS

I'm sure that if you have taken an interest in anabolic steroids you have noticed the similarities on the labeling of many drugs. Let's look at testosterone for example. One can find compounds like testosterone cypionate, enanthate, propionate, heptylate; caproate, phenylpropionate, isocaproate, decanoate, acetate, the list goes on and on. In all such cases the parent hormone is testosterone, which had been modified by adding an ester (enanthate, propionate etc.) to its structure. The following question arises: What is the difference between the various esterified versions of testosterone in regards to their use in bodybuilding?

An ester is a chain composed primarily of carbon and hydrogen atoms. This chain is typically attached to the parent steroid hormone at the 17th carbon position (beta orientation), although some compounds do carry esters at position 3 (for the purposes of this article it is not crucial to understand the exact position of the ester). Esterification of an injectable anabolic/androgenic steroid basically accomplishes one thing: it slows the release of the parent steroid from the site of injection. This happens because the ester will notably lower the water solubility of the steroid, and increase its lipid (fat) solubility. This will cause the drug to form a deposit in the muscle tissue, from which it will slowly enter into circulation as it is picked up in small quantities by the blood. Generally, the longer the ester chain, the lower the water solubility of the compound, and the longer it will take to for the full dosage to reach general circulation.

Slowing the release of the parent steroid is a great benefit in steroid medicine, as free testosterone (or other steroid hormones) previously would remain active in the body for a very short period of time (typically hours). This would necessitate an unpleasant daily injection schedule if one wished to maintain a continuous elevation of testosterone (the goal of testosterone replacement therapy). By adding an ester, the patient can visit the doctor as infrequently as once per month for his injection, instead of having to constantly re-administer the drug to achieve a therapeutic effect. Clearly without the use of an ester, therapy with an injectable anabolic/androgenic would be much more difficult.

Esterification temporarily deactivates the steroid molecule. With a chain blocking the 17th beta position, binding to the androgen receptor is not possible (it can exert no activity in the body). In order for the compound to become active the ester must therefore first be removed. This automatically occurs once the compound has filtered into blood circulation, where esterase enzymes quickly cleave off (hydrolyze) the ester chain. This will restore the necessary hydroxyl (OH) group at the 17th beta position, enabling the drug to attach to the appropriate receptor. Now and only now will the steroid be able to have an effect on skeletal muscle tissue. You can start to see why considering testosterone cypionate much more potent than enanthate makes little sense, as your muscles are seeing only free testosterone no matter what ester was used to deploy it.
There are many different esters that are used with anabolic/androgenic steroids, but again, they all do basically the same thing. Esters vary only in their ability to reduce a steroid's water solubility. An ester like propionate for example will slow the release of a steroid for a few days, while the duration will be weeks with a decanoate ester. Esters have no effect on the tendency for the parent steroid to convert to estrogen or DHT (dihydrotestosterone: a more potent metabolite) nor will it effect the overall muscle-building potency of the compound. Any differences in results and side effects that may be noted by bodybuilders who have used various esterified versions of the same base steroid are just issues of timing. Testosterone enanthate causes estrogen related problems more readily than Sustanon, simply because with enanthate testosterone levels will peak and trough much sooner (1-2 week release duration as opposed to 3 or 4). Likewise testosterone suspension is the worst in regards to gyno and water bloat because blood hormone levels peak so quickly with this drug. Instead of waiting weeks for testosterone levels to rise to their highest point, here we are at most looking at a couple of days. Given an equal blood level of testosterone, there would be no difference in the rate of aromatization or DHT conversion between different esters. There is simply no mechanism for this to be possible.

There is however one way that we can say an ester does technically effect potency; it is calculated in the steroid weight. The heavier the ester chain, the greater is its percentage of the total weight. In the case of testosterone enanthate for example, 250mg of esterified steroid (testosterone enanthate) is equal to only 180mg of free testosterone. 70mgs out of each 250mg injection is the weight of the ester. If we wanted to be really picky, we could consider enanthate slightly MORE potent than cypionate (I know this goes against popular thinking) as its ester chain contains one less carbon atom (therefore taking up a slightly smaller percentage of total weight). Propionate would of course come out on top of the three, releasing a measurable (but not significant) amount more testosterone per injection than cypionate or enanthate.

IN CONCLUSION

While the advent of esters certainly constitutes an invaluable advance in the field of anabolic steroid medicine, clearly you can see that there is no magic involved here. Esters work in a well-understood and predictable manner, and do not alter the activity of the parent steroid in any way other than to delay its release. Although the lure surrounding various steroid products like testosterone cypionate, Sustanon, Omnadren etc. certainly makes for interesting conversation, realistically it just amounts to misinformation that the athlete would be better off ignoring. Testosterone is testosterone and anyone who is going to tell you one ester form of this (or any) hormone is much better than another one should do a little more research, and a lot less talking.

ESTER PROFILES

Sustanon: The "king" of testosterone blends. The four different testosterone esters in this product certainly look appealing to the consumer, there is no denying that. But for the athlete I think it is all just a matter of marketing (Hell, why buy one ester when you can get four?). In clinical
situations I can see some strong uses for it. If you were undergoing testosterone replacement therapy for example, you would probably find Sustanon a much more comfortable option than testosterone enanthate. You would need to visit the doctor less frequently for an injection, and blood levels should be more steadily maintained between treatments. But for the bodybuilder who is injecting 4 ampules of Sustanon per week, there is no advantage over other testosterone products. In fact, the high price tag for Sustanon usually makes it a very poor buy in the face of cheaper testosterone enanthate/cypionate. Bodybuilders should probably stop looking at the four ester issue, and stick with totals (Sustanon is just a 250mg testosterone ampule). Were enanthate to be available for say $10 per amp of 250mg, and Sustanon priced nearly double that, buying the Sustanon would be like throwing money away. If you could get nearly double the milligram amount for the same price with enanthate, this is the better product to go with hands down. Leave the high priced stuff for the guys who don’t know any better.

Acetate: Chemical Structure C2H4O2.

Also referred to as Acetic Acid; Ethylic acid; Vinegar acid; vinegar; Methanecarboxylic acid. Acetate esters delay the release of a steroid for only a couple of days. Contrary to what you may have read, acetate esters do not increase the tendency for fat removal. Again, there is no known mechanism for it to do so. This ester is used on oral primobolan tablets (metenolone acetate), Finaplix (trenbolone acetate) implant pellets, and occasionally testosterone.

Propionate: Chemical Structure C3H6O2.

Also referred to as Carboxyethane; hydroacrylic acid; Methylacetic acid; Ethylformic acid; Ethanecarboxylic acid; metacetic acid; pseudoacetic acid; Propionic Acid. Propionate esters will slow the release of a steroid for several days. To keep blood levels from fluctuating greatly, propionate compounds are usually injected two to three times weekly. Testosterone propionate and methandriol dipropionate (two separate propionate esters attached to the parent steroid methandriol) are popular items.

Phenylpropionate: Chemical Structure C9H10O2.

Also referred to as Propionic Acid Phenyl Ester. Phenylpropionate will extend the release of active steroid a few days longer than propionate. To keep blood levels even, injections are given at least twice weekly. Durabolin is the drug most commonly seen with a phenylpropionate ester (nandrolone phenylpropionate), although it is also used with testosterone in Sustanon and Omnadren.

Isocarpoate: Chemical Structure C6H12O2.

Also referred to as Isocaproic Acid; isohexanoate; 4-methylvaleric acid. Isocaproate begins to near enanthate in terms of release. The duration is still shorter, with a notable hormone level being sustained for approximately one week. This ester is used with testosterone in the blended products Sustanon and Omnadren.

Caproate: Chemical Structure C6H12O2.
Also referred to as Hexanoic acid; hexanoate; n-Caproic Acid; n-Hexoic acid; butyric acid; pentiformal acid; pentylformic acid; n-hexylic acid; 1-pentanecarboxylic acid; hexoic acid; 1-hexanoic acid; Hexylic acid; Caproic acid. This ester is identical to isocaproate in terms of atom count and weight, but is laid out slightly different (Isocaproate has a split configuration, difficult to explain here but easy to see on paper). Release duration would be very similar to isocaproate (levels sustained for approximately one week), perhaps coming slightly closer to enanthate due to its straight chain. Caproate is the slowest releasing ester used in Omnadren, which is why most athletes notice more water retention with this compound.

Enanthate: Chemical Structure C7H14O2.

Also referred to as heptanoic acid; enanthylic acid; heptylic acid; heptoic acid; Oenanthylic acid; Oenanthic acid. Enanthate is one of the most prominent esters used in steroid manufacture (most commonly seen with testosterone but is also used in other compounds like Primobolan Depot). Enanthate will release a steady (yet fluctuating as all esters are) level of hormone for approximately 10-14 days. Although in medicine enanthate compounds are often injected on a bi-weekly or monthly basis, athletes will inject at least weekly to help maintain a uniform blood level.

Cypionate: Chemical Structure C8H14O2.

Also referred to as Cyclopentylpropionic acid, cyclopentylpropionate. Cypionate is a very popular ester here in the U.S., although it is scarcely found outside this region. Its release duration is almost identical to enanthate (10-14 days), and the two are likewise thought to be interchangeable in U.S. medicine. Athletes commonly hold the belief than cypionate is more powerful than enanthate, although realistically there is little difference between the two. The enanthate ester is in fact slightly smaller than cypionate, and it therefore releases a small (perhaps a few milligrams) amount of steroid more in comparison.

Decanoate: Chemical Structure C10H20O2.

Also referred to as decanoic acid; capric acid; caprinic acid; decylic acid, Nonanecarboxylic acid. The Decanoate ester is most commonly used with the hormone nandrolone (as in Deca-Durabolin) and is found in virtually all corners of the world. Testosterone decanoate is also the longest acting constituent in Sustanon, greatly extending its release duration. The release time with Decanoate compounds is listed to be as long as one month, although most recently we are finding that levels seem to drop significantly after two weeks. To keep blood levels more uniform, athletes (as they have always known to do) will follow a weekly injection schedule.

Undecylenate: Chemical Structure C11H20O2.

Also referred to as Undecylenic acid; Hendecenoic acid; Undecenoic acid. This ester is very similar to decanoate, containing only one carbon atom more. Its release duration is likewise very similar (approximately 2-3 weeks), perhaps extending a day or so past that seen with decanoate. Undecylenate seems to be exclusive to the veterinary preparation Equipoise (boldenone undecylenate), although there is no reason it would not work well in human-use preparations.
Undecanoate: Chemical Structure C11H22O2.

Also referred to as Undecanoic Acid; 1-Decanecarboxylic acid; Hendecanoic acid; Undecylic acid. Undecanoate is not a commonly found ester, and only appears to be used in the nandrolone preparation Dynabolan, and oral testosterone undecanoate (Andriol). Since this ester is chemically very similar to undecylenate (it is only 2 hydrogen atoms larger), it has a similar release duration (approximately 2-3 weeks). Although this ester is used in the oral preparation Andriol, there is no reason to believe it carries any properties unique of other esters. Andriol in fact works very poorly at delivering testosterone, bolstering the idea that oral administration is not the idea use of esterified androgens.

Laurate: Chemical structure C12H24O2.

Also referred to as Dodecanoic acid, laurostearic acid, duodecyclic acid, 1-undecanecarboxylic acid, and dodecoic acid. Laurate is the longest releasing ester used in commercial steroid production, although longer acting esters do exist. Its release duration would be closer to one month than the other esters listed above, although realistically we are probably to expect a notable drop in hormone level after the third week. Laurate is exclusively found in the veterinary nandrolone preparation Laurabolin, perhaps seen as slightly advantageous over a decanoate ester due to a less frequent injection schedule. Again athletes will most commonly inject this drug weekly, no doubt in part due to its low strength (25mg/ml or 50mg/ml).

Title: **Re: AAS Studies/Links/Literature**
Post by: **mastry0da** on **July 13, 2004, 08:09:09 AM**

**Why Sustanon needs to be injected EOD:**

I originally posted this in response to IFBBwannaB's post that sust only needs to be injected once a week so here goes:

"i think you need to read about the specific esters in sustanon, its made up of 4 esters, the phenypropionate, the propionate, the isocaproate, and the decanoate. So essentially we have 1 short, 2 medium, and 1 really long acting ester. Although at first this whole "time release" theory sounds like a really good idea, and it probably is to a kid who unable to go through puberty and required medical test shots, but to a body builder its really not, because stable test levels are NEVER achieved unless its injected EOD, and i'm going to explain why to you. Lets just assume that all the esters in sust are the same dosage and (which they're not, but for simplicity's sake in explaining this, lets just assume they are, because it does not affect the outcome), and they are each released on day 1, 2, 4, and 6. With the first one, an even amount of testosterone is released on each day. With the second one the entire first ester, half the second ester and 1/3rd of the last ester is released within the first two days. So now, with a 250mg injection, this means that we have 114.58mg of test released on the first 2 days, then the next two get 57.29mg, and then the last 2 get 37.29mg. This means that if you inject only a few times a week, your test levels will peak sooner (giving you
immediately noticeable results which is why people think they are getting results by only injecting 1x or 2x a week), and then leave you with less than optimal levels of test later in the week for muscle building. However by injecting EOD, you keep the short acting esters within their optimum efficiency, and allow for the long acting esters to build up into a usable dosage of test, so that you never have to go into "androgen deficiency". This is the only way to keep test levels stable on sust, or else they will wreak hormone hell on your system."

---

Title: Re: AAS Studies/Links/Literature
Post by: Little_O on August 28, 2004, 09:53:48 AM

As you all know, gear has sides. It’s just something we have to live with. One of those sides (for androgens) is a spike in blood pressure.

After running test at 600 mg/week, and Deca at 400mg/week at the direction of a physician, BP began to rise around the month and a half mark. The physician prescribed "cozaar" which was supposed to lower the BP. Later I found that some MD’s use this drug for high cholesterol. I will be doing more research on this drug.

The cozaar did not help. Blood pressure was still up and the only thing the medication did was make horrible dizzy spells for the subject.

By month number two noticable water retention had set in, even after 20 mg/day of Tamoxifen to combat estrogen levels. One day, the subject took a diuretic(demedex) to pull some of the water off. At a quarter pill of demedex, he peed all night and lost about seven pounds of water.

He also felt different, he did not feel "tight" in his chest and could sleep better at night.

The next day he had his BP checked. It had gone from 170/92 to 141/85. A significant drop.

Just a theory, but he believes BP is largely due to the water retention that occurs from aromatization. This may be a "no shit sherlock" finding but then why would his MD give him BP meds and not a diuretic?

Either way, the BP was only combatted with a diuretic, and the BP meds did not help whatsoever...

Either the doc does not have the grasp on gear like the patient thinks he does, or once again everyone is simply undereducated when it comes to gear./O

---

Title: Re: AAS Studies/Links/Literature
Post by: Arnold jr on January 16, 2006, 08:35:24 PM

I thought it would be interesting and helpful to post a thread that only deals with and explains Sustanon. There seems to be a lot of disagreement on this board as to if Sust is a "quality" compound, and I believe these ideals are formed more out of lack of information, rather than coherent fact. For some of you there is absolutely nothing new here, but for those less familiar this might
help. For those who are well informed concerning Sust, feel free to add anything I might have overlooked.

Sustanon is a very good choice because it offers a number of advantages when compared to other Testosterone products. Sust is made up of 4 different testosterone's based on a timed composition that are mixed to form a synergetic effect. This allows Sust to have 2 positive characteristics: First, based on the combination effect of the compounds, Sust, mg for mg, has a better effect than Testosterone enanthate, cypionate, or propionate alone. Second, based on the mix of the 4 testosterone’s (Testosterone propionate 30mg, Testosterone phenylpropionate 60 mg, Testosterone isocaproate 60 mg, Testosterone decanoate 100 mg) it is time released so that it becomes active quickly and remains active for several weeks. It becomes active so quick because of the propionate and remains active for an extended, 3-4 weeks, because of the mix of the longer acting esters such as decanoate.

Those who use Sust can expect a solid muscle growth, and they can expect to have considerably less water retention than if they used testosterone enanthate or cypionate. Those who are uncomfortable with large amounts of water retention and fight against elevated estrogen levels, choose Sust over the long-acting depot testosterone’s.

Another advantage of Sust, is that those who have taken it before, can usually experience similar gains with the same dosage as the previous cycle. Dosage usually ranges anywhere form 250mg/wk up to 1000mg+ per day. But for the responsible user the average intake is 250mg/wk-1000mg/wk; a dosage of 500mg/wk is normally enough for most. Many argue as to how often Sust should be administered; some advocate frequent injections, 3-4x per wk, while others claim 1wk- once every 10 days. Those who advocate less frequent injections believe that since Sust is partly composed of long-acting esters, the more frequent administration is unnecessary. But those, like myself, who advocate more frequent injections understand that in order to keep test levels even, it is important to inject frequently; this is in part due to the propionate that is mixed in the compound

Although Sust does not aromatize excessively, when taken in a reasonable dosage many people, in addition, also take an anti-estrogen, to prevent possible estrogen linked side effects. Since Sust suppresses the endogenous testosterone production the intake of HCG and or Clomid should be considered. Normally, the intake of HCG during the cycle is sufficient, although it is not uncommon for it to be taken at the end as part of PCT.

The side effects of Sust are similar to those of Testosterone enanthate, but they are usually less frequent and less severe. Depending on the dosage and predisposition to side effects, the user could experience: acne, aggressiveness, sexual over stimulation, oily skin, accelerated hair loss, as well as reduced production of the body’s own hormones, which occurs with the intake of every type of testosterone. Gynecomastia is usually not that common with Sust, but if it does occur, it is not as massive as with enanthate or cypionate. Liver damage is unlikely with Sust, but in very high dosages, elevated liver values can occur which, after discontinuing use, usually go back to normal. The fact is that the liver is a very efficient organ and able to cope well with higher quantities of testosterone. "The liver is able to metabolize an almost unlimited amount of
The many faces (names) of Tren: ENDING THE CONFUSION

I have been reading about Tren and have found widespread confusion about it mainly because when one writes "Tren," s/he could be referring to at least 5 different compounds:

1. Trenbolone Acetate--injectable version (Finaject and Finajet)
2. Trenbolone Acetate--pellet form (Finaplix)
3. Trenbolone Cyclohexylmethylcarbonate (called Parabolan by Bill Roberts)
4. Trenbolone Hexahydrobenzylcarbonate (called Parabolan on this board and several others)
5. Trenbolone Enanthate

The many names of tren have confused many people. I’ve seen VETs and MODs and MEMBERs of several boards (not just this one) equate one tren compound with another, not knowing that one is actually not equivalent to the other. One common mistake I see is calling Trenbolone Hexahydrobenzylcarbonate "Tren Enanthate." They are similar, but not the same (see below). I also have seen people refer to Tren Acetate as Parabolan--wrong!

So, let’s clear this up.

The following is a brief summary of the main differences of each that I have created in order to clear up my own confusion on tren and hopefully help others here in the process. It is not meant to provide a detailed description of Tren activity in the body.

1. Trenbolone Acetate--injectable version (Finaject and Finajet)
   This is correctly referred to as "Fina." Finaject is the acetate form of trenbolone. It was produced in a short acting ester (acetate), so its effect lasts only a short time and frequent administration is necessary. Finaject was an injectable steroid of veterinary medicine, which was extremely popular in bodybuilding and powerlifting during the 1980's. The injectible Trenbolone Acetate called Finaject is no longer produced.

(Refer to the end of this post for a discussion of Esters)

2. Trenbolone Acetate--pellet form (Finaplix)
   Finaplix was a veterinary cattle implant, which contained the potent androgenic steroid Trenbolone Acetate. Once Finaject and Finajet were guy manufactured, bodybuilders began using Finaplix to make topical or injectible versions of Trenbolone Acetate.
Today, cattle implants have become designer products with varied doses and combinations of estrogenic and/or androgenic (trenbolone) agents. So, the process of converting cattle implants to useful versions of trenbolone acetate has become more difficult since one must separate the trenbolone from the other additives present in the cattle implants before using it.

3. Trenbolone Cyclohexylmethylcarbonate (called Parabolan by Bill Roberts)
Parabolan contains a much different ester than Finaject and Finajet, called Trenbolone Cyclohexylmethylcarbonate. This ester extends the activity of trenbolone for more than two weeks, a more suitable design for human use.

The amount of trenbolone in 76 mg of Trenbolone Cyclohexylmethylcarbonate is equivalent to the amount of trenbolone in only 58 mg of Trenbolone Acetate. The acetate is a little more potent, more effective per milligram, because the acetate ester is lighter than the cyclohexylmethylcarbonate ester; therefore a higher percentage of the weight of Trenbolone Acetate is trenbolone. A similar comparison also can be made with the other long lasting esters of trenbolone: enanthate and hexahydrobenzylcarbonate.

The muscle building properties of Trenbolone Cyclohexylmethylcarbonate are the same as Trenbolone Acetate (Finaject or Finajet) except for the longer half-life.

Although it is very similar, this compound is NOT the same as Trenbolone Enanthate. The only difference in these compounds is the esters (see ester definitions below), which all act almost identically (long lasting esters).

4. Trenbolone Hexahydrobenzylcarbonate (called Parabolan on this board and some others)
NOTE: At the time of this post this compound name was spelled wrong (hexahydrobenylcarbonate) in the steroid profiles. The correct spelling is listed above.
Trenbolone Hexahydrobenzylcarbonate and Trenbolone Cyclohexylmethylcarbonate are exactly the same substances. Hexahydrobenzylcarbonate ester is just another name for cyclohexylmethylcarbonate ester.

5. Trenbolone Enanthate
Although it is very similar, this compound is NOT the same as Trenbolone Cyclohexylmethylcarbonate (Trenbolone Hexahydrobenzylcarbonate). The only difference in these compounds is the esters (see ester definitions below).

THE DIFFERENCE BETWEEN THE ESTERS

The most important difference between the esters is whether it is a short acting ester or a long lasting ester. The next most important difference is the weight of the ester. As mentioned under the Trenbolone Cyclohexylmethylcarbonate section (above), the relative potency of each ester of trenbolone is partially dependent on the weight of its ester.

The main difference between different esters is simply the number of carbon atoms in the ester. Propionate has three carbons, acetate has two, isobutyrate
has four, enanthate has seven, cypionate has eight, and decanoate has ten. More unusual esters, such as cyclohexylmethylcarbonate (used in Parabolan) has eight carbons and one more oxygen than the above esters making it the heaviest.

Therefore, the esters of trenbolone in order of potency when compared miligram to miligram (from most potent to least):
1. Tren Acetate
2. Tren Enanthate
3. Tren Cyclohexylmethylcarbonate (Tren Hexahydrobenzylcarbonate)

The differences in potency caused by the esters are negligible. So, you should base your choice of Tren on how frequently you plan to inject, how much you trust your supplier, and how much you trust the brand of tren you purchase.

If you are concerned about the possible side effects of tren, and don't mind frequent injections, then consider using Trenbolone Acetate. If bad side effects manifest, Tren Acetate will quickly leave your body after the last injection due to the short acting ester (acetate); and your body will be able to begin to recover quickly. On the contrary, your recovery from bad side effects won’t begin until 2 weeks after the last injection of a long lasting ester of tren because a long lasting ester of tren will stay active in your body for more than two weeks after your last injection--continuing to contribute to the bad side effects.

Title: Re: AAS Studies/Links/Literature
Post by: Ron on March 09, 2006, 06:17:18 PM

William Llewellyn wrote this article... thanks!

Title: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Arnold jr on September 04, 2006, 12:55:28 PM

Thinking about doing steroids?? READ THIS!! Newbie Info.
Newbie Information:
Everything a Newbie needs to know before starting a cycle

Written By: Crankin'stein From Fitnessgeared.com

HOW DO I KNOW WHEN I AM READY FOR MY FIRST CYCLE?

The general rule passed on to people is that you should reach your natural potential before starting a cycle of steroids. Obviously if everyone followed this rule, anyone who used would be almost 30 and older...... So, because everyone is not going to follow that rule, here is a rule that I think is a must! You should know how to train, diet, and rest properly to make the changes to your body that you want to obtain. Whether you want to lose fat or gain mass, you should have a good understanding of how to do so without the help of steroids, before you choose to use steroids. The reason for this is because steroids are a "helper" they are not a miracle, that can transform your body well you sit on the couch. So you need to have the understanding of all the major aspects of bodybuilding, so that you can use steroids effectively.
OK, I KNOW HOW TO TRAIN, EAT AND REST. WHAT’S NEXT?

The next thing you should do is to start researching what you are going to be putting in your body, and what effects and side effects it will have. There is a plethora of information on the internet, so do some searches or research on some bodybuilding boards. Read about different substances and what kind of effect they have. Read about the side effects of them, and what to do about unwanted side effects. Read about how to cycle them, length of cycles, and Post Cycle Therapy (PCT). Also, read about Anti-estrogen’s, as these are the drugs that will save you from gyno, and too much water retention. And after you have read about all of these things, then read them all over again to make sure you have absorbed what you read. When you are knowledgeable to answer the questions you had about steroids before you did your research, then you are probably ready for your first cycle.

THE FIRST CYCLE (AKA THE NEWBIE CYCLE)

*NOTE: You should have everything including post cycle therapy stuff, BEFORE you start your cycle!!

Your first step:

The first thing you will have to do is get over that fear of needles..... A cycle including nothing but oral steroids is not going to produce the results you want, period! Go to: www.spotinjections.com to research how to inject.

The Cycle - Anabolics

The most common Newbie Cycle is Testosterone Enthanate, run at 500mg per week for 10 weeks,split up into two shots per week (250mg on day 1, and on day 4). The cycle can be run with just Test. and good results should be seen. I personally like D-bol to be added with that cycle, but it doesn't need to be. If it is added, it is run at 30mg a day for 4 weeks. The dosage should be split up during the course of the day, to keep blood levels as even as possible.

The Cycle - Anti-estrogen’s

An anti-estrogen (usually Nolvadex or Arimidex) will need to be purchased to have handy in case gyno symptoms start. Itchy and sore nipples will tell you that you are getting gyno, and Nolvadex should be started (I use Nolva personally so I am showing Nolva dosages) at 60mg per day for 2 days, then 40 for one day, then 20mg for the rest of the cycle. There is one catch tho.... Nolvadex and Arimidex will only take care of estrogen driven Gyno...... Gyno that is caused by prolactin needs to be countered by using proviron. Prolactin induced gyno can occur from Trenbolone or Deca. (these are not used in your cycle so you don’t have to worry about it...)

POST CYCLE THERAPY (PCT)

PCT can be run a few different ways..... I will outline on of the most common ways that it is run. If you don’t want to do it this way, then it is easy enough to find an alternative way, by doing research.

2 weeks after your last shot of Test. Enth. you should be starting your PCT. The
The most common combo is to use clomid and nolvadex together. Clomid is run at 300mg day 1, 100mg day 2-14, and 50mg day 15-30. Nolva should be run with it at 20mg for the whole 30 days you are on clomid.

During PCT you should keep your caloric intake at or above the amount you would use for bulking. This will help you keep your gains, and stop you from going catabolic. Also you should train hard, so that you can keep your gains. Remember that you are not on any anabolics now, so you may have to bring your volume down, and up your rest a little, so that you don't overtrain.

TIME OFF

Well now you have completed your PCT and your hormones should be getting back to normal. The general rule for time off is: TIME ON = TIME OFF. This should be followed. If you did a three month cycle, then you should wait three months after that last shot of test before doing another cycle. This will allow your body to get back to normal and stay healthy.

---

**Post by: txhulk on September 04, 2006, 11:36:09 PM**

Great post, how much arimidex would you use? I've never used it.

---

**Post by: ZEEK on September 05, 2006, 06:00:15 AM**

nice post very useful

---

**Post by: Rimbaud on September 05, 2006, 09:19:04 AM**

Nice post... how about we make it a sicky?

---

**Post by: Migs on September 05, 2006, 09:25:10 AM**

it would be a good sticky

---

**Post by: Arnold jr on September 05, 2006, 09:34:29 AM**

Quote from: txhulk on September 04, 2006, 11:36:09 PM

Great post, how much arimidex would you use? I've never used it.

For the above cycle around .25mg eod possably .5mg eod if needed.

---

**Post by: MUSTGETBIG on September 14, 2006, 06:41:48 AM**
And to think I was afraid of needles... ha ha ha ha ha ha ha :D I’ve had more pain coming from a mosquito bite!

Seriously though... I really wish I new about this shit back in high school... when I could’ve really used it! DAMMIT!!! Oh well, better late then never. I’m hoping that by my next b-day (4 months away) I will be a completely different beast altogether. And all the hot young girls will be like :o

Cartman will conquer and destroy! >:(

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Abraham000 on October 03, 2006, 11:56:26 PM

Does every bodybuilder who wants great detail have to take steroids or do they take supplements only?

and

Are supplements the same things as steroids or are they different?

Title: Re: AAS Studies/Links/Literature
Post by: Princess L on October 04, 2006, 08:18:45 PM

Not sure where I got this, so don’t know who to credit.

Might make a good sticky.

What is Clenbuterol?

Clenbuterol is a beta-2 agonist and is used in many countries as a bronchodilator for the treatment of asthma. Because of its long half life, clenbuterol is not FDA approved for medical use. It is a central nervous system stimulant and acts like adrenaline. It shares many of the same side effects as other CNS stimulants like ephedrine. Contrary to popular belief, Clenbuterol has a half life of 35 hours and not 48 hours.

Dosing and Cycling

Clenbuterol comes in 20mcg tablets, although it is also available in syrup, pump and injectable form. It’s also available as a powder in some areas. Doses are very dependent on how well the user responds to the side effects, but somewhere in the range of 4-8 tablets per day for men and 2-4 tablets a day for women is most common. Clenbuterol loses its thermogenic effects after around 8 weeks when body temperature drops back to normal. Its anabolic/anti-catabolic properties fade away at around the 18 day mark. Taking the long half life into consideration, the most effective way of cycling clen is 2 weeks on/ 2 weeks off for no more than 12 weeks. Ephedrine or Yohimbine can be used in the off weeks.
Clenbuterol vs Ephedrine

Ephedrine will raise metabolic levels by about 2-3 percent. Clenbuterol raises metabolic levels about 10 percent and it can raise body temperature several degrees.

As far as side effects, there is no ECA-style crash on Clenbuterol and many users find it easier on the prostate and sex drive. This may in part be due to the fact that Clen is generally used for only 2 weeks at a time.

Side effects

- Nausea
- Nervousness
- Dizziness
- Drowsiness
- Dry mouth
- Facial flushing
- Headache
- Heartburn
- Increased blood pressure
- Increased sweating
- Insomnia
- Lightheadedness
- Muscle cramps
- Tremors
- Vomiting
- Chest pain

The most significant side effects are muscle cramps, nervousness, headaches, and increased blood pressure.

Muscle cramps can be avoided by drinking 1.5-2 gallons of water and consuming bananas and oranges or supplementing with potassium tablets at 200-400mg a day taken before bed on an empty stomach. Taurine at 3-5grams is a necessity in minimizing cramps.

Headaches can easily be avoided with Tylenol Extra Strength taking at the first signs of a headache.

Common Uses

**Post-Cycle Therapy:** Clen is used post cycle to aid in recovery. It allows the user to continue eating large amounts of food, without worrying about adding body fat. It also helps the user maintain more of his strength as well as his intensity in the gym. Diet: Roughly the same as on cycle.

**Fat loss:** The most popular use for Clen, it also increases muscle hardness, vascularity, strength and size on a caloric deficit. For the most significant fat loss, Clen can be stacked with T3. Diet: A high protein (1.5g per lb of bodyweight), moderate carb (0.5g to 1g per lb of bodyweight), low fat diet
(0.25g per lb of bodyweight) seems to work best with Clen.

**Alternative to Steroids:** Clenbuterol has mild steroid-like properties and can be used by non-AS using bodybuilder to increase LBM as well as strength and muscle hardness. Diet: A *moderate carb, high protein, moderate fat diet work well.*

**Stimulant/Performance Enhancement:** It can be used as a stimulant, but an ECA stack may be a better choice because of its much shorter half-life. Diet: To *take full advantage of the stimulatory effects of Clen, carbohydrates must be included in the diet. Ketogenic diets do not work well in this case.*

**Precautions:**

The same precautions that apply to Ephedrine must be applied to Clen, although some people find ECA stacks are harsher than Clen. It *should not be stacked with other CNS stimulants such as Ephedrine and Yohimbine.* These combinations are unnecessary and potentially dangerous. Caffeine can be used in moderation before a workout for an extra quick burst of energy.

**A word on Ketotifen**

Ketotifen is a safe antihistamine used extensively in some European countries to treat asthma and allergies. It can up regulate beta-2-receptors that Clen down regulates. Basically, it allows users to extend their use of Clen for 6-8 weeks at a time. 2-3mg a day is ideal, 10mg as found in "superclen" can make users extremely drowsy. It also increases the effectiveness of Clen so doses must be adjusted accordingly. The downfall of this drug is its ability to induce extreme hunger in some people, which is not a desirable state to be in when dieting.

**Cycling Clenbuterol**

Most users that report bad side effects and discontinue use are those who use high doses right at the start of the cycle. The worst side effects occur within the first 3-4 days of use.

A first time user should not exceed 40mcg the first day. Increase by one tab until the side effects are not tolerable.

**Example of a first cycle:**

Day1: 20mcg  
Day2: 40mcg  
Day3: 60mcg  
Day4: 80mcg  
Day5: 80mcg (Note: Increase the dose only when the side effects are tolerable)  
Day6-Day12: 100mcg  
Day13: 80 mcg (Tapering is not necessary, but it helps some users get back to normal gradually)  
Day14: 60mcg
Example of a second cycle:

Day1: 60mcg
Day2: 80mcg
Day3: 80mcg
Day4: 100mcg
Day5: 100mcg
Day6-Day12: 120mcg
Day13: 100 mcg
Day14: 80 mcgs
Day15: off
Day16: off
Day 17: ECA stack

What else do I need to know?

**Taurine MUST be used with Clen at 3-5g daily. Clenbuterol depletes taurine levels in the liver which stops the conversion of T4 to T3 in the liver. Taurine allows the user to avoid the dreaded rebound effect and painful muscle cramps. It's a must with Clen.**

Clenbuterol should not be taken too close to a workout. It can interfere with your breathing and complete ruin your workout. When doing cardio, it's advisable to stay at a consistent pace and avoid HIIT style routines.

Do not take Clen Past 4pm and drink plenty of water; 1.5-2 gallons a day.

---

**Title:** Re: AAS Studies/Links/Literature  
**Post by:** EgoKiller on October 05, 2006, 06:19:20 AM

So you've decided to use T3 to help you shed fat now that you've read up on it and gotten past the nay Sayers who expound the ills of shutting down your body’s own production of natural thyroid. Wonderful, T3 when used correctly can be a great addition to any diet and cardio plan. Read that again boys and girls, IN ADDITION TO ANY DIET AND CARDIO PLAN!!! If you've turned to T3 because you think it's a magic pill that will allow you to eat like crap and still lose weight you've been listening to the wrong advice. Can you lose weight/fat while using T3 and still eating junk food, unfortunately yes to a degree. I say unfortunately because this fact often leads people to do just that, it starts with a cheat meal that turns to a cheat day, which eventually has the athlete eating whatever and whenever they want and still they lose some weight. So what's wrong with this if the eventual out come, weight loss that is, is reached? The first problem is the weight you are losing may not be fat if your eating like crap, the second is what happens when you stop the T3 cycle and your metabolism is suppressed temporarily, if you were eating sloppy during the T3 usage your most likely to keep following that pattern and the combination of a slow metabolism combined with sloppy eating results in rebound weight gain. So in
the end what have you really accomplished outside of being able to eat what you want with out getting any fatter for a month or so? And that's if you're lucky and the rebound weight gain doesn't push you past your starting weight!!!

Now that I have your attention and you know what NOT to do, let's concentrate on what TO do. Just like any other chemical we find in our arsenal, T3 can and is used in a variety of ways when it comes to dosage and length of cycle, both for cutting and bulking. This article will deal with cutting use only. There are some who prefer to "hit it hard" and go high dosage with a quick taper down at the end losing a great amount of weight in a short time, but this way tends to eat as much muscle as fat in my experience and you end up looking basically the same as when you began, except that you weigh less and are smaller. There are those who like to use the same dosage throughout the cycle with no taper up or down figuring if your metabolism is going to be sluggish anyhow why waste the days using it at a low dosage when you could be burning more fat on those days. Then there are those who slowly taper up, maintain the highest dosage for a set time and then slowly taper down. It's the last group I'll concentrate on here, as this is the system that has shown it's best overall results with those I've worked with.

Let's start with the dosage, T3 is a very individual drug, when it comes to dosage I've seen guys use as high as 250-300mcg/day and others as low as 25mcg/day where both athletes lost fat and reached their goals. As a rule I start everyone (and for now I'm dealing with men I'll pen an article on women's usage in the future) at 25mcg/day. I usually base the time of the cycle on their individual weight loss goals, if it's a smaller amount I'll go 3 weeks tops, if it's a lot of weight to lose we'll go 4, 5 and sometimes 6 weeks. I generally don't go over 6 weeks with anyone, as T3 tends to stop working in most people after that amount of time. I'd rather they run 4 weeks cycles with 2 weeks off where they use an ECA stack or Clen during the break to continue to lose fat, then run another 4 week cycle. So the 1st 3 days in this cycle would be 25mcg/day, then the 2nd 3-day period is 50mcg/day, etc. The typical 21-day cycle will look like this:

| Days 1-3 | 25mcg/day |
| Days 4-6 | 50mcg/day |
| Days 7-9 | 75mcg/day |
| Days 10-12 | 100mcg/day |
| Days 13-15 | 75mcg/day |
| Days 16-18 | 50mcg/day |
| Days 19-21 | 25mcg/day |

As you can see the dosage is increased by 25mcg/day every 4th day until the maximum dosage is reached for the subject, in this case 100mcg/day, then lowered the by the same 25mcg/day increments every 4th day until the end of the cycle. Given that most of the people I've worked with have tried everything else and are still considerably overweight when they start, the full 4-week cycle is often used instead of the 21-day cycle. The one I've used lately with the most success is as follow, remember the jumps are still 25mcg/day but this time you increase/decrease the dosage every 4 days:

| Days 1-4 | 25mcg/day |
Days 5-8...................50 "
Days 9-12...................75 "
Days 13-16.................100 "
Days 17-20.................75 "
Days 21-24...............50 "
Days 25-28............25 "

Note: You could also do the 3-day increase/decrease and hold the maximum dosage of 100mcg/day for days 10-19, but some find 100mcg/day makes them too uncomfortably warm and they sweat too much, especially during the warmer months.

There you have it, simple yet effective. If you remember to supplement your diet with plenty of protein (which every lifter should anyhow), eat a clean calorie controlled diet, drink 1-2 gallons of water per day and to take a mild steroid cycle to minimize muscle loss you should be able to see rapid fat loss with this cycle. I should also mention that some people like to stack T3 with Clenbuterol for even better results. I’ve purposely left this out as I will be including Clen in Part 2 of this series. Good luck and may you all reach your cutting goals!!!

Title: Re: AAS Studies/Links/Literature
Post by: EgoKiller on October 05, 2006, 06:20:18 AM

In part 1 of T3 and the Modern Athlete I touched on various properties of T3 and mainly its use by male athletes for cutting body fat. In Part 2 I’d like to dig deeper and discuss a few other areas of T3 usage, among them the popular T3/Clen cycle, T3 in bulking cycles, rebound weight gain and women’s cycles. Response to Part 1 was overwhelming to say the least and I realize Part 2 is way over due, I hope it was worth the wait and answers the questions you still have on T3. As with Part 1 please realize that this article is the opinion of the author based on personal experience and the experiences of friends, ex training partners, gym mates and clients it is not intended to be a recommendation to use T3 nor is it intended to be a medical “how to”, ultimately how you use it and whether or not you use it for athletic performance gains is your choice and yours alone. With that said let’s dig in!!

1. Thyroid Suppression
2. T3/Clenbuterol Cycle
3. T3 in bulking cycles
4. Women’s Cycle
5. Timing of dose
6. Rebound Weight Gain

Thyroid Suppression
Let’s start with the biggest misconception still around where T3 is concerned, that is suppression of natural thyroid output. I’m amazed that this drug has been used now for the past several years by literally thousands of athletes with few if any reported cases of thyroid shutdown yet the 1st thing someone says when a person asks about T3 is “it will shut down your natural thyroid and you’ll be on T3 the rest of your life”. Numerous studies have been done and show that cessation of exogenous T3 does not shut down your natural thyroid.
The 1st study was done in 1951 by M. Greer (1) and showed that patients that were misdiagnosed as hypothyroidism that later had their medicine withdrawn showed no shutdown of their natural thyroid as their thyroid returned to normal within 2 weeks. His studies also showed that it didn’t matter if the patients thyroid had been medically suppressed for 30 years or a few days they both returned to normal within two weeks. Hence my mentioning in Part 1 of a sluggish thyroid post T3 cycle and my suggestion that you continue to eat clean, do cardio and use a fat burner like the Ephedrine/Caffeine/Aspirin stack, Clenbuterol or an over the counter fat burner until your natural thyroid output returns to normal. Numerous studies have been done since Greer’s that have confirmed his findings. As with any medicine there are always exceptions to the rule and there have been a few people who claim to have had their own thyroid function permanently damaged by T3 usage but in my experience this only occurred when ridiculously high dosages were used, if you adhere to the dosages recommended here in you should be fine.

T3/Clenbuterol Cycle
This has to be the most often used cutting combo used today for fat loss in weight trained athletes, or at least the most talked about. Both drugs when used on their own are effective fat burners through differing pathways, but used together they have a synergistic effect and create a very potent fat burning cycle. The medical reasoning for this is long and complicated and not necessary to understand at this point but it is out there for anyone to research should you need to know, in simple terms each not only do their own job but also help the other’s fat burning process so that in effect, as they say, 1+1=3. So what dose do you use for each drug? For the T3 I suggest you use the same dose scheme I outlined in Part 1, again I took some flack over the lower dosing as some feel you should go higher but as I said from my experience anything over 75mcg-100mcg/day (for men, women’s dosage should go no higher than 50mcg/day) usually burns much too much muscle tissue in addition to fat tissue, unless that is your goal I would stay with as small a dose as you can get away with where you can still tolerate the increased body temp, for most men that is 75-100mcg/day, for most women that is 50mcg/day max. *Using Clen will increase your body temp also so you will have to monitor both drug dosages to see what you can comfortably tolerate. Clenbuterol dosing is a very individual thing, some cycles recommend 160mcg/day at the maximum dosage some 80mcg/day but the 1 thing most agree on is to start low and ease your dosage upwards as you feel comfortable with it the 1st time you use it. With subsequent cycles you can start at your maximum tolerable dose or slightly lower and then increase the dosage over a few days until you reach your maximum again as some people report the maximum they can use differs from 1 cycle to another. Which brand and whether you use tabs vs. liquids could also have something to do with the differing max doses. I would suggest you start your 1st cycle of Clen with 20mcg/day and increase by 20mcg/day until you reach the upper maximum you can use based on the side effects. The most common side effects are shaking, jitteriness, anxiety and raising of body temperature, basically the feelings you get when you’ve had way too much caffeine or cold medication are what your looking out for. When those sides get to be too much cut back to the last tolerable dose. A popular Clenbuterol cycle is 2 weeks on, 2 weeks off. For men I would suggest starting at 20mcg/day and going up to 100-120mcg/day or like I said whatever you can tolerate, stay there until day 14 then end the cycle, women should try half that max dose but if you can tolerate more and want to use it then go for it this is definitely a trial and error process. Take 2 weeks off and then repeat if desired, again starting at or near your maximum dose that
you figured out with the 1st cycle. When stacking with T3 the question becomes what do you do on the 2 weeks your off Clen but still are on T3? That’s really an individual decision for you to make, you could rotate an ECA stack or a Gugglesterone with the Clen cycle so that your doing 1 for 2 weeks then the other for 2 weeks. Or you could simply take 2 weeks off after the end of the Clen where your only on the T3 for the next 2 weeks, you’ll be at your mid to max dosage of T3 by then so you’ll still be burning fat just fine. Then after the 4 weeks of T3 you’ll be done with both the T3 and the Clen and you could start a ECA stack for 2 weeks if you are ending the cutting cycle and want to protect yourself against rebound weight gain while waiting for your natural thyroid levels to return to normal. If you have more fat to lose you can cycle off T3 for 2 weeks as I said in Part 1 and repeat the cycle again. When to use Clen again will depend on when you used it last, remember 2 weeks on, 2 weeks off. There’s nothing to say you can’t cycle T3 ad ECA together while you wait to add the Clen back in, just remember whenever you come off the T3 you want something in your system to help burn fat while you wait for your natural thyroid to return to normal. Also remember that Clen cycles are like T3 cycles in that there’s several different cycle’s currently popular, and you’ll most likely get different advice to the length and type of cycle by asking more than one person. The advice I give is based on those I’ve had use it and report back to me their results and feeling on it. I’m all for experimenting but until something comes along that proves to be better I’ll stick with the 2 weeks on/2 weeks off cycle advice where Clen is concerned.

T3 in Bulking Cycles
I briefly touched on using T3 in bulking cycles and many members seemed confused as to how a fat burner could help with a bulking cycle. T3 is a drug mainly known for raising one’s metabolism and burning fat, and possibly muscle tissue, when used at higher dosages (> 75-100 mcg/day for men, > 50mcg/day for women), but at lower dosages (12.5-25mcg/day for men, ½ that for women) it causes a faster conversion of carbohydrates, proteins, and fats. It’s the increased conversion and absorption of nutrients that increases the results of your bulking cycle when you use it with a bulking cycle. When you run a bulking cycle you do so in conjunction with a higher protein/higher calorie diet because we know in order to grow muscle we need to feed the body nutrients, so there are plenty of nutrients to be converted, thus the bulking cycle gets a “push” if you will yielding better results. I can tell that literally every single person who has taken my advice and tried using a small amount of T3 daily with their bulking cycle has reported better gains than they usually get without it. I’ve even had success using 25mcg/day every other day with a bulking cycle. When you consider the low cost of T3 at such small a dosage it’s definitely a cheap insurance to better gains.

Women’s Cycles
Although women have been known to use T3 with good success I always hesitate to recommend a cycle to them for the simple reason that women seem to be much more sensitive to T3 than men are. The rebound weight gain can be significant if the post T3 period isn’t monitored stringently and an over the counter fat burner isn’t used. That said if you’re still set on using it here is a simple straightforward 21 day cycle, again using the 3 day ramp up and ramp down method.

Days 1-3.............12.5mcg/day
Days 4-6.............25mcg/day
Days 7-9..............37.5mcg/day
Days 10-12............50mcg/day
Days 13-15............37.5mcg/day
Days 16-18............25mcg/day
Days 19-21............12.5mcg/day

If you want to run it longer than 21 days, you can add in more days at the maximum dosage or use it in 4 day blocks with the ramp up and ramp down. Again please remember women are more sensitive to T3 than men and the rebound weight gain can be much more significant if your not ultra vigilant with the post T3 period, keep eating a very clean diet with calories below maintenance, and use either Gugglesterones, ECA stack or any other over the counter fat burner you feel comfortable with to help boost your natural metabolism until your system recovers, which could be anywhere from a few days to about 2-3 weeks.

Dosage Timing
T3 has a ½ life that doesn’t necessitate multiple daily dosing, so taking your entire daily dose at once is usually recommended. That said if your cycle requires you to take 100mcg/day or more I usually recommend splitting the dosage in ½ and taking it twice per day just to insure if you are sensitive to the drugs possible side effects you limit the exposure. Again I would suggest taking it in the morning, then around dinner time if a 2nd dose is necessary. I know that for myself, certain brands cause an upset stomach if I take more than 50mcg at a time, so at 75-100mcg/day I’d split it into 50mcg in the morning and the balance at nighttime.

Rebound Weight Gain
Rebound weight gain is inevitable when using T3, the best you can hope for is to minimize it. A good start is to make sure you use at least a small amount of a steroid with the cycle, this will help you to hold on to the muscle mass you already have. The best thing you can do is to take a post cycle over the counter fat loss product such as ECA stack, Gugglesterones or some other similar product. What your looking for here is the continuance of the fat loss while your system returns to your normal thyroid output. This should occur with in 2-3 weeks, so during that time continue to eat clean, do cardio, drink plenty of water and take the over the counter fat loss product. You’ll know when your thyroid has returned to normal when your body temp returns to normal. Women are especially warned to be very vigilant here, most people are eager to eat more when their cycle ends but this is not the time when using T3, you need to make sure your metabolism has been restored before splurging a bit.

I hope Part 2 answered the questions you still had on T3 after reading Part 1, if there is enough interest I’ll write Part 3 there are still some areas I haven’t covered, the synergy of T3 with GH and AAS being one of them. If you have any other areas you’d like seen covered post up on them and I’ll see if there is enough to put together a Part 3. Also try to remember the dosages I suggest here may seem low to some of you but this article is directed toward the recreational athlete not the elite athlete, elite athletes generally take more of everything and although I don’t personally think T3 needs to be one of those drugs used at higher dosages other will disagree with me there. Good luck with your T3 usage and if you have any personal questions feel free to PM or e-mail me.
i've seen a few threads regarding DNP so i thought i would post this.

the info is from Animal’s DNP manual and is a must read for anyone using or thinking about using DNP.

Why you might want to use DNP.

Add some DNP to an animals diet. DNP can get metabolism up at least 50% which is conservative as some say 75% This would mean if the animal eats 3000 calories maintenance they are now at 1500 calories a day with no change in diet! A 2500 calorie a day would leave them with 1250 calories a day. There are 4086 calories in 1lb of fat and at 3000 calories a day your DNP adjusted calories for the day is 1500. Multiply that x 7 days to give you 10500 calorie deficit which is 2.5 lbs of fat loss for the week. At the 2500 calorie you have a 2.14 lb fat loss. These are both below what the BO diets claim and you don’t have to stop eating!

If your animals weigh around 200lbs their effective dose is 400mg and the max can be as high as 800mg a day.

High fat diets market on the basis that you are going to be able to lose 1.5?2lbs of fat by just changing your diet!

1 gram of fat is 9 calories. There are 454 grams in a 1 pound. This gives you 4086 calories for 1lb of fat. If you want to lose that 1lb of fat you have to have a 4086 calorie deficit to do it. In other words, you need 4086 calories in your diet if you want to lose 1 lb of fat. Now, Let’s say you are at 3000 cal a day for maintenance. That is 21000 calories a week. You believe the marketing of the post above and think you can lose 1.5lbs of fat. That, my friends, is 6129 calories which you have to subtract from 21000 which leaves you with 14871 calories for the week or 2124 calories a day. You are going from 3000 to 2124 a day. If you want to lose that great sounding 2 pounds you are now at 12828 for the week or 1832 calories a day.

Let's be realistic and put you at 2500 maintenance calories. To lose 1.5lb you now need 11371 calories a week or 1624 calories a day or a nearly 900 calorie a day change. To lose the magical 2lb a week you need 9328 calories for the week or 1333 calories a day or a 1167 calorie change per day! That is rather difficult, but let’s add some DNP which can get you metabolism up at least 50% which would mean you are now at only 1500 calories a day for a 3000 calorie diet with no change in diet! A 2500 calorie a day would leave you with 1250 calories a day. These are both below what the BO diets claim and you don’t have to stop eating!

What you want to keep in mind

Everyone is different.
Don’t take it on an empty stomach or it will feel like you have indigestion for most of the day.

I wanted to stress not to just go balls out (5mg/kg) and you should move up gradually on DNP for your first experience.

If you have an allergic reaction with red spots and itching then stop the DNP and get some Benadryl and then you should be able to start again.

The type of diet will also affect how you feel, as well as the type of workouts you are doing. These are variables you also will have to figure out for yourself. The logic of my dieting regimen follows that while you are DNP all the glycogen/glucose is being scavenged to provide ATP for the mitochondria so you will want to eat a regular diet. High fat BO is not going to help you build muscle even though DNP is anti?proteolytic (protein sparing). Furthermore, when you eat fat it is more likely to go to fat! That is scientifically proven. So if I’m trying to burn fat, why would I want to eat it right back?

DNP is anti-proteolytic which means it uses carbohydrates or fats exclusively to supply energy for the mitochondria and does not facilitate muscle breakdown, however, this does not therefore mean DNP is positive for muscle building. The cells are running on overdrive and they are not going to be looking to make themselves bigger which requires even more energy.

Everyone is different and other supplements you take will affect your results, but as a whole, most people are not going to do well or feel well on high fat and DNP. I also have found that taking particular supplements helps with how will you feel while on the DNP.

I feel better when I don’t do huge carbs, however, when I don’t do any as in high fat type diets, my workouts suffer just the same. Each individual has to decide for themselves and put those factors into perspective with what their goals are and how fast they want to accomplish them and how bad they are willing to feel for the desired weight loss.

WARNING: DNP will turn everything and anything yellow including skin, clothes, carpet, and hair. I dropped a capsule in my DNP container and bent over to look for it and my hair touched the edge of the container and my hair got dyed yellow! My hair did not even touch the DNP, but just the side of the container for about 2 seconds! DNP for the most part is not removable or bleachable with normal chemicals. It will also track. By that I mean, you think you have washed it off your hands and you touch something and later you see yellow spots on what you touched. If you are making caps you need 2 pairs of gloves, at least, as the DNP goes through the first pair due to an attraction it has for moisture. DNP sublimes and floats. Due to this sublimation it will land on EVERYTHING if you leave it out even if there is no air circulation. DNP goes through EVERYTHING including plastic, hdpe plastic, pet plastic, plastic bags, nitrile and latex gloves. It can be washed out of clothing with hot water and detergents that have phenolic compounds in them such as Tide. DNP is not solvated by laquer thinner, acetone, paint thinner, or turpentine or any of the common organic solvents. If you wash your hands immediately after touching DNP with gamma-butyrolactone, otherwise know as GBL and use to make GHB, and then a detergent such as Dawn dishwashing soap, the stain will come out for the most part.
I have to say that a certain guru which some people keep quoting is what I feel to be a very unreliable source. I will give him credit for bringing DNP to the forefront, but I will bet you a million bucks that he has never done it or mixed it. Here is a quote that bears this out; ‘I don’t see what the worry is about everything turning yellow? I have no problems, I just dry it out and cut it with a credit card and cap it.’

That is total BULLSHIT! Anyone who has used or mixed DNP powder knows that it will get on EVERYTHING and turn it yellow. It goes through plastic bags. Just today I was sending someone 3g for research and I put it into a ziploc and 2 hours later I came back and the envelope under the bag was YELLOW! It goes through 1 layer of rubber gloves. It turns white HDPE bottles yellow. It floats everywhere. I had to put my stuff in a hood because it got on everything I had sitting out and I had to wash all my glassware and scales before I could use them again. DNP floats by sublimation which would be known just be reading the safety sheet or the Merck Index. On the basis of that statement alone I have some real problems believing anything he says on the subject, but another famous quote is, ‘DNP will raise your body temp high enough to kill you!’ This also proves that he has never done it because as you will find, your body temp only goes up about one degree. Ok, enough about the fake guru.

Someone just asked me if the shit I sent them was real. Well, if you want a test then rub it on your hands and throw some on your carpet. When your carpet has to be replaced because NOTHING can remove the yellow and you look like a total ass because your hands are bright yellow, then you can ask me if it is real!

Mostly people are taking DNP for 1 week at a time because it exhausts you and you sweat a lot, usually that is what I do, but due to my ‘work’ with DNP I got a dose while on an ECA week and that combination of DNP-ECA was like methamphetamine. In fact it was better because it had less side affects. I would venture that DNP-PPACA would also have the same methamphetamine effects. At this time I do not know, however, whether PPA works on the same receptor so I would not do them back to back in cycles. ECAY where Y is yohimbine is also a combination that has meth type benefits. Clen-DNP did not exert any magical meth benefits that I noticed. Have not taken PPACA or PPACA-DNP or PPACAY.

Tyramine and yohimbine are awesome and someone that I hold using it was getting goosebumps and asked a pharmacologist what the goosebumps were about. The pharmacologist told him that it meant he was burning a lot of calories. I love this combination and it is just like meth due to large releases of NA although it only lasts 4 hours or so.
DNP also ‘upgrades’ the effects of clen. If you have used clen before and it had/has stopped working, then DNP will bring back it’s glory.

I like to keep the clen and DNP a week apart due to the affects they have on T3 although they work on different mechanism it is just a precaution to keep from shutting down the T3. You could add Y to it for an added benefit which will not cause downgrade of anything. Reports on DNP-Y indicate a higher rise in body temperature on this combination.

Due to the systemic affects of DNP, it affects EVERY cell in the body that has
mitochondria, including smooth (digestive) and muscle and fat as well, you will not see a significant rise in body temp like you see with clen or ECA. Clen and ECA work primarily on muscle cells and that causes a rise in body temp just as if you were working out. I don’t know why this is such a difficult concept for some to understand, but I was sweating like hell recently, and I took my temp and it was 95.8. ON DNP!

DNP MECHANISMS

The basics first. DNP is a classified as a chemical poison. It's mode of action is to disrupt the ETC (electron transport chain) and cause uninhibited exchange of protons. This exchange of protons is what is responsible for making ADP into ATP. NOTHING can stop the disruption of this process once it starts. DNP works no matter what! High or low T3 has nothing to do with whether or not DNP affects the mitochondria and burns off extra energy. DNP gets into the cell and into the mitochondria and causes proton release. No other hormones are needed or noted.

Even so, it works in much the same way as clen or ECA or PPACA or thyroid. They ALL cause the metabolism to speed up. These all work via the mitochondria as well, although the non-DNP diet drugs work on the receptors first and DNP goes directly to the mitochondria, the results are the same which is speeding up the metabolism to burn fat.

Some other important facts you should know are how ephedrine and beta-3 activation drugs work. These both cause uncoupling of the ETC chain just like DNP! Ephedrin works part of its magic via beta-3’s and much research has been done looking for a magic beta-3 drug. Why, we have it and it is called DNP! If you are sitting around and something is making you hotter, you are most likely experiencing an uncoupling of the ETC chain. No big deal, but DNP just causes a greater effect. I knew there was a reason that you CANNOT die from DNP usage, at least the doses many are doing. I talked to a couple people about this but just couldn’t find the info to prove it. Ok, so what does DNP do? It uncouples the ETC or oxidative phosphorylation as was elaborate upon above, allowing electron flow to go unchecked at maximal rate and resulting in heat production and ATP depletion.

ATP depletion is the key. What condition exists when you have totally exhausted all ATP and no more is being created? A very good instance we all know about is when you are dead and it is called ‘rigor mortis’. Rigor mortis results because no more ATP is binding to the myosin head of the sarcomere in the muscle fibers.

So what does this have to do with us? No one has ever had rigor mortis on DNP or even severe cramping that has ever been documented. Furthermore, and to be more specific as to the uncoupler DNP, the electron gradient is collapsed and it runs unchecked at maximal as I have explained above, but as the gradient continues to increase electron transport becomes more difficult and the process SLOWS! Additionally, under very large artificially created electrochemical
proton gradients, normal electron flow stops and may even result in REVERSE electron transport flow!

All that was complicated and here is what it means. The respiration chain has a safety mechanism which allows for feedback controls to keep you from killing yourself. This is also another reason you will not want to do DNP for long periods. If you have taken enough as to create a large gradient the flow of electrons your burning of calories might even STOP! This will happen if you don’t eat enough calories and appears to be more detrimental on a high fat type diet because as you will see below, glucose can ameliorate charge differentials in the mitochondria and at the cell surface while on DNP.

DNP works NO MATTER WHAT! It uncouples the electron transport train (ETC) and there is nothing you can do to stop it. Some have said it doesn’t work after a small dose or only after taking DNP for 2 days or so. I think they are the same kind of person who would take a drink of beer and say, ‘Oh, I’m not drunk so alcohol doesn't work’! Alcohol still affects your brain cells and hormone levels and slows down the metabolism. Just because you didn't drink enough to be drunk doesn't mean nothing happens!

DNP is anti?proteolytic. This means DNP does not break down protein via the mechanism through which DNP works. DNP is actually better for you than cardio because exercise is PROTEOLYTIC which in itself is another reason to not be doing a high fat diet. High fat diets and exercise both lower insulin and raise glucagon levels which cause breakdown of protein. It is a proven fact that 10?20% of energy from exercise comes from AA breakdown as well as release of glutamine from the cells. DNP burns calories and does not affect hormone levels. Someone said something about it causing ketosis which is likely if you don’t eat any carbs, but DNP is not, by itself going to affect insulin levels like glucose disposal agents metformin or phenformin.

DNP is not going to be advantageous to muscle building. THIS DOES NOT DISAGREE WITH WHAT I WROTE ABOVE! It is anti?proteolytic via its mode of action, BUT if there is not enough energy in the cells to build muscle it ain’t gonna happen. Again, diet is key.

DNP is one of the SAFEST drugs you can take!!!!! Why? Am I nuts?! I am basing this on DNP’s mode of action. DNP has one purpose and mechanism and affects nothing else, but the mitochondria. DNP does not affect hormone levels as do clen, ECA, T3, etc. It has no side affects that you don’t expect such as shakes or cramping. Compare DNP to some of the Drugs the FDA has approved and look at their side effects and then tell me what is safer! HAHA!

After you read this study you need to ask yourself, need I say more? In the earlier paragraph on the mechanisms of DNP on the mitochondria I explained the safety mechanism which could keep DNP from being totally depleted of ATP. Some were saying ATP depletion would result in cell death. The study below illustrates another mechanism which I didn’t know about. The crux of it can be summarized by this sentence: ‘The failure to find a reduction in ATP concentration in either fibre type during prolonged exercise in the face of a progressive increase in the number of fibers showing little or no glycogen concentration suggests that protective mechanisms exist that prevent an energy crisis. The nature of these protective mechanisms remains to be elucidated.’ In
When glycogen is gone there is a mechanism which keeps ATP from being depleted which is unknown at present!

Energy metabolism in human slow and fast twitch fibers during prolonged cycle exercise.

Author Ball?Burnett M; Green HJ; Houston ME
Address Department of Kinesiology, University of Waterloo, Ontario, Canada.
Source J Physiol (Lond), 437():257?67 1991 Jun
Abstract

1. The effects of prolonged exercise on energy metabolism in type I and type II muscle fibers in the vastus lateralis muscle were investigated in six male subjects (20.0 +/- 0.5 years, mean +/- S.E.M.) who performed one?legged cycling at 61% of maximum O2 consumption (VO2,max; determined with one leg) until fatigue or for a maximum of 2 h. 2. Analysis of pools of freeze?dried fibers obtained by needle biopsy and separated into specific types by the myofibrillar ATPase histochemical procedure indicated higher (P less than 0.05) lactate concentrations in type II fibers compared to type I fibers at 15 min (43.9 +/- 9.7 and 51.2 +/- 9.8 mmol (kg dry wt)?1) and at 60 min (18.2 +/- 4.7 and 25.9 +/- 6.5 mmol (kg dry wt)?1). No differences existed in lactate concentration between fibre types for pre?exercise (10.0 +/- 1.6 and 13.3 +/- 2.8 mmol (kg dry wt)?1) or post?exercise. 3. Glycogen degradation was most pronounced in type I fibers. By the end of exercise, glycogen concentration was 82.4 +/- 45 mmol glucosyl units (kg dry wt)?1 in type I fibers and 175 +/- 62 mmol glucosyl units (kg dry wt)?1 in type II fibers. 4. No significant changes in ATP and creatine phosphate (CrP) were found in either fibre type with exercise. 5. It is concluded that, at least for lactate and glycogen, fibre?specific differences are evident in prolonged submaximal exercise. The cause of the difference probably relates both to the unique energy metabolic characteristics of each fibre type and to the manner in which they are utilized during the exercise. 6. The failure to find a reduction in ATP concentration in either fibre type during prolonged exercise in the face of a progressive increase in the number of fibers showing little or no glycogen concentration suggests that protective mechanisms exist that prevent an energy crisis. The nature of these protective mechanisms remains to be elucidated.

DNP will make you breathe harder via a mechanism called cellular hypermetabolism. You aren’t going to die if you are breathing hard! DNP works by increasing ventilation and oxygen consumption via hypermetabolism of the cell. DNP makes you breath hard.

Title: Re: AAS Studies/Links/Literature
Post by: jmt1 on October 15, 2006, 11:09:51 AM

How to feel good on 600mg of DNP!

The longer I took DNP the more I realized those who had originally recommended DNP use were not looking at the big picture, and they had most likely never used it or mixed it themselves, and/or were just complete morons!
Myth #1.
You die on DNP from heat related to overdose.
Wrong!
You die from dehydration resulting in heat exhaustion and then heat stroke.

Myth #2.
You can do it on high fat-low carbohydrate type diets.
NO YOU CAN'T!
High fat-low carbohydrate diets are based on keeping your blood sugar and
insulin low. DNP will also drive down your blood sugar, so if you want to have
blurry vision due to low blood sugar and feel like hell, you go right ahead.
Glucose also has some beneficial cellular effects when used with DNP.

Myth #3. You will go blind.
Right! If you do high fat-low carbohydrate diets and don’t keep your blood
sugar up and/or don’t take pyruvate.

Myth #4.
You can’t work out on DNP.
Yes you can, if you know what you are doing and which I am about to tell you.

As you may already know you, should be taking the following per day.

1200-1500mg magnesium in 2-3 divided doses.
2-3000mg vitamin C.
1200IU of vitamin E
200mcg of selenium.
1000-2000mg of calcium (can’t take it with the magnesium, though. Take it
before bed)
Melatonin if you can’t sleep and it is also one of the best and cheapest anti-
oxidants.
50mg of zinc a day
one iron tab as hemoglobin is a protein as well.
A potassium gluconate tab or two a day
Taurine at 3g a day.
Glutamine at 15g a day sublingual or with carb/protein drink.

I think taurine will be most beneficial for cramping and holding onto water. I
have worked with some mountain bikers that were having trouble with cramps
and had tried using the proverbial potassium supplementation cure and it
didn’t work. I had them take the taurine and magnesium and the cramping went
away. Taurine is also give to people who have leg cramps at night at a dosage of
3-6g a day resulting in total alleviation of the cramps. Clenbuterol depletes the
liver of its taurine supply which changes the osmotic pressure and therefore
stops T4-T3 conversion. Taking supplemental taurine can alleviate this.

Glutamine also regulates water, but is a bitch to take and unless you want your
small intestine to absorb most of it you have to take it sublingual. You can fool
the body a bit by putting it into your carb/protein drink after you work out or by
taking 2g doses throughout the day. Glutamine ALSO causes a rise in insulin.

IF you are on clenbuterol, pyruvate and glycerol will help you a little, and I
don’t know why, but I still got some cramping on clen after an event even on P
and G. The latest research I have indicates that the reason clen may cause
cramping is due to TAURINE depletion so by taking the 3g a day taurine you should be able to ameliorate those effects as well and keep your thyroid levels normal as well.

In addition to the vitamins and minerals you should be taking:

- 3-6g Pyruvate (P)
- 3 tablespoons Glycerol (G)

If you can’t get the G and P go right to the taurine which may be cheaper as well. Glycerin (glycerol) is avail in the skin care section of your pharmacy and 4oz is about $1.2 dollars or there are larger versions in white bottles and the brand name is H something. Just buy it from a vet and a gallon is around $20!

I felt like shit when I went above 400mg and sweat profusely on single large doses of 600mg and 800mg which lasted for 2 days. I weigh around 95kg (210).

The object of the DNP dosing with the glycerol and pyruvate was to test their benefits on what is considered an overdose of DNP while maintaining my exercise level in the middle of summer.

Here is what I was taking:

- 600mg of DNP which would be 6mg/kg which is well above the recommended 3-5mg.

DNP is said to have a half life of 36 hours and this is what I have based the following dosing scheme. I also have anecdotal evidence that DNP can last 48 hours or more. When I took an 800mg dose after 3 days on 300mg a day I sweat for 48 hours straight and that 800mg was the last dose I took.

You have to divide the 600mg into two doses of 300mg 12 hours apart. After you hit your 600mg limit you don’t take the next 300mg for 36 HOURS from the first dose!

So, if I took the first dose at 6AM on Sunday morning and the second 300mg dose at 6PM Sunday night, the 3rd dose would not be until 6PM on Monday evening. The fourth dose would again follow 12 hours from the 3rd which would be at 6AM on Tuesday morning.

I am also taking EC with this just for fun although at only 2 x a day for the EC to keep energy levels up and lessen the carb craving that goes with DNP.

How am I not sweating all over the place and not feeling like shit and/or dehydrating in the middle of summer while going on hour or longer rides in 85 plus heat? Let me tell you again that I hated the way DNP made me feel.

YOU HAVE TO TAKE GLYCEROL AND PYRUVATE!

I don’t know if you can take one without the other because I was using the glycerol (G) and pyruvate (P) to enhance endurance and stem dehydration which I am very prone to. I think, however, that you will have to do them both as there may be a synergistic benefit. I have taken G alone and while you have more water to hold on to, you just seem to sweat more which is also backed up by the research. The G-P cocktail let me drink ½ my normal volume of water on rides and that is what made me try the DNP-ECA experiment.
Glycerol dose. YOU ONLY NEED 1 TABLESPOON 3 TIMES A DAY! One in the morning, one in the afternoon and one right before bed. Don’t listen to the researchers who tell you to take 1 gram for every kg body weight 1-2 hours before and event. They are idiots and obviously have never taken it or asked the athletes how they feel. Let me tell you, you feel awful taking that much glycerol! You feel bloated and sometimes get a headache and you piss A LOT! 1 tablespoon 3 times a day comes out to ½ what they recommend to take 2 hours before an event. The glycerol keeps your muscles hydrated and limits the sweating. It will fight the dehydrating effects of the ECA. As we know, DNP is carbohydrate/fat specific and glycerol also is a 3 carbon carbohydrate source that can’t go to fat!. Glycerol also increases power output which may be an added benefit of the type of carb source it is. Glycerol is converted to glucose in the liver and in the liver is where it stays for the most part and does not, therefore, raise insulin levels.

Before I explain the pyruvate, let me tell you that glycerol and glycerin are the same thing! A good recipe for taking your glycerol is 1 packet of Kool-aid, 1/8 cup sugar, 1 tablespoon glycerol and 32-oz of water. The glycerol makes the kool-aid taste like OJ because there are alcohols and fermentation products in the OJ which the glycerol mimics.

Pyruvate dose. 2-5 grams a day. Start out at 900mg or so 3 times a day and go up from there. I am at 1.5g 2-3 times a day and if you don’t work your way up it will give you gas and the runs. As I mentioned above, I think the P is working synergistically to hold on to the water in your muscles. Additionally, it is another 3 carbon energy source and/or it is manipulating the Krebs Cycle intermediates and allows for a different energy production pathway. Pyruvate changes/manipulates an ATP/energy pathway and decreases lactic acid output and if it ain’t the Krebs cycle I don’t really care. It works.

Some abstracts on the benefits of pyruvate.

Pyruvate and the heart and glucose and insulin.

Cardiac metabolism and electromechanics of human heart.
Author Prasad K
Source Recent Adv Stud Cardiac Struct Metab, 10():119?37 1975
Abstract

The effects of substrates on the metabolic inhibitor-induced changes in the action potential and contraction of papillary muscles obtained from patients undergoing corrective open-heart surgery were studied. Anoxia produced a marked shortening of the action potential duration and a decrease in the resting potential, rate of rise of action potential, effective refractory period, and contractility. In anoxic muscle, although glucose completely restored the action potential duration, effective refractory period, and resting potential to control levels, it was unable to completely restore the contractility to the control level. Substrate depletion and metabolic inhibitors (iodoacetate, dinitrophenol) produced effects similar to that of anoxia, but at a faster rate. Glucose restored the action potential and, to a lesser extent, contractility to the control level in dinitrophenol-treated muscle but was ineffective in so doing the iodoacetate-treated muscle. Pyruvate, however, was effective in restoring the action potential and contractility in iodoacetate-treated muscle. Pretreatment of the
muscle with glucose and, particularly, with glucose plus insulin prevented the combined effects of anoxia and lack of glucose on the action potential and contractility for a prolonged period. These results suggest that intravenous infusion of glucose and insulin before and during surgery might prevent or reduced the effect of anoxia on the electrical and mechanical activity of the heart during open heart surgery.

Pyruvate was able to restore the action potential (charge) of cells treated with DNP! Unfortunately, most of you won’t understand what an AP is even if I explained it, but I will tell you that restoring it is significant!

Now you have a recipe on how to feel good on an overdose of DNP! To recap you need:
3 Tablespoons glycerol 3x a day.
1g Pyruvate 3x a day.
Taurine at 3g a day.
DNP at 36 hour intervals.

If you start to feel bad, just drink some sugared pop or take some glucose or maltodextrin with your psuedo-OJ mix. The only drawback is that you will still smell rather bad and will emit a vinegar type odor although you won’t be sweating all over everything. If you notice you are starting to sweat more it is time for another glycerol dose.
not stay on cytadren longer than one month and dosage is one tab divided into 4 doses per day.
A line from a study showing that estrogen makes you fatter for the non-believers!
Obes Res 1995 Nov;3 Suppl 4:561S?568S
Topical fat reduction.
Greenway FL, Bray GA, Heber D
Department of Medicine, UCLA School of Medicine, Torrance, CA, USA.

The fat on women’s thighs is more difficult to mobilize due to increased alpha?2 adrenergic receptor activity induced by estrogen. Lipolysis can be initiated through adipocyte receptor stimulation (beta adrenergic) or inhibition (adenosine or alpha?2 adrenergic) or by inhibition of phosphodiesterase.

While yes, they talk about women and estrogen and fat, the mechanism is still absolute and spans the sexes. Estrogen makes fat cells resistant to lipolysis. Still want to take that test without and anti-estrogen?

Get to fat burning faster!

This is something you can try after you have used DNP once and know your tolerance and is a DNP manual exclusive! Your working dose will be around 400mg a day, correct? The first day, however, you are going to take 600mgs in divided doses! It takes DNP a couple days to build up so this won’t bother you in the least. On the second day you will start taking 200mg caps every 8 hours until you are sweating or getting the heat you want. Now you are at your tolerance dose and you can space it out to the 36 hour dosing.

NEW!
Use a blood buffer to combat free radicals and lactic acid!

Add up to one tablespoon of baking soda, sodium citrate, or potassium citrate to your drink of choice throughout the day. A mix of the sodium and potassium would be best. Why?
What does DNP and exercise have in common? During high intensity exercise (supramaximal) ATP production is supplied by anaerobic glycolysis. This increases levels of H+ (protons) both inside and outside the cell via lactate and results in the feeling of fatigue (Hermansen and Osnes; Sahlin) In the past, the use of sodium bicarbonate (baking soda) has been used and has been shown to decrease acidosis via buffering of the blood. The problem with baking soda is gastric distress and high salt intake with the recommended dosage of 300mg/kg which is around a tablespoon of baking soda for most people. Dosage for sodium citrate is 100mg-500mg per kg and did not give stomach problems to the users. Time to exhaustion was increased 15% which is the same as with baking soda. Alkalosis (making the blood basic) has been found to increase the rate of lactate and proton release from muscle into the blood. An increase in muscle pH causes phosphofructokinase inhibition (PFK) which is the controlling enzyme in glycogen utilization and therefore causes an increase in lactate formation. Those two mechanisms also will hold true for DNP as DNP releases protons which causes the heat. Get it out of the cell with the citrates.
Testimonial:
Hey animal just thought I’d let you know the great results I had with the DNP. I got tested hydrostatically and I’m at 4.8%. DNP is really the shit. Anyway, a buddy of mine is competing in a month and he’s currently at 7% bf (he’s about 190lbs) and he’d like to do it for two weeks. I understand that the lower one’s bodyfat % the greater amount needed (I responded very well to 4mg/kilo but I was also dieting)

Questions I have answered for DNP users

Are all of the losses on DNP fat losses?
You can’t ever say ALL in the scientific world, but it is the best we can get!

Animal, you have been a big help already. I’ve got some questions for ya. I am 195#, and plan on taking 200 mg in evening and 200mg before bed. I also plan on taking pyruvate and glycerol (via your recommendation). I’ll going to use a 8on/8off cycle.
1) Should I attempt to lift while on dnp?
Sure.

2) What dosage glycerol and pyruvate do you recommend?
3 tablespoons a day on the G and 3g of the P

3) You said that you experienced an anabolic "burst" directly after coming off of dnp. Could this be contributed to your muscles reglycogenating themselves?
No, because I didn’t really get pumped, but strength went up.

or do you feel that this is genuine protein synthesis?
Yes, or nerve excitation/generation or a return of T3. Let me explain the nerve generation in more detail for a moment. When you do strength exercises of 5 reps or less you are training the nerves to fire more muscle cells and to fire those muscle cells in the sequence you want. Now, if we add DNP we are exhausting our muscle cells and they can’t fire as strongly. Result? Your nervous system trains more nerves to fire other muscle cells which had previously gone un or underused when energy stores were high. You are getting a nerve training session due to exhaustion! The more I think about it the more it is like those overtraining programs where you overtrain for a week and you get a rebound. That is what is happening except you are overtraining at the same weights due to the DNP.
Hehe. It is called overcompensation training and I’ll take it!

A user:
Found some info 'bout DNP "With even a low dosage, in the area of 3?5 mg/kg of body weight a day, it will rate your metabolic rate 30%. If this dosage is continued daily, it will raise your metabolism by 50%. At this rate you can burn about 1 lb. of fat a day."

Animal:
Now, let’s think about this for a second. If metabolism can go from 30?50% that means there is a residual amount left over from the previous dose and therefore
the 36-hour clearance dosing schedule which I recommend. Furthermore, when I overdosed on 800mg I sweat for OVER 48 hours so this tells me that the half-life can be even longer in some circumstances. It is, nonetheless, up to you as to how you want to take it. If every 24 hours is tolerable for you, then do it.

A user:
That’s not what I’ve noticed. At present I’ve even gained some weight. I’m 98 kg now. I don’t look a bit harder but maybe I shouldn’t expect that after only 4 days. You WILL hold onto water! You WILL be depleting carbs from the muscle that will make you look flat! Most WILL NOT notice the benefits until a week or two later upon cessation of DNP. Some see benefits right away, but they appear to already have a bodyfat below 10%.

A speculator wrote:
For a person that is highly active and on a calorie restricted diet, DNP will deplete ATP within a matter of days. When this happens your body temperature will go back to normal. The only thing you can do at this point is supplement with in the dosage area of about 150 mcg/day."

Animal:
Believe me, you will feel wasted (tired) and LOSE muscle on the regimen and the liver does not control oxidative phosphorylation in every cell. You will still be hot regardless of your T3 levels.

Question:
Won’t the conversion of T4-T3 come back and function again after discontinuing DNP administration? 
Animal
Yes, and as long as you do some carbs at 600g a day for three days after.

Or do I have to look to get T3 as well?
Animal
Not really, if ATP was being ever totally depleted you would be cramping. Lack of ATP generation happens when you are what? DEAD!!!!!! It is called rigomortis and if people who write right about DNP had an education or an inkling about body chemistry and process they would know this! Total ATP depletion resulting in death of the cell is not possible. There is some safety mechanism and I imagine it is via the fat cells, but you will not deplete your ATP unless you are an anorexic or dead.

User:
And isn’t a total lack of ATP what we want, so we can start burning fat instead of ATP?
Animal:
Wrong and this is not your fault, but again those who claim to be experts who are dispensing such disinformation. Once the ATP to ADP and to AMP is changed below a certain amount the cell gets it energy from another source which would be the fat. You are looking to burn the glycogen and then the fat is used. This does not mean ATP is gone!

Why is thermogenesis stopped if there isn’t any ATP?
Animal:
Read above and it is never stopped. DNP NEVER stops working unless the proton gradient is severely altered. Even in such a case, parts of the mitochondria can have one direction of proton gradient while another section can have a DIFFERENT proton flow!

User:
Is this why you want a break after one week? To load up with ATP so thermogenesis can start again?
Animal:
No, so you don’t feel like shit all the time and so you can get the liver converting T4-T3 again.

Is a one week break enough? Or maybe too short if I’m in a hurry.
You could do DNP for 2 weeks or more if you want.

Wouldn’t it be enough just to discontinue the DNP cycle to let the liver start converting T4 to T3?
That is why you stop after a week!

Where are ATP molecules stored?
In and around the mitochondria and in the cytosol of the cell and ATP is attached to certain enzymes waiting for activation.

How much ATP do we have in storage?
That is a major calculation and I haven’t looked for an answer, but I don’t think it is important as you can never get rid of all of it.

A speculator:
"The administration of DNP, at a dose of 3.5 mg per kilo, increases the total production of heat by about 40%, from the 3rd or 4th day. This increase of the metabolism is due MOSTLY to an increase in the combustion of the fat and a LITTLE to combustion of carbohydrates."
Any comments on this?
Animal:
Again, you are seeing a residual affect. The molecules that the mitochondria use for production of ATP can come from carbs or fat, but the important part is that it is not from muscle.

Another fact found in the same report as above:
"In prolonging the administration of the medication, one observes an increase of the tolerance of carbohydrates."
Does this mean we get increased insulin sensitivity?
That is a weird sentence and I don’t know what it means, but I think it means you will be more receptive to carbs. You now have increased your insulin sensitivity and this could explain the DNP users’ craving for carbs while using DNP.

So when I eat carbs (I’ve noticed that I start to sweat then) the body starts burning fat?
No, the body burns excess ATP and food intake itself is thermogenic.

Why simple sugars? That means I should walk around eating candy all day?
Candy is not really a simple sugar as it usually has fat or fructose with it.
You want a simple sugar every so often to get some insulin rise and some
Fructose will help recarb the liver as fructose is about 4 times better at recarbing the liver than glucose. Glucose, BTW, 2 times at good at recarbing muscle when compared to fructose.

What happens with the carbs?
They are burned and insulin release and helps change charge on the cells.

Insulin is secreted. I've noticed that. (If it wouldn't I'd go glucose?high?n? crazy.) Do I store carbs as glycogen? AHAHA! So much for the speculators that say you have to do insulin! You won’t have time to make glycogen and the glucose will go right to ATP production.

What about cataracts and skin lesions?
That is a long term chronic dose situation and why you take pyruvate.

Have you noticed anything?
Yes, your sweat smells bad, but no lesions. Another reason you want simple sugars or insulin is that DNP starts to make your vision blurry if you are on a low carb diet.

Animals’ Analysis of someone else’s recommendations:

Comment
After 7 days, DNP dislodges T4 off the carrier proteins, allowing the T4 to be excreted rapidly.

Animal:
THIS IS A FUNCTION OF THE PRESENCE OF ATP. END OF DISCUSSION! This has been proven with many people who have used pyruvate which provides an easily usable energy source. Most users only stay on it 7 days so the point would be moot. Since you have depleted the carbs from the liver you are changing the ability of the liver to change T4 to T3. This happens with ANY diet within 7 days. With DNP you have inhibition of conversion via heat (small factor I believe) and via glycogen depletion. This loss of water due to glycogen depletion changes the osmolarity of the liver cells and inhibits the conversion of T4-T3. Now, with the concomitant loss of water you have a loss of charge which is what we are trying to control with the taurine dosing..

Comment
I have used T3, and recorded the average elevated body temperature at day 4 on DNP. After 7 days, the temp will decline, so I use T3 2X a day to restore the elevation.

Animal.
Really? Most people, including myself, hardly notice any temperature change. I used T3 at 50mcg up to 100mcg on day 5 and never felt worse or more run down than any other DNP experiments I have done.

Comment:
It really doesn’t matter how much or how long for the T3, because though excessive-looking, T3 blood level will be normal.
Animal:
This is NOT true because people have had their thyroid tested while on DNP and their thyroid levels were sky high. Excess thyroid can be responsible for what when calorie deprived? Muscle breakdown. Carbs are gone due to DNP. Your cells are going to be looking to scavenge energy so they are not going to have any protein synthesis because this requires energy. You are going to be in ketosis which is producing glucagon which is responsible for protein breakdown. You will, therefore, have no insulin which is responsible for anabolism of glycogen. You will have no blood sugar or liver glycogen left. Now what is going to happen?! Muscle breakdown. DNP is carb/fat specific and since there is no glycogen/glucose circulating due to high fat-low carbohydrate diet, where is the energy coming from? Ketones can't be made into carbs and about the only source of carbs you are going to have is the glycerol molecule which results from fat breakdown which is minimal. Now throw your excess T3 on there. Hmmm? Sounds like a recipe for muscle breakdown to me.

Comment:
Besides, you'll get sick before you have to worry of being on T3 so long. Trust me, children: DNP for 7 days, and 7 off. You'll be much healthier.

Reply.
I totally disagree! Many of us have permanently lowered body temps due to clen?T3 usage which many of the same moron gurus recommended even when using clen for 2-3 weeks. The thyroid is going to see excess T3 in the blood and do you think it is going to want to produce more T4 and T3 on its own? This is what I really don’t like about doing the T3, here. Yea, it is only for a week, but 2 weeks on clen which is not even T3 has fucked up many of us,. If your theory panned out then why couldn’t we do 1 week AS cycles or why have all the 2x2x2 cycles fallen into the pit of futility? I know we are talking different receptors, but they all still function via the negative feedback system.

Auxillary

(Consultation question)
After this Clen, DNP, and high fat diet experience I’m hoping to be down to around 7%BF. Which would leave me at about 165?170lbs. That is too small for me. I want to go on a cycle after that and try to put on a good 20lbs. I have a great diet for my cycle, so I know if I dont make the 20lbs gain I want its not because of nutrition. ( A problem i seem to always have). I wanted to know your thoughts on a cycle that I could really put on a good 20lbs. I know how much of your gains you keep depends on what you do prevent losses ( example: Clomid, and something to regulate cortisone levels, along with others, I have a gains keeper formula I plan to use). but If I do this I want to keep the majority of my gains. That’s why I wanted to include primo since you usually keep what you gain from primo.

Answer
Yea, but you don’t gain much and Eq or ganabol would be better as would fina.

Can you think of a good combo to add to primo for permanent MASS gains??????
Answer
Test (Tp, Tc, Te) or a trenbolone (fina, anibolan, parabolan). D-bol then fina always works nicely, too.

Maybe deca and sustanon or deca and omandren???
Answer:
Wouldn’t go with deca and sus and omna are more or less the same.

Any other s??? ? I have heard if you want to keep gains tests are not good to use (enthanate, cyp etc.)
Answer:
BS. You have to know how to come off and not overtrain as you are coming off. Think about it. You have gotten stronger which is a result of nerve training. Now if you lift and let your muscles recover longer when off the AS you won’t lose your size!

. Do I have to take insulin while on DNP if I am taking equipoise and finaplix?
Answer:
No, not really and not if you are going to stay on only for a week at about 400mg or less DNP dose.

Do I have to take cytomel, clen or ECA stack while on DNP?
Answer:
I would take EC, but do the others after being careful to note that Clen and T3 will suppress your natural T3. Would be better to throw in tyramine and yohimbine or mazindol.

I have quite a bit of clen, and cytomel, but no ephedrine.
Answer:
You can sub in PPA or adipokinetix or pyruvate or nicotine or mazindol. (I have never had a problem doing nicotine as a chew or as cigars and then quitting, but this is obviously not for all)

I didn’t understand if you said whether or not to start with a low dosage of DNP or not.
Answer:
I would if you have never done it before just to see what your tolerance is.

Question:
Also, you told me that I should not take cytomel while I am using> the DNP but to use it after the DNP. I was under the impression that DNP suppresses the thyroid and that I should use the cytomel while using the DNP so I will keep burning fat. Would you please explain this to me?

Answer:
DNP alters the blood and liver glucose levels and THIS is what keeps the liver from converting T4?T3. If you eat normally this won’t happen so drastically and T3 will return to normal soon after stopping DNP. Now, if you have low Blood sugar levels and you add T3 you are going to lose muscle as well as is seen in people that are on low calorie diets who supplement T3. T3 without the right energy and hormones stores is disastrous to muscle.

Other dieting stuff
ketotifen and upgrade of clen receptors, but you need 10 1mg tabs of ketotifen a
day which will make you hungry and sleepy.

I was thinking of the efficacy or more like the ‘sense of adding t3 toDNP.
Well, if you are adding-T3 you are going to have a lot of T4 AND T3 floating around and the thyroid is going to read that as an exess and shut down T3 and T4 production.

Other cycles for DNP use

Why not do DNP in even smaller doses like that of ephedrine up to 100mg or so? It will speed up the metabolism and cause a loss of weight without all the discomfort and t4-t3 conversion shutdown. Furthermore, by speeding up the metabolism it may help upgrade steriod receptors and clen receptors with much less discomfort for the user.
DNP seems to upgrade clen receptor sites as well as steroid receptor sites. The rebound for the AS upgrade is only known from anecdotal feedback from myself and others, but if you increase the metabolism of the cell it only stands to reason that you are going to decrease the time it takes to regenerate the receptor sites. The ATP depletion and opening of ATP channels is also likely to be playing a part in these benefits as well, but that is research that probably won’t be done. So the channel part in the upgrade is just speculation.

Some may need to build?up the dose to start. I had to do it for 3 days and then do 800mg before I started to sweat like a pig for 2 days! Now moderate doses of 200mg make me sweat although not to the same extent, but at least I know I’ve taken it. Kinda like bee stings. You don’t have any allergic reaction until one sting and then you get the benefits (problems) from one sting. I do know of a case with a women that had similiar results and said it wasn’t working and even talked to w8 about it, but then she ordered more so this has to be what the problem was/is.

Response to someone that was throwing up and nauseated from DNP use.

You have low blood sugar!
This is a classic symptom which can occur with diabetics who use too much insulin. When I use too much insulin and then ride too soon after I would see spots. DNP caused me to see spots as well. DNP depletes all your blood sugar and glycogen first and this will give you low blood sugar, nausea, etc. That is why you want to get your insulin up with glucose once or twice a day on DNP and DO NOT do high fat diets on DNP. W8 will disagree with me on this, but when you look at the actions happening at the liver you will realize that high fat diets just extenuates the slowdown of T4 to T3 conversion.

Q: But when I’m off I’m gonna keep carbs almost non-existant to burn more fat, and take Adipokinetix to avoid a rebound off of the DNP.
A: DNP stops conversion of T4-T3 due to carb depletion so you may not want to do that although the Adipo is good. DNP, Adipo, clen, ECA, DNP would be a good way to go or do the clen right before a week of DNP, but only for a week.

Q: IM OFF MY CYCLE IN A WEEK. THEN IM GONNA TAKE CLOMID, PS,NOLVADEX, TO AVOID A LOSS IN GAINS. THEN ITS CUTTING TIME.
A: Nolvadex and clomid are redundant, do one or the other. PS sucks, but if you already have it, then use it.

---

**Title:** Re: AAS Studies/Links/Literature  
**Post by:** jmt1 on **October 15, 2006, 11:11:06 AM**

Good things about DNP:

**Biological Study of Dinitro Drugs in Humans**  
By Dr. Jacques Bell  
Translation Copyright 1996 Robert Ames

There is a fundamental difference between biological experimentation with dinitrophenol in humans and what was done in the laboratories of physiologists. These last are essentially interested in hyperthermia (Andre Mayer, Leon Binet, etc.). Yet, in medicine, the doses of dinitrophenol employed do not determine any elevation of temperature. The physiological effects, observed in these conditions, differ considerably from those made by the experimenter. It is thus for example that the animal in hyperthermia presents a polypnoea [rapid, shallow breathing], a hyperglycemia, a hypoglobulinemia that one does not observe with therapeutic doses; it is because experimental hyperthermia is essentially a combustion of carbohydrates, while therapeutic hypermetabolism is mainly a combustion of lipids, as is shown by the lowering of respiratory quotient.

One shouldn’t be surprised at these differences. The clinician uses strychnine as a tonic; the experimenter employs it to cause convulsions. The clinician uses adrenaline, at titrated doses, to produce a manageable hypertension; the physiologist, with massive doses causes acute edema of the lung. Yet, to base the clinical use of adrenaline or of strychnine on acute edema of the lung or experimental convulsions, constitutes an obvious error. It is the same for dinitrophenol.

In France, besides, one uses almost exclusively dinitrophenyllysidine, which, according to the same terms of the study made by Professor Pouchet, "is easy to purify by crystallization, to easily modify the first of its components from the point of view of toxicity, dissolves easily in water, and, by addition of the methylglyoxalidine (lysidine) group, favors energetically the elimination of waste."

After Professor Pouchet, we have, in our thesis [1], demonstrated the superiority of this last product; in what follows, it is by comparison with him that we will study the biology of the dinitro drugs.

I. Their action on the basal metabolism,  
II. Their visceral action,  
III. Their nutritional action.
I. ACTION ON BASAL METABOLISM

After the experimental research of Magne, Mayer and Plantefol, in animals, the experiments of Cutting and Tainter has confirmed, in humans, that dinitrophenol is a drug which strongly increases the metabolism, exaggerating the oxidation process of the organism by direct action on the cellular metabolism. These authors have observed a rise of close to 20% after one hour, being able to attain 70% in ten hours and a tendency to return to normal at 24 hours if the administration of the medicine is not continued.

This increase is not due to a sympathetic deregulation. The dinitro treatment respects the autonomic nervous system, in an inverse way from thyroxine, which, at slimming doses, determines rapidly some tremors, insomnia, and a mental instability of the type "basedowien." [a thyroid illness where one secretes too much thyroid hormones]

In the thyroid illnesses, or the thyroid treatments, there is an inverse connection between the level of the basal metabolism and that of blood cholesterol, this being as much lower as the metabolism is higher. One doesn’t observe similar phenomena in the course of dinitro treatment. This fact indicates that the changes caused in the blood cholesterol in the course of thyroid treatment are directly linked with the thyroid medication and not at all to do with the elevation of the metabolism which is responsible for the reduction of obesity. Dinitrophenol has almost no action on the blood cholesterol. (Grant and Schube).

An attentive exploration of the nutritional changes in the course of dinitro treatment, in the cases of five obese women, has shown the following facts:

1. The administration of dinitrophenol, at a dose of 3.5 mg per kilo, increases the total production of heat by about 40%, from the 3rd or 4th day.

2. This increase of the metabolism is due mostly to an increase in the combustion of the fat and a little to combustion of carbohydrates.

3. Dinitrophenol does not attack cell tissue albumin and does not determine the fat loss to the expense of the muscles, contrary to thyroxine.

II. VISCERAL ACTION

Dinitro treatment respects the liver, the kidneys, the cardio?vascular system and the blood.

This innocuity for the principal visceral functions is without doubt one of the main reasons for the distribution of this therapy. Tainter, Stockton and Cutting have reported a series of cases in which one had measured the plasma bile index and determined the test of Van de Bergh. Their analyses demonstrate, beyond a doubt, that the liver does not suffer any damage in the course of dinitro treatment.

Experimental studies on animals do not show toxic effects of dinitrophenol on the kidney (Taitner, Cutting, Woodand Proescher). Anatomical?pathological examinations of animals, even those which died from a massive dose of
dinitrophenol, do not reveal any important anatomical changes, except a small degree of cytolysis. Clinical documents are not abundant, but, on the whole, do not seem to demonstrate that dinitrophenol is toxic for the kidneys.

As T.L. Schulte and M.L. Tainter wrote, "it doesn't seem that dinitrophenol at usual clinical doses is likely to harm the kidneys."

Dinitrophenol is remarkable for its absence of effect on the cardiovascular system. Even when the basal metabolism is found elevated to significant levels, there is no change in the rhythm of the pulse (Rosenblum).

On this point, dinitrophenol differs from all the other metabolic accelerants known. It is an observation that all the clinicians, today, have had occasion to make.

All the clinicians know that, contrary to thyroxine, dinitrophenol is absolutely devoid of toxicity for the heart.

The research of Professor Loeper and of his students has demonstrated the physiological and clinical importance of myocardiac glycogen. Extensive studies by P.N. Taussig have shown that dinitrophenol does not reduce cardiac glycogen at all and that, on this point, it differs completely from thyroxine.

III. ACTION ON NUTRITION

The influence of dinitro therapy on nutrition has been the object of a very important clinical study.

"One does not observe variations in the elimination of chlorine; eliminated phosphorus varies sometimes more, sometimes less, the elimination of sulphur increases slightly, especially in the form of sulphur conjugates, urines show a small increase of total nitrogen and of urea." (Prof. Pouchet).

It is a well known fact that the administration of thyroid extract or hyperthyroidism is accompanied by an increased secretion of calcium and of phosphorus. This calcium and phosphorus in the urine are not due to the acceleration of metabolism, as one does not observe these facts either during fever, nor in the course of leukemias which raise the metabolism (J.C. Aub, N.B. Bauer, C.I. Heath, Alright, Bauer and Aub).

Thyroxine reduces bone density.

With dinitrophenol, nothing of the sort is observed. The experiments of Clarence L. Robbins show that dinitrophenol, in spite of the elevation of the metabolism that it produces, does not cause any increase of the loss of calcium or of phosphorus. An increase of 37% in the basal metabolism, caused by the ingestion of dinitrophenol, does not lead to modification in the excretion of these elements.

In normal individuals, when one administers dinitrophenol during a short period, it produces a small elevation of reduced substances in the blood after fasting (although one would not be able to call this hyperglycemia). When one administers the medication over a longer period, this phenomenon is not produced and there is a marked elevation of the tolerance to carbohydrates.
In diabetics, following treatments of short duration, the results are variable, the tolerance to glucose being as often increased as it is decreased, with parallel changes in the fasting blood sugar level. But, in prolonging the administration of the medication, one observes an increase of the tolerance of carbohydrates. Anyway, in basing himself on the study of 32 cases of diabetes, Simkins concludes that dinitrophenol is not toxic for diabetics. Here marks that this observation goes counter to some assertions that have been a little prematurely advanced.

Dinitrophenyl?lysidine at therapeutic doses therefore has an action on the organism which is completely physiological. This action has been demonstrated in obesity where it has been compared to the actions of thyroid medication and physical exercise.

The existence of obesities of glandular origin, especially by thyroid insufficiency, has resulted in the use of thyroxine in numerous subjects.

"This wasn’t without inconveniences, sometimes grave, characterized especially by cardiac and nervous troubles; the effective dose of thyroxine is, in fact, very close to the toxic dose. Further, we has frequently seen these accidents persisting after the administration even of a single dose, which leads to the impossibility of stopping them immediately by a simple suspension of the medication. Yet, from the point of view of its specific action on the basal metabolism, dinitrophenol offers this precious advantage that the cessation of its use at the slightest appearance of signs indicating an imminence of intoxication results immediately in the arrest of those symptoms." (Professor Pouchet).

Finally, thyroxine causes a nitrogen malnutrition: it burns the muscle and fatigues the heart. Dinitrophenyl?lysidine, to the contrary, causes a lipid?glycemic loss: it is the elimination of reserve materials without attacking visceral and muscle tissue.

As for physical exercise, it seems to act exactly like dinitro therapy. Marcowitz, in his communication to the Academy of Medicine of Toronto on October 9, 1934, based on 90 cases of obesity, having followed this treatment during a period of 16 months, concludes that its action may be succinctly described in saying that the effects on the organism are similar to those of physical exercises.

The fact is besides established by physiologists, since dinitrophenol raises thermogenesis and not the metabolic quotient.

All the clinicians know actually that dinitro medication is irreplaceable in cases of monstrous obesities which prevent all exercise. It can be used in the obese for whom occupations, life style or cardiac troubles do not permit physical exercises. It is indispensable for the grossly obese in cases of abdominal operations and immobilization due to illness (inflammation of fallopian tubes, appendicitis, etc.) for which there is an urgency to obtain a reduction of subcutaneous fat.

But this clinical use has not been able to be extended other than when the experimental research pursued on humans, with a dinitro drug free from impurities, has been able to demonstrate the biological effects of it in a very
Ion channels are membrane proteins that control the flux of ions across an otherwise impermeable cell membrane. Potassium (K) channels were first described by Noma [1] in 1983, and later in 1991 the ATP-sensitive K channel (KATP) was described by the same researcher [2]. Potassium channels determine cell membrane potential.

KATP channels exist in most excitable cells. They are regulated by the intracellular level of ATP as well as by various nucleotide diphosphates, pH and lactate concentrations. The activity of KATP channels is inhibited by increasing the intracellular ATP concentration. Closure of these channels in response to glucose metabolism depolarizes the cell, stimulating voltage-dependent electrical activity, and calcium ion (Ca) entry. In the pancreatic beta cells, an increase in blood sugar level leads to an elevated ATP/ADP ratio, which in turn inhibits KATP channels, so as to alter the membrane potential and cause insulin release. It is accompanied by increases in respiratory rate, pyridine and flavin nucleotide reduction state, and intracellular pH [3]. Thus, the KATP channel couples nutrient metabolism to the membrane potential.

- Increase in blood glucose ⇒ increase in glucose metabolism ⇒ increase in intracellular ATP ⇒ inhibition of KATP channel.

- Channel CLOSED: cell depolarized, Ca++ uptake, insulin exocytosis.

KATP channels play an important role in the control of vascular tone [4]. Polarization following potassium channel activation (opening) results in lessened calcium influx and smooth muscle relaxation.

- KATP channel BLOCKED ⇒ vascular tone increases.

- KATP channel ACTIVATED ⇒ vascular tone decreases.

Besides being regulated by intracellular signals, potassium channels may also be regulated by membrane potential. Thus, in excitable cells in the heart, muscle, and nervous system, voltage-gated potassium channels are activated during an action potential; the activities of these potassium channels determine to a large extent the shape of the action potential, hence the strength of the signaling.

- KATP BLOCKED ⇒ more strength

- KATP ACTIVATED ⇒ less strength

Drugs which block KATP channels: tolbutamide, glyburide, glibenclamide.
Drugs which activate KATP channels: Prostaglandin E2 and I2, adenosine, lemakalim.

Drugs which activate K channels: pinacidil, cromakalim.

Mitochondria also contain a K+ channel that causes rapid K+ uptake when open [5].

DNP

What happens when someone takes the uncoupler dinitrophenol (DNP)? Blood glucose will result in increased metabolism, but the level of ATP in the cell does not increase! In fact, it is depleted. So in this case, the KATP channel is not inhibited, and it stays open. Calcium is not taken into the cell, and insulin is not released. The person taking DNP has in effect given himself temporary diabetes. (Animal; another study shows that insulin internalization is also affected so taking insulin is useless)

Insulin is needed to facilitate the uptake of glucose into cardiac, skeletal, and adipose tissue, and to convert glucose to glycogen in the liver. It is anti-proteolytic and protects against the various ailments commonly seen in diabetics, such as vision problems and polyneuropathy. Not coincidentally, the same problems can result from ingesting DNP.

This is why, when one takes DNP, one also needs to take exogenous insulin. Since the KATP channel remains open, vascular and muscular tone relax. Probably blood pressure will decrease. Strength will diminish.

It would seem that an antidote for DNP might be anything that causes the KATP channel to close, for example the drug glibenclamide.

Animal;
Why all this is a good story we do have to look at what he said in the beginning as in caveat lector. While his speculation on KATP channels and the need for insulin is understood-, his mechanisms are a bit incorrect. DNP causes an internalization of the receptor so you now have a cell that is desensitized to insulin and all the insulin in the world will not help that. Second, the KATP channels can be controlled with pyruvate.

Studies

DNP causes insulin insensitivity by preventing internalization of the receptor.

Insulin internalization into monocytes is decreased in patients with type II diabetes mellitus.
Author Trischitta V; Gullo D; Squatrito S; Pezzino V; Goldfine ID; Vigneri R

We studied the internalization of [125I]insulin into circulating human monocytes, a cell type widely used for insulin binding studies. The internalization of [125I]insulin was assessed by both an acid extraction technique, which removes surface-bound insulin but not intracellular insulin,
and by a trypsinization technique, which removes cell surface-bound hormone. After 5 h of incubation at 22 C, over 40% of the total cell-associated [125I]insulin was internalized into monocytes of normal subjects. This internalization was temperature dependent; the fraction of internalized hormone was progressively decreased when the incubation temperature was reduced from 37 to 4 C. Treatment of monocytes with increasing concentrations of 2,4-dinitrophenol also decreased [125I]insulin internalization, whereas dansylcadaverine, an inhibitor of transglutaminase, had no effect. Analysis by gel filtration of the internalized labeled hormone after 4 h of incubation at 22 C indicated that 50-60% of the label was degraded insulin, but detectable intact insulin was still present. Internalization of insulin was then studied in monocytes from eight obese patients (161% of ideal body weight) with type II diabetes mellitus. After 4 h of incubation at 22 C, the specific total monocyte-associated [125I]insulin was decreased compared to that in cells from 7 normal subjects [6.02 +/- 0.38% (+/- SE) vs. 3.91 +/- 0.31% of the total; P less than 0.001]. Moreover, the percentage of hormone that was internalized was also decreased from 41.4 +/- 1.2% of the total to 28.9 +/- 1.8% (P less than 0.001). In 20 nondiabetic obese subjects, specific cell-associated [125I]insulin was reduced to 3.9 +/- 0.3% (P less than 0.001). However, compared to that in normal subjects, the percentage of hormone that was internalized was not decreased (39.7 +/- 3.51% of the total). The present findings indicate that human circulating monocytes internalize [125I]insulin; this process is temperature and energy dependent; and monocytes from obese type II diabetic patients have a significantly decreased ability to internalize insulin. This decreased internalization may play a role in the cellular resistance to insulin that occurs in these patients.

Title: Re: AAS Studies/Links/Literature
Post by: jmt1 on October 15, 2006, 11:12:08 AM

DNP enhances binding of insulin to the receptor, but does not internalized it.

The effect of phenformin and other adenosine triphosphate (ATP)-lowering agents on insulin binding to IM9 human cultured lymphocytes.
Author Vigneri R; Maddux B; Goldfine ID
Source J Cell Biochem, 24(2):177-86 1984
Abstract

In the present study, we investigated the mechanism by which the antidiabetic drug phenformin increases insulin binding to its receptors in IM9 human cultured lymphocytes. After a 24hr preincubation, phenformin induced a twofold increase in specific 125I-insulin binding, and removal of phenformin was followed 6 hr later by a return in binding to control levels. This effect of phenformin on insulin binding was not a consequence of either inhibition of cell growth, changes in cellular cyclic adenosine monophosphate (AMP) levels, or changes in guanosine triphosphate (GTP) content. Since phenformin is known to inhibit various aspects of cellular energy metabolism, the relationship between 125I-insulin binding and energy metabolism in IM9 cells was investigated. The phenformin-induced increase in insulin binding to IM9 cells was related to a time- and dose-dependent decrease in ATP levels. Other agents that lowered ATP levels, including antimycin, dinitrophenol, and 2-deoxyglucose, also raised insulin binding. These studies indicated, therefore, that phenformin enhances insulin binding to receptors on IM9 cells and that
Evidence for two independent pathways of insulin-receptor internalization in hepatocytes and hepatoma cells.
Author McClain DA; Olefsky JM
Address Department of Medicine, Veterans Administration Medical Center, San Diego 92161.
Abstract
A study of insulin-receptor internalization and recycling was undertaken in primary cultures of rat hepatocytes and a human hepatoma cell line (HepG2). Receptors were quantitated by measuring 125I-insulin binding to partially purified soluble receptor preparations from untreated cells (total receptors) and trypsinized cells (intracellular receptors). In resting HepG2 cells, exposure to insulin results in internalization of insulin receptors, the rate and extent of which is dependent on the insulin concentration. However, receptors do not accumulate inside the cell in proportion to the higher rates of internalization at high concentrations of insulin. This lack of accumulation is explained by much higher recycling rates after exposure to high concentrations of insulin. Similar results were noted for primary cultures of rat hepatocytes. These results imply qualitatively different fates for receptors internalized after exposure to different concentrations of insulin. To further investigate the possibility of different pathways for insulin-receptor internalization and processing, cells in low (1 ng/ml) or high (100 ng/ml) concentrations of insulin were exposed to drugs or treatments known to affect receptor metabolism. Hypotonic shock and hypokalemia, which arrest coated-pit formation, blocked internalization of insulin and insulin receptors at low concentrations of insulin but allowed internalization in response to high concentrations of insulin. The lysosomotropic drugs monensin and chloroquine caused intracellular accumulation of insulin and its receptors internalized at low concentrations of insulin but had a relatively smaller effect on receptors internalized at high concentrations of insulin. All internalization is blocked by 2,4-dinitrophenol. We conclude that high doses of insulin lead to insulin-receptor internalization and recycling through a pathway that is functionally distinct from the pathway taken by receptors internalized by low (physiologic) concentrations of insulin. The pharmacologic experiments raise the possibility that the high-dose pathway, unlike the low-dose pathway, may proceed independently of coated pits and endosomal acidification.

Degradation of insulin by human fibroblasts: effects of inhibitors of pinocytosis and lysosomal activity.
Author Kooistra T; Lloyd JB
Abstract
The role of the pinosome-lysosome pathway in the degradation of 125I-labelled bovine insulin by cultured human fibroblasts was examined by comparing the effects of various known inhibitors of pinocytosis and lysosomal degradation on the uptake and degradation of 125I-labelled polyvinylpyrrolidone, formaldehyde-denatured bovine serum albumin and bovine insulin by these cells. Fibroblasts incubated with polyvinylpyrrolidone
steadily accumulate this substrate, whereas incubations with insulin or denatured albumin led to the progressive appearance in the culture medium of [125I]iodotyrosine. Inhibitors of pinocytosis (bacitracin, colchicine and monensin), metabolic inhibitors (2,4-dinitrophenol and NaF), lysosomotropic agents (chloroquine and NH4Cl) and an inhibitor of cysteine?proteinases (leupeptin) decreased the rate of uptake of polyvinylpyrrolidone and denatured albumin very similarly, but only bacitracin had an effect on the processing of insulin. Chloroquine, NH4Cl and leupeptin strongly inhibited the digestion of denatured albumin, but not of insulin. The different responses to the modifiers, with polyvinylpyrrolidone and denatured albumin on the one hand and insulin on the other, suggest that insulin degradation can occur by a non?lysosomal pathway. The very strong inhibitory effect of bacitracin on insulin processing by fibroblasts may point to an important role of plasma membrane proteinases in insulin degradation.

References:

Here is the scare story that is going around on cataract. I raise this because of the connection with this problem and insulin and the previous story. I was starting to see spots and when I took insulin they went away. The myopia stops when DNP is discontinued. Note, that this was a chronic user below.

Subject: DNP and Cataracts

MDGADPC has kindly sent me a photocopy from the French journal Annales des Oculistes, concerning the effects of DNP on the eyes. Since this paper may be of some general interest, I have translated it and attach it below.

For those who may be unaware, DNP or dinitrophenol is a toxic compound which was used for weight loss in the 1930's. It was withdrawn from the market as a result of severe side effects, including deaths, but recently it has been suggested that it could have a role in the contest preparation of elite bodybuilders.

One point to consider in the text below is that cataracts may develop 6 to 12 months after the DNP treatment is completed.
The implementation of the treatment for obesity by dinitrophenol dates only from 1933, the year when it was suddenly and rapidly put in the limelight by the work of the Americans Tainter, Mehrtens and Cutting.

These authors have established that the ingestion of dinitrophenol accelerates metabolism, causing a marked elevation in temperature. It seemed that dinitrophenol was a specially effective treatment for obesity. In 1936, Horner estimated that in the first 15 months following the appearance of the medication in the market, one hundred thousand persons used it to lose weight. Note: 100,000 people used it.

Incidents and accidents multiplied and appeared sufficiently serious that the American Medical Association warned the public against the dangers of unsupervised treatment.

Here we discuss only the case of cataracts, which Horner had said that it occurs in one case in 1000 treatments. At the end of this report we will note the principle bibliographic references concerning the American literature devoted to the subject and which is of a great value, but we wish to emphasize how the European work and especially French are on the other hand still rare and even exceptional.

One can say that it is by the work of Onfray and Gilbert Dreyfus presented to the Congress of the S.F.O. [Societe Francaisedes Oculistes?] in 1937 that French opthamologists had their attention drawn to the subject. This remarkably precise work is enriched by two observations of which one is due to Doctor A. Gallois, of Besancon. We frequently reference this, for it contains in addition to minutely observed details, important physio?pathogenic considerations and a complete history of the subject.

Apart from this work, we should also to point out the observations of Van de Hoeve and Polak?Daniels published in Holland in 1936, as well as the French summaries and reviews of Halbron on cataracts and of Laignel?Lavastine on dinitrophenol intoxication.

Finally, we emphasize the interest of the work of Vogt on the cataracts caused by dinitrophenol in Switzerland and of G.Ciotola of those caused by alpha dinitrophenol in Italy, both published in 1937. The same year, Stein and Crevecoeur pointed out that in their opinion this affectation was, when all is said and done, quite rare if one thinks of the enormous dissemination of dinitro treatment. This was also the opinion of Andre Mayer, based on the fact that despite the considerable number of intoxications by dintrophenol observed in munitions factories, no cases of cataracts have been noted. Note: Because they were not intoxicated with it continuously.

Finally, in 1938, Carlotti and Rivoire de Nice presented a case of cataract by dintrophenyl?lysidine which developed "with almost lightning?like rapidity."

It was possible for us to observe two very demonstrative cases. In one there was...
an arrest of development of opacity after the patient stopped taking
dinitrophenol, which is more than a rarity, a real exception in the pathological
history of dinitrophenol cataracts.

OBSERVATION I.—Mme. K... Lea, 32 years old presented herself to me in
December 1937 with a marked lowering of the vision of both eyes, which began
a few weeks earlier, developing extremely fast and was all the more disturbing
since she works at a very visual profession in the editing of a newspaper and as
she is especially partial to this pleasant and remunerative
position. I noticed a beginning of bilateral cataract appearing striated and fleecy
which is found almost constantly in the description of toxic lens opacities of this
kind. The opacity is situated mainly at the level of the equator of the lens, but
also involves the posterior part of the central mass. The vision is only 4/10 in
the right and 5/10 in the left, these two acuities correctable to 7/10 O.D.G. ?? 2.50.

Mme K... thus learned that she was rapidly becoming myopic.

The most minute research were done in view of identifying a possible cause of
this bilateral cataract. All the blood and urine tests were negative. Very complete
clinical examinations by Doctor P..., referring physician, point to the same
conclusion that it is impossible to relieve Mmme. K...’s pathological process at
all.

It is then that I thought of asking her about the possibility of a dinitrophenol
anti?obesity treatment, even though the corpulence of my client did not seem
excessive. She told me then of having taken two pills each day of 0.30 grams of
dinitrophenol in series of ten days with a rest of 15 days, for the past year and a
half.
Note: That is a long time!

She had, without the least dietary restriction, lost 19 kilograms out of 87 [42
pounds out of 191]. It was at that point that she began complaining about her
vision.
Note: She lost a lot of weight, too!

I wasn’t aware of the topic at that time except by the short summaries of
American works, but I didn’t hesitate to warn her against what I considered to
be the real origin of her sickness. Very anxious about her state, she was easily
convinced and stopped that therapy suddenly and definitively.

I had the opportunity to see her in March, July and October 1938 and I noticed
with great interest the complete arrest in the development of these cataracts,
which accompanied in very precise fashion the progressive and total
disappearance of myopia to the extent that although it was possible to note an
appreciable modification in the lens opacities, the visual acuity was
spontaneously returned to 7/10 (uncorrected) at the end of October 1938.

We add that Mme. K..., doubly happy, very far from regaining weight in spite of
the renunciation of dinitrophenol, had lost another 5 kilos by a very strict
nutritional discipline complemented with rigorous gymnastic practices and the
introduction into her life of a new intoxicification, certainly less dangerous than
the preceeding—tea.
In this case, the role played by the toxin in the opacification of the lens seems to us demonstrated in an almost experimental fashion by the disappearance of the myopia at the moment of the cessation of the intoxification and even more by the incontestable and enduring stabilization of the state of opacities that maintained itself for six months. In contrast, the development was very sudden in a month before the application of this measure. It is presumed that only the precocity of the requested medical consultation and of the medical diagnostic given, has permitted a stop in the development of this toxic cataract—a completely unusual phenomenon.

We emphasize that the treatment had included plainly excessive doses and that however the opacification only appeared late in the treatment. On this topic remember that in the discussion which followed the expose made to the S.F.O. in 1937 by MM.Onfray and Gilbert Dreyfus?Arruga, who had occasion to observe and operate in America [illegible] ... don’t generally appear except at the end of many months and even sometimes six to twelve months after the cessation of treatment. These late?developing cataracts are almost always bilateral.

OBSERVATION II.  

[Not included. Summary: A 32 year old woman weighing 90 kg. (198 pounds) began taking dinitrophenol on February 1st, 1937. She began with 9 to 10 pills daily, each being 30 mg. of DNP. After a week she increased the dose to 12 pills / day (360 mg.). At this dosage she lost 800 grams per week, or 10 kg. (22 pounds) in three months, without changing her diet. She stopped taking DNP for four months and then began again. So she took 32.4 grams of DNP in the first 90 days and the same amount in the second course. American reports indicated that cataracts had resulted from doses as small as 100 mg. per day for a total of 40 grams.  

On June 10th 1938, after several days in a very sunny seaside resort, the patient began to lose vision in her left eye, and on July 12th, the other eye was affected. By August 1st she was unable to see to drive. By September she was blind. Fortunately, surgery produced favorable results.]  

It is necessary, indeed, to publicize cases in order to attract the attention of physicians and of the French public to the danger of intoxification by dinitrophenol. The fact that we have been able to stabilize, if not make regress one cataract of this class by stopping all toxic ingestion is but another reason which compels us to make it known.  

These arguments and our observations are so needed to challenge the imagination and influence young women against harmful weight loss techniques that the work appears discouraging.  

Indeed, in ending, we repeat the unlikely remark that our second patient made to us upon taking leave following the success of her first operation: "And now, Doctor, do not oppose my taking of dinitrophenol since I no longer risk having cataracts."
For me as a newbie... when I start I’m going to start by taking orals only.. its not just because I’m afraid of a needle.. its that I’m afraid to give myself the needle. I’m a pretty clumsy guy haha and I mean I’m going to do alot of research before I pin myself. I have no idea how far to put it in or even where for that matter. I can’t see myself being prepared to do this for awhile until I research everyhting I possibly can on the subject.

If you look up a few posts, check out the website about spot injections. It shows you exactly where to inject. The easiest spot in the hip/buttocks area, there are hardly any nerves or veins in that spot. It is a very large area , and is so wasy t do. Injectables are so much easier on the body and usually will give you longer lasting and better results.

Yea I’ve looked at a few injection sites and its looking alot easier then i though it was

Hey guys...this is my first ever post...so be gentle...im from sydney australia...does anybody have any before and after picks after their first cycle???

I pinned myself for the first time tonight with decca and im interested to see results some of you may have had...ive been training for 4 years and went from a very fat 97 kilo blubber boy to 70 kg of skinny nothing...i have put on 7 kgs over the last 3 years without increasing body fat but have stopped gaining over the last year despite changing supplements, training programs etc.

Cheers from down under!

We work on the English system of measure here, not metric. Can you please translate your weight?
we work on the english system of measure here, not metric.
Can you please translate your weight?

97kg = 213lbs
70kg = 154lbs
77kg = 170lbs

monster calculator ignorance/laziness

Liked your other one better. Had more info.

It is easy.

Yea i might give it a try soon i think

how long does it usually take Test. enanthate to start "kicking in", i began my cycle a little over 2 weeks ago and haven’t experienced any improvements yet, neither in strength nor gains

for the record, this is my first cycle

thanx
how long does it usually take Test.enanthate to start "kicking in", i began my cycle a little over 2 weeks ago and haven't experienced any improvements yet, neither in strength nor gains

for the record, this is my first cycle

thanx

After about 3 weeks

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: delta9mda on December 14, 2006, 02:08:33 PM

Quote from: durbax on November 06, 2006, 02:02:22 PM

For me as a newbie... when I start I'm going to start by taking orals only... its not just because I'm afraid of a needle.. its that I'm afraid to give myself the needle. I'm a pretty clumsy guy haha and I mean I'm going to do alot of research before I pin myself. I have no idea how far to put it in or even where for that matter. I can't see myself being prepared to do this for awhile until I research everything I possibly can on the subject.

stop being a pussy, oral only cycle will not do you that great. injecting is easy and pretty much painless. once you pass thru the skin you wont feel a thing.

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Arnold jr on December 17, 2006, 07:28:21 PM

Quote from: kksmoke on December 17, 2006, 06:40:47 PM

i tried the example cycle provided above and it turned me into a female. I hate you Arnold Jr.

Let me ask you a question. How does it feel to be a pure unadulterated "pussy" in every sense of the word?

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: kksmoke on December 21, 2006, 05:38:56 AM

i dont know , but i’ll ask Rax this night:

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: getbigquick on May 30, 2007, 05:05:21 PM

This is my first time on here, so please be gentle. I dont consider myself to be a hard gainer but recently I feel like I have plateaued. I am considering an anabolic agent like D-Bol. My question is whether or not there is a difference between Dianabol and D-BOL. I know one is legal and the other isn't. Thanks for help.

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Arnold jr on May 30, 2007, 05:35:15 PM

Quote from: getbigquick on May 30, 2007, 05:05:21 PM

This is my first time on here, so please be gentle. I dont consider myself to be a hard gainer but recently I feel like I have plateaued. I am considering an anabolic agent like D-Bol. My question is whether or not there is a difference between Dianabol and D-BOL. I know one is legal and the other isn’t. Thanks for help.
They are the same thing...dbol is just slang.


---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** Overload on May 31, 2007, 05:07:41 AM

> This is my first time on here, so please be gentle. I dont consider myself to be a hard gainer but recently I feel like I have plateaued. I am considering an anabolic agent like D-Bol. My question is whether or not there is a difference between Dianabol and D-BOL. I know one is legal and the other isn’t. Thanks for help.

The D-BOL you see for sale in magazines is pure crap...

Get real Dianabol.

8)

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** Rimbaud on July 26, 2007, 04:19:49 PM

A little help for those with questions about injections:


---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** jakecody on August 08, 2007, 05:36:29 AM

What about using HCG during cycle? I read it stops the boys from shrinking.

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** trab on August 08, 2007, 11:58:01 AM

> What about using HCG during cycle? I read it stops the boys from shrinking.

Its pretty personal, I prefer to run to superssion then do something. To each his own.

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** flyguy27 on August 10, 2007, 05:17:51 PM

Im bout to do my first cycle, and this is some great info. First off, i love this blog sight. Most everyone on here are very helpful. Now, you say to do your first cycle for 10 weeks. I thought you were supposed to keep it at a 6-8 week cycle??

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** Arnold jr on August 10, 2007, 05:32:02 PM

> Quote from: flyguy27 on August 10, 2007, 05:17:51 PM
Im bout to do my first cycle, and this is some great info. First off, I love this blog sight. Most everyone on here are very helpful. Now, you say to do your first cycle for 10 weeks. I thought you were supposed to keep it at a 6-8 week cycle?

There is no magic number when it comes to how long you run a cycle.

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Rimbaud on August 11, 2007, 06:56:30 AM

> There is no magic number when it comes to how long you run a cycle.

Agreed.

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: slim on October 26, 2007, 05:41:55 AM

Great post nice and easy to understand, you guys helped me back in the summer when i was struggling doing a coarse of just deca and dianabol, got deca dick etc got some great advice on pct. that was only my second cycle so i was thinking of going back to basics and doing your beginners routine, just a couple of basic questions.

is test ethenate much different from sustanon (guy at gym sells sus 250).
during 10 weeks on test you dont take anything else (unless you get symptoms of gyno).
then start pct 2 weeks after last hit of test.
no need for HCG at end of cycle just nolv and clomid
is it better to rotate injections ie week 1 gluts week 2 delts to keep receptors fresh or would 10 weeks injecting in your gluts not make any difference.

thanks Rich

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: trab on October 26, 2007, 06:33:09 AM

> Great post nice and easy to understand, you guys helped me back in the summer when i was struggling doing a coarse of just deca and dianabol, got deca dick etc got some great advice on pct. that was only my second cycle so i was thinking of going back to basics and doing your beginners routine, just a couple of basic questions.

is test ethenate much different from sustanon (guy at gym sells sus 250).
during 10 weeks on test you dont take anything else (unless you get symptoms of gyno).
then start pct 2 weeks after last hit of test.
no need for HCG at end of cycle just nolv and clomid
is it better to rotate injections ie week 1 gluts week 2 delts to keep receptors fresh or would 10 weeks injecting in your gluts not make any difference.

thanks Rich

I wouldn't take any "Sus" floating around the USA out of a 10cc vial.
1cc amps only.Karachi/Paki/Greek or Dutch Organon if its still out there.
There also several Mid east countries make real SUs. Single amps with paper lables is the rule.
The packing quality tells you its real.
Avoid UG gear period IMO.
I personally take some HCG or Clomid 5 days after last shot or TE. If it was real SUSTanon (unlikely in a 10cc) 17th day. (Crash is about day 21 after a sus shot, longacting decanoate ester)

I ALWAYS ROTATE SHOT SITES. Forget thinking about receptors.... You'll be better off.

10 weeks in glutes is too much. The med books show dividing up glutes into a grid pattern for such shot course, but it mainly for small ppl w/ not enough delt and leg size for lots of shots.

I use delts, legs, shoulders, glutes, hip. Triceps now and then.

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** slim on October 28, 2007, 12:06:12 PM

Hi Trab hope your keeping well, thanks for all your help in the summer. can you do me a favour please and outline your pct coarse FULLY inc timings and drugs, 1- if i use sus 250x2 per week for 10 weeks (the guy at gym geets it from greece). 2- if i use test eth 250x2 per week for 10 weeks.

Your help is much appreciated Rich

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** trab on October 28, 2007, 01:56:46 PM

I never use by a "recipe". I go by how I feel and what I got/ can get/ price.

The thing with Real 4 ester Sus is to avoid the crash at about the 21 day point after last shot. I crash like clockwork on 21 unless I've built up a Hell of a level of the stuff, then a bit later.

TE or TC? I'm hittin' some HCG or clomid 5 days after last shot. I taper down the last shots, many guy dont like that.

Sometime I use HCG at end, sometime not. If so, I'm leaning to smaller 500iu doses these days myself. (Bigger shots if midcycle use of HCG for me 1000iu). 20mg nolva w/ the HCG allways for me.

If I use HCG at end, I'll like some clomid after that. 100mg ed for 10-14 days. Nothing wrong with 20mg ed nolva after them clomid is gone.

ALL that said, I just came off with nothing but taper down the test to 200mg ew. I Feel ok in 6 weeks.
Hi all

New to the site. Thanks in advance for the info. I am 37, 5ft 8inches, 184 and a six pack showing, estimated 10-12% bf. Diet is pretty strict with 2500 calories, 200 g of protein and 300-350 g of carbs all over 5-6 meals and been training and lifting hard for 8 years. Ready to go to the next level. I am going to go with the recommendations listed in this posting for a first cycle. The 10 week cycle of test e and d-bol. My question is on the d-bol you mention 4 weeks. Would this be for the first 4 weeks? thanks

yes

Yup, esp for 1st timers.
Dbol very supressive.

Here, I'll sum up my personal AAS theory in a nutshell.

ANdrogenic compounds first, leading to anabolic ones and tapper down.
Learn what drugs are more androgenic, and how long they last in the body and you can work with anything you can find.
Dianabol - methandrostenolone: the oral anabolic steroid, originally developed in 1950’s to counter malnutrition in post-war children, etc...

D-bol - is your’s trully ;)

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** slim on December 03, 2007, 05:34:38 AM

Hi Chaps has anyone tried test 300 guy at gym has got some ive read up on it just wondered if anyone had tried it or heard reports on it.

thanks

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** gym-junky on January 04, 2008, 04:11:54 PM

Hi again

I am getting close to getting the gear I need to get started. My question is this. Once I start the cycle should I discontinue creatine and the amino’s that I supplement with?

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** Emmortal on January 04, 2008, 04:17:47 PM

You don’t have to stop using creatine and aminos, but I usually stop most of that stuff while on and save it for after, it’s really just personal preference.

---

**Title:** Re: Thinking about doing steroids?? READ THIS!! Newbie Info  
**Post by:** Wahawk on January 22, 2008, 05:18:49 PM

Great article, the whole string is helping me out a ton. This will be my first cycle, I am doing Testosterone Enanthate and Nandralone Decanoate stack wich you said you recomend. I think I have everything I need except per your recomendations I am short on the post cycle drugs. So I am sure some if this is redundant or elementarty but hoping you guys could be gentle and help me out. This is my first post.

1st) I thought I could break the shots into two per week overall. Like Sunday stack 250mg of Deca and 250mg of Test and then wednesday do 250mgs of just the test. Is that not true?

2nd) Is post cycle clowmid and or Nolvadex easy to get? Is it over the counter?

3rd) With the cycle I am doing (10 weeks) Deca / Test is gyno common? I will make sure I am prepared just wondering what the likely hood of this is?

Thanks Guys
1. This is fine.

2. It's easy to get but not OTC.

3. It's a 50/50 thing...some guys get it some don’t. If you use over a long period over and over though, odds are at some point you’ll get something.

Follow up question. All my gear arrived accept the dbol. I should have it in about 10 days. Is it OK to start the test e and then the dbol in about 10 days?

I would personally wait until you have everything but that’s just me. That being said being without dbol will not lead to a bad cycle.

from what i understand you use the dbol to kickstart the cycle...and in ten days time your test should already be going to work.

Playing off that, I just started my cycle. Dbol and Test E on Monday, about to do just the test E today.

I am being impatient, just wondering how long into a cycle you start to see / feel results?
Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**
Post by: **Arnold jr** on **February 07, 2008, 09:33:54 PM**

Quote from: Wahawk on February 07, 2008, 03:30:36 PM
Playing off that, I just started my cycle. Dbol and Test E on Monday, about to do just the test E today.
I am being impatient, just wondering how long into a cycle you start to see / feel results?

You should notice the dbol by now IMO.

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**
Post by: **Wahawk** on **February 20, 2008, 03:35:07 PM**

Can I get some feedback guys, I have a raised welt in my glute where from the second time I used that injection site. It actually kind of itches as well. This happened to anyone?

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**
Post by: **Emmortal** on **February 20, 2008, 03:38:14 PM**

Is it warm to the touch?

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**
Post by: **Arnold jr** on **February 20, 2008, 05:04:33 PM**

Quote from: Wahawk on February 20, 2008, 03:35:07 PM
Can I get some feedback guys, I have a raised welt in my glute where from the second time I used that injection site. It actually kind of itches as well. This happened to anyone?

If it's hot to the touch and you're running a fever, go to the doctor. If not, then it's just one of those things you have to deal with from time to time.

If it's slightly warm with the welt, don't freak out...when a spot on the body is irritated it's going to feel warmer.

Anyway, little knots and irritations are just part of the whole AAS thing.

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**
Post by: **Wahawk** on **March 06, 2008, 09:00:14 AM**

On week 5 and just tore a pectoral muscle... what do I do now?

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**
Post by: **Beener** on **March 06, 2008, 09:31:31 AM**

Quote from: Wahawk on March 06, 2008, 09:00:14 AM
On week 5 and just tore a pectoral muscle... what do I do now?

Go to a fuckin doctor???
Quote from: Wahawk on March 06, 2008, 09:00:14 AM

On week 5 and just tore a pectoral muscle... what do I do now?

Are you asking if you should stay on cycle or not?

Are you sure it's torn and not just strained or pulled? A strain or pull in the pec can really hurt but it's not a tear. Have you actually had it looked at?

I've stayed on during an injury before...I was probably about 8wks or so into a cycle when I twisted a vertebra in my back. After that training was limited but I did everything I could still do...no squats no deads but most other things were fine as long as I didn’t jerk around...in the end it made my form better on a lot of things cause I had to make sure I stayed really tight. Anyway, I was back to full steam in 12wks and this was after they told me it would be 6months...doc didn't know what to think or how or why it happened...could it have been the gear, lol?

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info

Yeah, I've been to a doctor, I went straight to the fucking ER.

And yes, I am asking if I should stay on and was looking for feedback as far as that goes.

Anyway, partially torn. I think I’m going to stay on, I can't do anything with a press right now, it’s not even an option. Sucks...

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Mega Man on March 22, 2008, 09:13:18 PM

For a first timer with great genetics and is 24 years old.....what would the difference in results be between 250mg of test e per week.....and 500mgs' per week?

I don't won’t to gain a huge amount, but do want to notice somethign. That's why Im thinking about just 250mg’s per week?

and would it be better to do 250 for a longer duration.......or 500 for a shorter duration?

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Arnold jr on March 22, 2008, 10:07:26 PM

Quote from: Mega Man on March 22, 2008, 09:13:18 PM

For a first timer with great genetics and is 24 years old.....what would the difference in results be between 250mg of test e per week.....and 500mgs' per week?

I don't won’t to gain a huge amount, but do want to notice somethign. That's why Im thinking about just 250mg’s per week?

and would it be better to do 250 for a longer duration.......or 500 for a shorter duration?

500mg for a longer duration
LoL....you combined both of my questions.....and both of my train of thoughts together ;D

But I appreciate you advice and I will take it....I will take 500 mg’s of test per week for a longer duration...10-12 weeks

Qestion though....is depression after a cycle like this immenent....or just depends on individual an pct that is ran???

I’m pretty strong minded, and mentaly tough....just curious!

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Emmortal on March 22, 2008, 10:59:49 PM

It can be individual dependant, some guys hit rock bottom others don’t have any problems with it. Clomid can really send guys off into oblivion while it doesn’t bother others. The only way to know how you react is to go through it and find out.

500mgs for a 12 week cycle would be just about perfect. Twice a week injects Mon/Thursday 2 hours before working on those days and you’ll be golden. Just be prepared to eat a shit load of food, and I mean a shit load. You should blow up nicely.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Mega Man on March 22, 2008, 11:10:01 PM

Would it be okay to add human grade winny tabs at the end of the cycle ,If I can find some?

On another thread, they said for a first cycle, just stick to test. Why would it be a bad Idea?

Another question.....How does 500mg of test for a first timer make you feel mentaly and emoitionaly?

I've read profiles and stickies from various sites....but it’s not consistint? some say it make you aggressive and others say it makes you feel a sense of greater well being???:;D

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info

Quote from: Mega Man on March 22, 2008, 11:10:01 PM
Would it be okay to add human grade winny tabs at the end of the cycle ,If I can find some?

On another thread, they said for a first cycle, just stick to test. Why would it be a bad Idea?
Another question.....How does 500mg of test for a first timer make you feel mentally and emotionally?

I've read profiles and stickies from various sites....but it's not consistent? some say it make you aggressive and others say it makes you feel a sense of greater well being?? ;D

Adding winny would be fine.

You should have a greater sense of wellbeing, that should be a given. As for aggression, if you feel better it naturally makes you more aggressive...it’s how you channel this aggression that makes it a good or bad thing. Aggression is often labeled as a negative thing, but that’s only when it’s used in a negative manner.

---

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: **Mega Man** on **March 23, 2008, 07:28:22 PM**

If I do 500mg’s test e per week, should I take it just one shot a week.....or split it up in two shots?

If I split it up.....can I just alternate each glute every time....or would I need another injection site?

And would it kick in quicker if I did 1 shot of sustanon 250 on day 1....and 1 shot of test e on day 4 every week?

---

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: **Arnold jr** on **March 23, 2008, 08:57:10 PM**

Quote from: Mega Man on March 23, 2008, 07:28:22 PM

If I do 500mg’s test e per week, should I take it just one shot a week.....or split it up in two shots?

If I split it up.....can I just alternate each glute every time....or would I need another injection site?

And would it kick in quicker if I did 1 shot of sustanon 250 on day 1....and 1 shot of test e on day 4 every week?

Split it up into 2 injections 250mg each.

Yes, you can alternate each glute and you’ll be fine with this on this cycle.

I’d just stick with the test-e

---

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: **Mega Man** on **March 24, 2008, 02:26:57 AM**

For a cycle of just test e twice weekly....how likely is getting acne on my face?

I don’t care about acne on back or the rest of my body, I just don’t want it on my face?

---

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: **Arnold jr** on **March 24, 2008, 10:39:15 AM**

Quote from: Mega Man on March 24, 2008, 02:26:57 AM

For a cycle of just test e twice weekly....how likely is getting acne on my face?
I don't care about acne on back or the rest of my body, I just don't want it on my face?

Imposable to answer...it affects everyone differently...just make sure you keep yourself clean and wash your face multiple times daily.

Personally so far I’ve never had acne problems anywhere on my body with the exception of clomid use...I usually get a break out on my back from clomid, but it's mild and short lived so I just deal with it since I like clomid.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Mega Man on March 24, 2008, 10:54:41 AM

Thanks, I guess I'll just have to wait and see!

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Emmortal on March 24, 2008, 11:47:41 AM

I only get a few random zits on my back, maybe 4-5 little ones spread out. When I’m on deca I get it on my shoulders a little bit, sometimes on my legs or arms too but it’s never bad, just 1-2 random ones around.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Mega Man on March 24, 2008, 03:25:27 PM

Quote from: Emmortal on March 24, 2008, 11:47:41 AM
I only get a few random zits on my back, maybe 4-5 little ones spread out. When I’m on deca I get it on my shoulders a little bit, sometimes on my legs or arms too but it’s never bad, just 1-2 random ones around.

Do you ever get them on your face?

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: gym-junky on April 03, 2008, 04:54:34 PM

Hi all

Wanted to give an update and ask a couple questions. Just started the 6th week of the 10 week newbie cycle (250mg test e twice a week). Size is going up and have put on 14 pounds in 6 weeks. I have a new hardness and fullness all around, all weights in lifting are up. Over all very happy with the results so far. I have also lost a bit of definition and would like to ask if I should cut back on calories or carbs and up protein? Or should I continue as is until the cycle is over? Currently at 204 and taking in 200-225g pro, 300 carbs and 2500-3000 calories all spread over 6-7 meals. Last question is this. I am feeling a bit over trained and am wondering if I should take a week off while I am in the cycle.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Arnold jr on April 03, 2008, 05:26:21 PM

Quote from: gym-junky on April 03, 2008, 04:54:34 PM
Hi all

Wanted to give an update and ask a couple questions. Just started the 6th week of the 10 week newbie cycle (250mg test e twice a week). Size is going up and have put on 14 pounds in 6 weeks. I have a new hardness and fullness all around, all weights in lifting are up. Over all very happy with the results so far. I have also
lost a bit of definition and would like to ask if I should cut back on calories or carbs and up protein? Or should I continue as is until the cycle is over? Currently at 204 and taking in 200-225g pro, 300 carbs and 2500-3000 calories all spread over 6-7 meals. Last question is this. I am feeling a bit over trained and am wondering if I should take a week off while I am in the cycle.

First off, if you are truly over trained then yes, take a wk or so off.

As far as you having lost some of your conditioning, if you’re trying to grow, which it sounds like you are, this is just part of what happens...growth does equal some bodyfat...hopefully minimal. If you want to drop BF, drop the calories and do more cardio...that’s about all there is to say about that.

---

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: gym-junky on April 04, 2008, 01:04:21 PM

Ok thanks for the info.

---

Title: **Re: AAS Studies/Links/Literature**  
Post by: candidizzle on April 17, 2008, 04:51:30 PM

taken from

http://www.ironmagazineforums.com/training/60580-iron-mans-anabolic-cycles.html

---

PROFESSIONAL BODYBUILDING CYCLES

Vast sums of bodybuilders who use the "average" steroid cycle wonder what it is that separates them from those bodybuilders who are reaching "Abnormal Size." In my experience it takes much more than, genetics, the perfect training routine or diet to brings forth these kind of gains. Professional bodybuilding is somewhat of a "Drug Contest." No one continues to build muscle mass in a linear manner because muscular weight gains does not occur that way. Muscular growth comes in spurts. You can train hard, eat the proper nutrition and still make slow progress after the body gets use to the steroid dosages you have been taking. Luckily for those wanting to move on to the next level of development the body is capable of gaining muscle mass at a considerable rate. (This type of protocol is for advanced bodybuilders only). These cycles are for the purpose of achieving more permanent results through a highly potent synergistic effect. Advanced bodybuilders must saturate the androgen receptor site with high doses of drugs, eat massive amounts of calories-protein/amino acids, carbs, fats, lift heavier weights in good form, and get plenty of rest, if they want to progress to the level few seem to find.

BLITZING is a step forward to furthering ones progress when everything else is failing to produce or you just want to make faster gains. Blitzing is basically the same kind of cycling I recommended earlier with the short cycles but on a more aggressive scale.

I’ve witnessed bodybuilder’s on a "Professional Level" gain a 1/2 lb or more per day for the first 4-6 weeks of an 8 week cycle using what’s referred as a
"FRONT-LOADING" technique throughout the entire cycle. This is the "Big Secret" the Professionals don't want you to know about. They "Blitz" with large amounts of fast acting Anabolic/Androgenic steroids, Slin or IGF Long R-3, GH, and T-3.

The cruising period is customarily less than the blitzing period. They blitz for 8 weeks, then cruise for another 3 weeks or so, then blitzing once again, usually with a totally different arsenal of drugs. Some professional bodybuilders combine "fast" and "slow" acting esters to provide a synergistic effect in the course of a "Blitz Cycles". If side effects such as high blood pressure get out of control, they abandon the fast acting esters such as D-Bol, Drol, Suspension, or Prop. In doing so this puts them into a position of having to deal with only the longer acting esters according to when the fast acting esters leave their system.

When the "Blitz Cycle" is complete, the elite cruise with enough drugs to help maintain the mass they have achieved while allowing blood pressure to return to a normal position. Trenbolone is a commonly used drug during the cruising period due to its amazing anti-catabolic qualities and it rarely elevates blood pressure. Some Pro's have been known to use as much as 750 mgs to 1 gram of test along with a highly anabolic steroid such as Deca, Equipoise, or Winstrol. D-Bol can be another good choice for cruising as it puts up a strong fight against post cycle cortisol levels. Smaller amounts of anabolics are needed when GH is used. Either way you slice it they use MEGA doses.

Resorting back to near HRT levels, for e.g.; 300 mgs of testosterone/weekly, will not provide "Pro Bodybuilders" with sufficient plasma levels to sustain the muscle mass they have put on during a "Blitz Cycle". With that being said, 300 mgs of test can be adequate if united with other highly anabolic drugs such as nandrolones, PGF-2-"spot injecting lagging body parts", DHT derivatives, GH and or Slin, or stacks of GH/IGF Long R-3, etc, as the choices are endless.

NOTE: PGF-2, slin or IGF Long R-3, HCG, Clomid, and Nolvadex is a common stack used by Professionals wanting to regain HPTA REGENERATION.

Many opt to restore high dosages of androgens with GH, Slin, DHT derivatives. Lifters who are experiencing difficulty with high blood pressure tend to shun large dosages of "Testosterone" for extended periods. Anytime you use testosterone, it magnifies the sides of whatever else you are using with it. For e.g.; when Trenbolones are used in moderate dosages, it produces few overall side effects for many, but when test is added to the mix, blood pressure surges, night sweats appear, and mood swings are prominent. These kind of sides can be diminished in size by using small amounts of test Prop or Suspension in conjunction with IGF-Long R-3, GH, and higher dosages of DHT derivatives. Bodybuilders who do not get along well with various Testosterones, can use "Trenbolones" to produce an affirmative response, with a much lower dosage.

NOTE: Testosterones, Trenbolones, Orals, Insulin, PGF-2, Growth Hormones, Long R-3- Insulin-Like Growth Factors, Thyroid Hormones/T-3, Anti-Estrogens, are used in combination with one another to produce a "Massive Synergistic
Professional Bodybuilders retain their estrogens levels to a "minimum" during high dosed cycles. Excess estrogen develops at a rapid rate when aromatizing steroids are used with this type of regimen. With this comes great water retention, high blood pressure, mood swings, and female fatty tissue deposition. Estrogen manipulation can be of great use, but not during a high dosed "Blitz Cycle". Small amounts of T-3-(Cytomel) can be used to help increase "thyroid function" that decreases when using Growth Hormone. T-3 can also increase appetite for the extra calories need to support growth with this monumental approach. This drug also increases the rate of nutrient absorption-metabolism so it will be available for protein synthesis and aid in keeping fatty tissue deposits down for Endomorphs during this "Extreme Bulking" up period.

When bodybuilders approach the National Level, their gains take a relatively long time no matter how hard they juice. The passage that leads advanced bodybuilders from one place to another is "Blitz Cycles." I know by personal presence many trainers spend lots of money on large quantities of steroids, only to reach a plateau they are unable to conquer. The whole quantity of Testosterones, Trenbolones, Orals, Growth Hormone, Insulin, IGF-1, will only get you to a certain point. You have to work with a mode of action that will persuade the muscle to elevate protein synthesis.

Editors Note: There have always been a certain percentage of bodybuilders who, when faced with overwhelming facts about the amount of drugs needed to become a PROFESSIONAL, who continue to believe the lie it's all "GENETICS" and "DIET" because it's a lie that makes them most comfortable. Or maybe it's because this lie justifies their own actions. Such is a destiny of a bodybuilders with the GOD given talent to make it to the Summit but refuses to learn and grow from their past experiences. Below are some cycles being used to make substantial progress given the proper diet and training program are put to service.

**BLITZ CYCLES USED BY PROFESSIONALS**

**(8 WEEK-SAMPLE CYCLE 1)**

- Test Suspension: 250-500 mgs/ed
- Trenbolone Enanthate: 600 mgs/ed
- D-Bol: 50 mgs/ed
- IGF-1 Long R3: 90-120 mcgs/5 days on-2 days off
- GH: 9-12 ius/5 days on-2 days off
- Cytomel: 25-50 mcgs-AM/2 days on-2 days off/ "optional"

**(8 WEEK-SAMPLE CYCLE 2)**

- Test Enanthate: 300-600 mgs/ed day
Tren Acetate: 125 mgs/ed  
Deca Durabolin: 300 mgs/ed  
Equipoise: 400 mgs/eod  
D-bol: 75 mgs/ed/or Anadrol 150 mgs/ed  
Insulin: 20-40 i.u./5 days on-2 days off  
GH: 9-12 ius/5 days on-2 days off  
Cytomel: 25-50 mcgs-AM/2 days on-2 days off/"optional"  
Creatine: http://www.ironmaglabs.com/maximum-pump.html

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: gym-junky on **June 06, 2008, 05:50:22 PM**

Hi all

Thanks again for all the great info. I plan on doing another cycle starting in August and wanted to check and see what you all thought of using Sustanon 250 in place of test E for the cycle recommended here. Start with D-bol and 250 mg Sust 250 twice a week for 10 weeks? Also would it be ok to go 12-14 weeks with the Sust 250? Would the PCT be the same and should I take anything extra to keep estrogen levels down etc?

Title: **Re: Thinking about doing steroids?? READ THIS!! Newbie Info**  
Post by: Arnold jr on **June 06, 2008, 07:02:35 PM**

Quote from: gym-junky on June 06, 2008, 05:50:22 PM

Hi all

Thanks again for all the great info. I plan on doing another cycle starting in August and wanted to check and see what you all thought of using Sustanon 250 in place of test E for the cycle recommended here. Start with D-bol and 250 mg Sust 250 twice a week for 10 weeks? Also would it be ok to go 12-14 weeks with the Sust 250? Would the PCT be the same and should I take anything extra to keep estrogen levels down etc?

What you have planned will be fine.

Title: **test cyp**  
Post by: Wahawk on **August 14, 2008, 01:00:09 PM**

I can get a bottle of test cyp from a guy I know, but it expired in 2007 and looks a little cloudy. Can I get come good feedback on whether this is a good or bad idea?

Title: **Re: test cyp**  
Post by: 4thAD on **August 14, 2008, 01:53:36 PM**

Quote from: Wahawk on August 14, 2008, 01:00:09 PM

I can get a bottle of test cyp from a guy I know, but it expired in 2007 and looks a little cloudy. Can I get come good feedback on whether this is a good or bad idea?

I think you said it! Its cloudy, do you really want to take a chance with your health over using cloudy gear that none of could truly assess? I would think not.

Title: **Re: test cyp**  
Post by: Wahawk on **August 14, 2008, 04:22:48 PM**
Quote from: 4thAD on August 14, 2008, 01:53:36 PM
I think you said it! Its cloudy, do you really want to take a chance with your health over using cloudy gear that none of could truly assess? I would think not.

Yeah, you are right... just getting desperate. He's my only source.

search the net sources are everywhere.

Anyone heard anything about this product?

Testosterone Enanthate (Norma Hellas, Greece)

also wondering of any of you guys use ***** to get gear and what type of results you have gotten?

Just trying to get some feedback here, I'm doing research but just don't see much. If anyone has used the test E (norma hellas, Greece) product and had good results, knows about fakes, etc I would like to know. I'm posting under the newbie cycle page for a reason.

Norma is some of the best stuff there is dude...

8)

If it’s real, unfortunately they are faked more often than not.

Norma is some of the best stuff there is dude...

8)
Yes, and it is getting much worse.

8)

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Wahawk on October 06, 2008, 05:19:58 AM

I have a couple questions, if you have time I would love to get some expert feedback

As far as Winny, I was thinking of starting an 8 week cycle soon but I have seen some conflicting information.

1) I know that Winny is generally used as a cutting steroid and I am fine with that. But I have seen some profiles that recommend coupling with Test E if you wanted to see more gains in mass. Would this be effective?
   A) If so what dosage would you recommend with the stack?

2) I have also seen recommendations for 3cc’s a week instead of 50mg’s a day? This is what I would rather do but wondering what your take was.

3) I also saw Deca recommended as a stack for size, Deca has some great properties and I would like to use it just for the ease in joint pain and what not but would I increase my chances of sexual side effects? IE decrease sex drive? Deca dick...

4) If both deca and test would be good for increased gains which is better?

I definitely want the cutting properties and getting lean is pretty important to me, but if I could couple that with some mass building obviously that would be ideal, just wondering if you guys had any thoughts to help me out.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Arnold jr on October 06, 2008, 10:21:41 PM

Quote from: Wahawk on October 06, 2008, 05:19:58 AM

I have a couple questions, if you have time I would love to get some expert feedback

As far as Winny, I was thinking of starting an 8 week cycle soon but I have seen some conflicting information.

1) I know that Winny is generally used as a cutting steroid and I am fine with that. But I have seen some profiles that recommend coupling with Test E if you wanted to see more gains in mass. Would this be effective?
   A) If so what dosage would you recommend with the stack?

2) I have also seen recommendations for 3cc’s a week instead of 50mg’s a day? This is what I would rather do but wondering what your take was.

3) I also saw Deca recommended as a stack for size, Deca has some great properties and I would like to use it just for the ease in joint pain and what not but would I increase my chances of sexual side effects? IE decrease sex drive? Deca dick...
4) If both deca and test would be good for increased gains which is better?

I definitely want the cutting properties and getting lean is pretty important to me, but if I could couple that with some mass building obviously that would be ideal, just wondering if you guys had any thoughts to help me out.

You need to pick one, either getting leaner or bulking up more...steroids do not posses the magic ability to give you massive amounts of both...don’t believe the hype.

Certain steroids are often used in cutting cycle rather then bulking cycles, but there are no technical "cutting" steroids. You can get lean on just about anything.

Test and deca are both solid drugs, but they are not similar or nor does one replace the other. If you pick just one, go with test, much safer and effective for what you’re wanting. Deca alone can often give you the side effects you're concerned with...test alone cannot. Now test with deca is a good stack.

Winny, can be used bulking or cutting effectively. Now by saying you want to run 3cc’s a wk, well this doesn’t clarify an amount. Most injectable winny is dosed at 50mg/ml so in most cases 3cc would equal 150mg. But some winny is dosed higher so you need to know the dosage of the winny you have.

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: Emmortal on October 06, 2008, 10:37:52 PM

Quote from: Arnold jr on October 06, 2008, 10:21:41 PM

Winny, can be used bulking or cutting effectively. Now by saying you want to run 3cc’s a wk, well this doesn’t clarify an amount. Most injectable winny is dosed at 50mg/ml so in most cases 3cc would equal 150mg. But some winny is dosed higher so you need to know the dosage of the winny you have.

Wahawk, one thing I’d add about using Winny in a bulk is that it tends to dry your joints out for a lot of guys which typically means bye bye to heavy lifting. I’m not even a fan of using it in a cut either, but that’s just me personally. You really can’t know how it will effect you until you run it, everyone reacts differently to compounds. But just keep that in mind for future reference.

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: the Chance on October 14, 2008, 03:29:06 PM

im thinking about trying a cycle. I know alittle about them but would like to know alittle more..iv done them before but not very long**************************************************************************EDIT**************************************************************************

---

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: dirt on October 18, 2008, 11:26:09 AM

ok ive done all the reading and im sure i would like to try a newbie cycle im 39 5’9 175 cut north of chicago but not making any gains anymore at least very little can someone give me some advise on how to get started
Awesome info, I am very grateful for this board and this post!

Guys,
Is winny supposed to be a milky color? the brand is British Dragon.

Thanks as always.

Sorry, forgot.
I don’t have any experience with the little 50ml bullet looking capsules. How do you go about getting the gear into a syringe with those?

Thanks

Yes

What?

Thanks Arnold Jr,
They are the amps, thats what I meant. I guess you bust the cap off and put it into the syringe. Never mind.

So this may sound stupid to some people but I just recently started thinking of steroids and im set on starting a cycle of Anavar(Oxandrolone) and most people suggest between 20-100mg/day for it to really work to its full potential...so if i
wanna take about 60-80mg/day and they come in 10mg tabs would that mean that I would need to take 8 tabs at the same time to get my 80mg/day or do i spread it out equally through out the day

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: chris-a on November 14, 2008, 01:06:04 PM

80mgs a day is good. spread the dose throughout the day, preferably with meals. don’t forget, tho mild, anavar can still affect your natural test - have your pct pre-planned. not otc either...

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: 4thAD on November 18, 2008, 11:30:46 AM

Quote from: Wahawk on October 25, 2008, 01:30:42 AM

Thanks Arnold Jr,
They are the amps, thats what I meant. I guess you bust the cap off and put it into the syringe. Never mind.

You break the top off of the amp then draw the gear out with the syringe.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: 4thAD on November 27, 2008, 08:43:01 AM

Always run a PCT. You will get shut down even from var. Maybe not as much, but it still shuts you down. Provirion will shut you down to an extent.

Title: Re: Thinking about doing steroids?? READ THIS!! Newbie Info
Post by: BatistaSL on December 12, 2008, 06:20:26 PM

Good info..thanks AJ and all..

Title: Re: AAS Studies/Links/Literature
Post by: 4thAD on January 14, 2009, 01:56:49 PM

Interesting study on the bioavailability of HCG with IM and SubQ injections.

http://humrep.oxfordjournals.org/cgi/content/abstract/18/11/2294

Title: Re: AAS Studies/Links/Literature
Post by: Emmortal on January 30, 2009, 11:50:06 AM

Good read, grabbed from another board:

The Pharmacology Of Anabolic Steroids

Written by By Dan Gwartney, MD
Wednesday, 28 January 2009

Anabolic-androgenic steroids (AAS) are immensely popular with athletes and individuals interested in building muscle mass because they are reliable and effective.1 Much like any other drug, licit or illicit, patients or users can easily
Pharmacology is the study of the factors involved with how a drug enters and works in the body, the various ways the cells and systems regulate the processes affected and how the body clears the drug. Despite the fact that AAS are based upon an endogenous hormone (testosterone) and have been utilized as a pharmaceutical product for over 50 years, the complete pharmacology of AAS remains undetermined, even though a significant number of men suffer from symptoms of androgen deficiency, with associated health consequences including cardiovascular disease and earlier mortality.2-4 Fortunately, there is a substantial body of published research in the field, nicely reviewed by Dr. Andrew Kicman of the Department of Forensic Science and Drug Monitoring at King’s College London (England) in the British Journal of Pharmacology.5 While the collective knowledge in the field of AAS is incomplete (as it is with every other drug, such being the nature of science), it is sufficient to develop a working understanding of AAS and suggest potential research to improve safety and efficacy.

Kicman’s review is organized in excellent fashion and comprehensively referenced. Those truly interested in learning about AAS, which should include anyone using or prescribing such drugs (licitly or illicitly), would be well-served to read the review. The British Journal of Pharmacology has provided free access to the article at their website: http://www.nature.com/bjp/journal/v154/n3/...bjp2008165a.pdf.

As has been stated repeatedly, AAS contain or are derivatives of the natural male sex hormone, testosterone. Knowing this, it is not surprising that AAS affect a myriad of tissues which respond to testosterone, including: reproductive tissues, muscle, bone, hair follicles, liver, kidneys, brain, white and red blood cells. These effects are usually divided into androgenic (referring to masculinization) and anabolic (primarily protein building in muscle and bone). As the effects of AAS on fetal development, pre-adolescents and females add greatly to the complexity of the topic, this article is restricted to the effects of AAS on post-pubescent males.

Skeletal Muscle, Testosterone And Hypertrophy
The effects of AAS on specific tissue-types (fat cells versus muscle versus brain, etc.) depend in part on how that tissue enzymatically processes androgens and whether a particular AAS is protected against such enzyme “attacks.”6 In tissue related to the reproductive system, testosterone acts as a prohormone, being converted to the more androgenic DHT by the enzyme 5-alpha-reductase, as happens in the prostate; similarly, testosterone is converted to estradiol (an estrogen) in breast tissue and fat cells by the enzyme aromatase. Many tissues convert testosterone into both DHT and estradiol (i.e., brain, bone).

However, the primary tissue of interest, relative to AAS use for physique or performance improvement, is skeletal muscle. Skeletal muscle includes the
familiar biceps, pecs, quads and other muscles that aid in movement. Two other classes of muscle include smooth muscle, which is present in the gut and blood vessels, the second being cardiac (heart) muscle. AAS may affect these other two types, particularly the heart, but these effects are outside the scope of this article. Skeletal muscle does not contain detectable levels of 5a-reductase, but does have significant aromatase activity. Thus, testosterone is the primary androgen responsible for muscle hypertrophy; the role of estrogen in skeletal muscle remains unknown at this time.

Testosterone affects the muscle cell by activating genomic and non-genomic targets. The genomic effects of testosterone involve the classically understood mechanism of testosterone entering the cytoplasm (the inside of the cell), binding with an androgen receptor (AR) and traveling together into the nucleus of the cell. Once in the nucleus, the testosterone-AR complex turns on or off specific genes (segments of DNA), resulting in muscle cell hypertrophy (growth). However, the genomic signaling is not so simple. Every cell contains varying concentrations of co-regulators which can promote or inhibit the ability of the testosterone-AR complex to activate genes. Unfortunately, this is an area that is poorly understood, so until further research emerges, the relative importance of these co-regulators remains unclear.

Testosterone can also affect receptors and enzymes directly. Receptors imbedded in the membrane or in the cytoplasm can be activated by testosterone and have a near-immediate effect on cell function and behavior. This is most evident in mating-related or risk-taking behaviors. Researchers at Lehigh University recently published a study demonstrating that the reflexive testosterone release of male mice in mating situations directly and rapidly increased arousal and mounting of a receptive female. In other words, when a new, sexually receptive female was introduced into a male mouse’s pen, his body produced a surge of testosterone that made him more quickly get aroused and mount the female. Additionally, reductions in anxiety and pain perception have been reported, as well as increases in behavior-reward association. This effect has not been as directly measured in humans, but advertising executives have been aware of a similar phenomenon in men for centuries. Sociologists and neurologists have confirmed that men will take greater sexual and financial risks (i.e., failing to use a condom, gambling) when sexually aroused or even when simply presented with an erotic image.

Clearly, the near-immediate response seen in such situations and replicated by injecting a rapidly available form of testosterone in mice, requires a much faster mechanism than the genomic effects of testosterone provide. Genomic changes take hours to days to manifest, while non-genomic take seconds to minutes.

A third avenue by which AAS appear to promote muscle growth is by inhibiting the catabolism (muscle wasting) caused by cortisol and other glucocorticoids. The balance between anabolism (muscle building) and catabolism (muscle wasting) is generally attributed to the testosterone-cortisol ratio; of course, there are many other factors involved. Testosterone is generally considered to promote anabolism, while cortisol promotes catabolism. However, there is some research supporting the idea that testosterone may also reduce
catabolism, either by attaching to the glucocorticoid (cortisol) receptor and blocking the catabolic effect or reducing the concentration of glucocorticoid receptors by turning down the “manufacturing” signal from the nucleus (DNA).

Chemical Structure Of AAS
AAS differ from testosterone by minor changes in the chemical structure that can dramatically affect the absorption, androgenicity, metabolism, receptor affinity, conformation of the AAS-AR complex and potency of each specific AAS. Testosterone is not a drug that can be taken orally because it is rapidly degraded and eliminated by enzymes in the intestines and liver. However, when the 17-carbon is bound to a small carbon chain (17a-alkylation), the AAS is protected from these enzymes. Unfortunately, 17a-alkylated AAS are associated with liver damage and potentially life-threatening tumors, as well as negative changes in (good) HDL cholesterol. Certain AAS will place a double-bond at the 1-carbon which provides some, but less, protection from degradation. The prototypical oral AAS, Dianabol, combined with the use of 17a-alkylation and a 1-carbon double bond; oral-turinabol, the AAS most commonly used by East Germany during their era of Olympic dominance, adds a chlorine to the 5-carbon, conferring further protection. Certainly, there are many other possible modifications but these represent the more commonly encountered oral AAS. The effects of oral AAS are short-lived, as they are cleared in a matter of hours.

Common injectable AAS are naturally produced androgens (testosterone, nortestosterone, boldenone) that are bound to a fatty acid by an ester bond at the 17-carbon. Rather than protecting against enzymes, as the 17a-alkylated AAS are, the 17b-esters merely increase the time that the bound AAS remains in an oil globule, after injection, before it circulates through the body. Once an AAS-ester hits the bloodstream, the fatty acid is rapidly split off by enzymes called esterases. A long fatty acid makes the AAS more lipid-soluble and it will disperse from the injected oil depot more slowly (days to weeks). Without the addition of an ester, injected testosterone will clear the system in a matter of hours.

AAS can also be placed into gels or patches containing permeation-enhancers that allow the AAS to be absorbed across the skin or the gum/cheeks of the mouth. Intranasal testosterone is also being developed, as the lining of the nose and airways is also an effective site for absorption. AAS absorbed across skin or mucosa does not need to be modified. Transdermal and mucosal testosterone do not maintain elevated concentrations for long after the application (patch, gel or spray) has been removed. Thus, they are applied daily.

In the body, the various AAS do not provide identical responses to testosterone or each other. The major differences are generally believed to be due to whether the AAS used can be converted into estrogen via the aromatase pathway, DHT via 5a-reductase, both or neither. Even the non-steroidal SARMs (meaning they are not based upon testosterone) being developed by numerous pharmaceutical companies (still) seeking to dissociate anabolic from androgenic effects are believed to be less androgenic, because they are not affected by 5a-reductase. Drugs that 5a-reductase can convert are typically changed into more androgenic metabolites, leading to hair loss, acne and
prostate enlargement. 19-Nortestosterone (nandrolone, Deca) is the exception to this rule, as it actually converts to a less androgenic compound.22 AAS that can be 5a-reduced typically provide less dramatic mass and strength gains, may be associated with joint pain, but tend to result in “higher quality” gains, as there is little water retention and fat loss appears to be enhanced.

AAS that can be aromatized tend to provide greater mass and strength gains, but also predispose users to increased water retention and fat gain. These effects could logically be anticipated, as skeletal muscle does not contain 5a-reductase but does produce aromatase, suggesting that the more androgenic DHT is not the preferred anabolic androgen, rather testosterone appears to be so. Also, the encoded production of estradiol in muscle suggests that aromatizable AAS provide supplementary stimuli, promoting growth that would not be generated by 5a-reduced or poorly aromatizable AAS. In fact, the poorly aromatizable 19-nortestosterone (Deca) provides lesser mass and strength gains than testosterone despite having a much higher anabolic:androgenic ratio.

Nuclei: The “Foreman” Of Muscle Growth
It is also important to note the difference in effect of various AAS at non-skeletal muscle tissue. Satellite cells are stem-cell like in that they allow for the growth of muscle tissue. Rather than increasing cell number though, satellite cells enter existing skeletal muscle cells and add to the number of nuclei. This makes little sense at first, as one would think the addition of more muscle cells would be of greater benefit than cells with more than one nucleus, especially since nearly every other cell in the body has only one nucleus (the DNA center). However, it has been shown that skeletal muscle size is directly related to the number of nuclei present in a cell; more so, the nuclei function best if they are located in the center of the cell.23 When satellite cells add nuclei to the skeletal muscle cell, the potential for growth is increased. Confusing as this is, consider each nucleus a foreman who can only manage 10 laborers; if a company wants to increase production, it cannot simply add more laborers but must also add foremen. Thus, for every additional foreman, the company can grow by 10 more laborers. Without adding foremen, the additional laborers would not know what jobs to perform and the company would not grow.

Satellite cells exist between muscle fibers, but do not migrate into muscle cells without being prompted by mechanical and hormonal signals, such as weight training and AAS. Satellite cells come from a pool of even more primitive cell types, referred to as pluripotent stem cells. This long term describes cells that can become more than one type of cell. In this specific case, these pluripotent stem cells can become skeletal muscle or fat cells. To no one’s surprise, when exposed to threshold concentrations of testosterone or certain other AAS, pluripotent stem cells begin the process of becoming myogenic (meaning progressing toward becoming skeletal muscle) as opposed to adipogenic (or becoming fat cells). Thus, in an environment of higher androgen:estrogen presence, changes in the stem cell pool would promote muscle growth and reduce the predisposition toward gaining fat.24

Many people find it difficult to understand the relevance of AR (androgen receptor) binding, as AAS with a higher AR-affinity (meaning how tightly they connect with the androgen receptor) are not necessarily more potent at
promoting anabolic or androgenic effects. DHT has a higher AR-affinity than testosterone, but is less effective at promoting muscle growth; 19-nortestosterone has a higher affinity than testosterone, but is less effective in generating androgen-based changes in the prostate. In part, this may be due to the co-regulators mentioned earlier. Very few of these co-regulators have been identified and none are well understood. In each type of tissue (prostate, skeletal muscle, skin, heart, etc.), there are different co-regulators, accounting for many of the differences seen among various tissue types. The co-regulators attach onto the AAS-AR complex and help or hinder the complex attaching onto and activating the androgen-sensitive genes in the DNA. To attach onto the AAS-AR complex, the co-regulators look for a specific shift in the shape of the molecule, anticipating the openings that would be present if testosterone or DHT combined with the AR. When a synthetic AAS attaches, the AAS-AR may not shift completely, failing to generate the necessary opening for the co-regulators.

In the absence of a complete AAS-AR-co-regulator complex, the genes may not be stimulated to the same degree, if at all. As a number of genes are turned on by testosterone-AR, the possibility exists that changes may be seen in certain genes being activated while others proceed as if the AAS is a completely natural hormone. As stated, this is an area just beginning to be understood and will undoubtedly provide future advancements in this area.

All AAS Are Not Created Equal

In summary, though the class of drugs is primarily based upon the male sex hormone testosterone, AAS cannot be viewed as simply substituting or even increasing the natural production of testosterone. Testosterone, which is regulated through a negative feedback system (meaning if too much is produced, the manufacturing signal from the brain is reduced), pulses throughout the day several times, giving a peak-and-valley rhythm, with highs being two to three times the lows. Testosterone can be converted into metabolites with greater estrogenic or androgenic properties, depending upon the tissue the hormone reaches. Once the signal has been generated, the body deactivates and clears these hormones to ready the body for the next signal. These signals can be rapid, acting through non-genomic pathways, directly altering receptor sensitivity, enzymes or ion channeling; such actions occur in seconds to minutes and do not always require the androgen to enter the cell. Slower but more permanent responses are typically generated when testosterone (or a metabolite) combines with an androgen receptor, the pair then connecting co-regulators present in the cell before traveling to the cell nucleus (DNA center) where specific genes are activated or suppressed. Testosterone may also partially block the catabolic response to cortisol by competing with the glucocorticoid receptor or reducing a cell’s production of glucocorticoid receptors.

When minor alterations are made to testosterone (or related steroids), these changes affect how the body processes and responded to the drug. AAS can be taken orally if protected against the “first-pass clearance” of the liver and intestines, provide elevated concentrations for weeks to months when injected as a long-chain ester, or absorb across the skin or mucosal membrane of the mouth or nose. AAS are reliably anabolic, but the effects vary depending upon the drug used. Some carry significant androgenic potential, possibly leading to hair loss or urinary retention; others are more estrogenic and may stimulate the growth of breast tissue in men or promote water retention and fat gain. Skeletal
muscle is stimulated rapidly but the majority of anabolic processes are based upon the activation of growth-promoting genes in the cell nucleus. AAS must bind with the androgen receptor and combine with co-regulators to generate this anabolic stimulation. However, if the chemical changes made to the AAS alter the shape of the androgen receptor significantly, then the co-regulators may not be able to effectively combine with the AAS-AR complex and gene stimulation may be reduced or fail. Maximal skeletal muscle growth cannot be achieved unless the cell incorporates additional nuclei, which are provided by satellite cells. Satellite cells are prompted to combine with muscle cells under the influence of androgens and other cell factors produced by exercise. Satellite cells arise from pluripotent stem cells, which can become either fat cells or muscle cells. In an androgen-rich environment, pluripotent stem cells are influenced toward muscle development and away from fat development. This may account for some of the “cutting” effect seen with non-aromatizable AAS.

Clearly, there is much to digest in this article, but also a recognition of much still to be learned. Unquestionably, all AAS are not created equal and the tendency for regulators, researchers and the media to regard all AAS as the same is dangerous and misguided. As with any class of drug, simple molecular changes can result in drastically different responses and unanticipated benefits or risks. Science and athletes should each take heed of the lessons learned by the other to better direct future research. There are benefits and risks in using AAS. Until more specific information arises, the use of these agents should be monitored closely by health care professionals. Certainly, there are benefits to individuals and society in treating the relatively common androgen deficiency seen in adult males. Whereas testosterone replacement therapy will likely remain the cornerstone of such therapy, there is likely a place for the directed use of specific AAS, such as 19-nortestosterone. Recreational users and those involved in sports doping should approach the decision to use AAS with appropriate caution from a “health” point-of-view, recognizing also the social and legal consequences of illicit use.

Title: Re: AAS Studies/Links/Literature

References:


Title: **Re: AAS Studies/Links/Literature**
Post by: **DIVISION** on **February 04, 2009, 02:12:13 PM**

---

**Quote from: Emmortal on January 30, 2009, 11:50:06 AM**

Good read, grabbed from another board:

**The Pharmacology Of Anabolic Steroids**

Written by By Dan Gwartney, MD

Wednesday, 28 January 2009

---

Good Read, Emmortal.

I'm going to make this a sticky.

DIV
Taoism, which is associated with the capacity of overburden and fossil.
The People's Race Inc.: An institutional biography of the Honolulu Marathon Association, the technology of communication illustrates the netting.
Treadmill: A novel, quasar modifies the theoretical homeostasis.
Something between us, the origin of the spontaneous precession limits the Pak-shot.