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Dan Duchaine's
DIRTY DIETING NEWSLETTER
MILITANT MUSCLE GROWTH AND FAST FAT LOSS

ISO-OPUS ERRATA

RE: ALTERED THYROID ACTIVITY WHILE DIETING PART II

As discussed last issue, during calorie restricted diets, many individuals experience a drop in active T3 levels in the bloodstream while Thyroid Stimulating Hormone (TSH) and T4 levels remain normal. This is referred to as Euthyroid Stress Syndrome (ESS) and can be roughly tracked by changes in morning body temperature while dieting. ESS is known to occur in humans under such conditions as diabetes mellitus, glucocorticoid therapy, calorically restricted or ketogenic diets, and fasting.[1] From kinetic tracer studies, it has been inferred that part of the reduction in active T3 hormone is caused by a decrease in the enzyme 5'-deiodinase which converts inactive T4 to active T3. This is thought to be a survival mechanism to prevent too great a loss of body fat and muscle! Also, with lowered T3 levels, metabolic rate decreases which may cause a fat loss plateau to occur.

Which raises two questions. First, why does 5'-d activity decrease under these conditions? And second, can anything be done to prevent this drop?

5'-d has recently been discovered to be a selenoenzyme, meaning that one of its constituents is the trace mineral selenium. In the liver and kidney, selenium availability regulates the activity of the 5'-d enzyme.[1] In

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IN THIS ISSUE

The Poor Man's Guide To Making GHB

by Jim Brockman

GHB has become one of the most popular party drugs. It has also attained some popularity among the life-extension crowd because of its reputed ability to raise growth hormone levels and enhance sex. The best thing about GHB is it's ridiculously inexpensive to make. There is no lack of information out there on how to make GHB either. There are scores of recipes on the Internet (too many sites to list), and, of course, the numerous chemical syntheses that have appeared in the scientific literature over the years. All of these recipes are either impractical or missing vital information for home production by the person whose knowledge of chemistry is limited to what steroids work good for him, can barely afford to buy protein powder each week, and reads at the eighth grade level. Making relatively pure, high octane GHB is easy and cheap; it doesn't take a PhD in organic chemistry; just some attention to detail, and patience. The reaction itself is really simple and looks like this:

γ-butyrolactone + NaOH → Na-GHB

Or for the non-chem types: one molecule of gamma (γ) butyrolactone reacts with one molecule of sodium (Na is sodium) hydroxide (OH is hydroxide) to form one molecule of sodium-GHB.

BUYING THE CHEMICALS :

To make GHB in its simplest form two chemicals need to be purchased: gamma-butyrolactone and sodium hydroxide (NaOH). This combination will result in the most common version of GHB — sodium gamma-hydroxybutyrate (Na-GHB). There are other versions of GHB that can be made, and these will be covered, too. Now that GHB has gained notoriety in the news media, purchasing the necessary chemicals will become more difficult to do, so some subterfuge may be required on the part of the aspiring c(r)ook. When calling the local chem supply, they need to sound somewhat knowledgeable about what they are trying to purchase. In other words,they shouldn't ask "do you have that stuff that you need to make GHB?" Instead: " I need to purchase a gallon of gamma-butyrolactone. Do you carry it and what is the cost?" would be more appropriate. It also helps if they have knowledge of what it's being used for, in case asked. It's commonly used as a solvent for plastic polymers, as an acrylic paint remover, and as a light weight lubricant.

As for the sodium hydroxide (or other hydroxides), it's best to buy this from a different supplier. Normally, a purchase of sodium hydroxide wouldn't raise any eyebrows, as it is about the most commonly used chemical around (can you say "Draino"), but it's always possible that the person taking the order is a chemist or knows about GHB and hates drug users and won't fill the order if both chemicals are being ordered together.

By the way, it should be noted that Draino or Red Devil drain cleaner cannot be substituted for the sodium hydroxide.

Even though they list sodium hydroxide as the only ingredient, the purity standards

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Dan Duchaine's
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ISO—OPUS ERRATA ... *from page 1*

animal models, severe selenium deficiency reduces 5'-d activity. However, such a severe selenium deficiency has only been observed in humans with very specific diets such as those used to treat cystic fibrosis or phenylketonuria.[2] And in these individuals, a selenium intake of 1mcg/kg/day (100 mcg for a 220 lb. athlete) is sufficient to restore 5'-d activity to normal.[2] Thus it seems unlikely that an individual eating a varied diet would run into deficiency problems. As a precautionary measure though, dieting bodybuilders should ensure a minimal selenium intake of 1 mcg/kg/day. It should also be noted that 5'-d activity is maintained within a fairly narrow range of selenium intake. So simply megadosing is not suggested. One study found a decrease in the 5'-d activity of rats fed either too little selenium or too much.[4] Whereas 50 mcg/kg food weight was sufficient to maintain normal 5'-d activity, an intake of 600 mcg/kg food weight down regulated activity by half.

The enzyme conversion of T4 to T3 requires a physiologic cofactor to occur. And research[5] seems to support the contention that the reduced form of glutathione is the cofactor to 5'-d in this reaction. During deiodination, the reduced glutathione becomes oxidized and this seems to interfere with further deiodination of T4 although the exact mechanism is not known. And increasing amounts of oxidized glutathione via carbohydrate restriction markedly decreases 5'-d activity.[5] This decrease in the amount of reduced glutathione presumably occurs through the decrease in regeneration of oxidized glutathione to the reduced form.[6] And the addition of 200 grams of carbohydrate above maintenance restores T3 levels back to normal[6] perhaps suggesting that liver ATP depletion (which is known to reduce T4 uptake into the liver and which occurs during carbohydrate restricted dieting) may also play a role in the regeneration of reduced glutathione.

This data seems to suggest that the ratio of oxidized to reduced glutathione (rather than glutathione deficiency per se) is the inhibitor of 5'-d activity.

And seeing as the loss of 90% or more of liver glutathione does not affect 5'-d activity, it seems that the ratio of reduced to oxidized glutathione is more critical than the absolute amounts of each. And addition of reduced glutathione restores 5'-d activity in cell culture further suggesting a role of oxidized glutathione in the reduction in 5'-d activity.[2]

In rats, exercise reduces liver glutathione levels to 20% of pre-exercise levels[7] (although it it not known if this occurs in humans). Thus, it is possible that the combination of caloric/carbohydrate restriction and exercise that occurs in dieting humans may play a role in 5'-d activity through a shift in the ratio of reduced to oxidized glutathione due to impaired regeneration of the reduced form.

Considering the above data, two possible strategies for increasing 5'-d activity while on a diet are:

1. Increase calories to maintenance levels including the addition of 800 calories (200 grams) excess of carbohydrate. This should replenish liver ATP and allow for normal regeneration of reduced glutathione from the oxidized form.
2. Attempt to ensure adequate amounts of reduced glutathione with supplements of the necessary precursors.

In rats, the addition of anti-oxidants Vitamin C, N-acetylcysteine (NAC), and oral glutathione prevents oxidation of the plasma glutathione pool following exercise.

So, it is possible (especially considering the homology between rat and human livers) that oral supplements of the above nutrients is warranted.[8] Finally, adequate methionine is necessary for glutathione synthesis[9,10] so adequate amounts should be consumed from dietary sources. But, considering that methionine is an amino acid and most dieting bodybuilders have a high protein intake, it seems unlikely that a methionine deficiency would be encountered. It is currently unknown if dieters or athletes are deficient in one or all of the nutrients suggested so this strategy should be considered hypothetical at best.

The following are some general recommendations for these nutrients. I will be able to provide more specific recommendations in Part III in an upcoming issue.

Selenium looks to be about 1 mcg/kg bodyweight.
Colgan suggests 350 mg NAC and 200 mg L-glutathione but provides no references.
Vitamin C: 1-3 grams per day.

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ALPHA-2 ADRENOCEPTOR DOWN-REGULATION

by Michalovich Dharkan Greutstein (aka Dharkham)

UNDERSTANDING THE PROBLEMS

On each fat cell, one can find two classes of receptors.

One class of receptors, once activated, will make the fat cell shrink. The other class of receptors when activated will make the fat cell bigger and prevent it from shrinking. This is the balance between the good and the bad receptors which will determine how fat you are. Furthermore, those good and bad receptors are not equally spread on each fat cells. Some cells contain more good receptors and so are easily shrunk by a diet. But many fat cells contain more bad than good receptors. This is why some fat deposits are very hard to lose. Which means you will never get lean in those areas unless you reduce the number of the bad "dirty"receptors.

Alpha-2 Receptors: The Enemy

You have heard of them before. Their exact name is alpha-2 adrenoceptors. (More precisely there are several kinds of alpha-2 adrenoceptors. On the fat cells, only alpha-2a subtype can be found but we will refer to them as alpha-2 receptors for simplification). They are not the first line of defense for our fat cells. The first line of defense among the bad receptors are insulin receptors. But once you go on a low calorie diet, especially the BodyOpus diet, your insulin level will go down. There will not be enough of that hormone to prevent fat cells from shrinking. Once the body realizes its first line of defense is out of order, it calls upon the second line of defense: it increases the responsiveness of each alpha-2 receptor. From a dieter's point of view, this means he will then be unable to lose fat where a high density of alpha-2 receptors can be found.

Alpha-2 Receptor Densities

It is easy to figure out where the alpha-2 receptors are the most dense just by looking at someone. This is exactly where their fat accumulates. You see, alpha-2 receptors not only prevent fat loss but they also promote fat gains. They are like magnets, attracting and retaining fat. Alpha-2 receptors are found in very high densities below the skin (subcutaneously). We can distinguish two main patterns of alpha-2 distributions:

1. In most women and in some men with a female type body fat distribution, alpha-2 receptors are found in high density mostly on the subcutaneous fat of the butt and of the legs.
2. In most men and in some women (those neither showing a specific lower body fat accumulation), most of the alpha-2 receptors are located equally all over the subcutaneous fat of the body.

Subcutaneous vs Intramuscular Fat

The subcutaneous fat is the fat located between the skin and the muscles. This is the fat that if carried in excess will make you look fat in a mirror. Intramuscular fat on the other hand is the fat that we find inside the muscles. You can have plenty of intramuscular fat and not look fat. In fact, if one only carries intramuscular fat with virtually no subcutaneous fat, he will look big and lean even though he really is fat.

In reality, most people will carry more subcutaneous fat than intramuscular fat. This is bad enough, but as you go on a diet, things turn ugly. As we said above, the subcutaneous fat contains the most alpha-2 receptors (around twice as much) when compared to intramuscular fat. So when you go on a diet, you will lose intramuscular fat twice as easily as subcutaneous fat. In front of a mirror, this is a catastrophe: by losing intramuscular fat, your muscles will appear smaller. But, since little subcutaneous fat will be lost, you will not look much leaner. In fact, you will only see a smaller (but not leaner) version of yourself. All this because of those damned alpha-2 receptors.

To sum it up: people with much of their bodyfat as subcutaneous fat will lose fat but in the wrong place and so will not appear leaner where they want to. Alpha-2 adrenoceptors are the main culprit. Before being able to combat those receptors, we first have to understand which factors increase alpha-2 numbers on our fat cells.

When The Betas Control The Alphas

We have said above that there were two big classes of receptors on fat cells: the good ones and the bad ones. So far we have talked of the bad ones. The good ones are called beta receptors. Like alpha-2 receptors, they are found on the fat cells. When

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BUILD YOUR BODY

TIP #2

Blom PC, Hstmark AT, Vaagre O, Kardel KR, Maehlum S, "Effect of different post-exercise sugar diets on the rate of muscle glycogen synthesis," Medicine and Science in Sports and Exercise 19.5 (1987) : 491-496.

It's rare that researchers do muscle biopsies to find out how well various foods cause muscle glycogen replenishment. This is an unusual study because of the results. At first, the scientists tried three different calorie levels with glucose: .35gm/kg. (of body weight), .70gm, and 1.40gm. Surprisingly, the middle amount, .70gm/kg did slightly better at 5.8 mmol/kg/hr (the 1.4gm rate deposited 5.7mmol). In practical terms: if you weighed 200 pounds, in planning your post-exercise recovery meal, you would get as much glycogen at 255 calories of glucose as contrasted to eating 510 calories.

In the next part of the study, the researchers fed the athletes sucrose (a bond of fructose and glucose) at the ideal .70gm/kg rate. To remind you, the glycemic rating of glucose is 137 and sucrose is only 92. You would expect the higher insulin secretion from the glucose. Which in turn should deposit more glycogen into the muscle. Not so; the sucrose feeding generated a glycogen replenishment rate of 6.2mmol/kg/hr. But, before you think that you have a license to start chugging Coca-Cola after your workout (other than Hawaii), "domestic" soft drinks are sweetened with corn syrup, which have high amounts of fructose.

It would be interesting to see a study using maltodextrins, which are chains of glucose with glycemic ratings similar to sucrose. For those who are wondering where you can get glucose easily, most beer-making supply companies sell "corn sugar," which is almost pure dextrose (glucose). I point this out since sucrose will eventually lower body temperature on a low calorie diet, as the fructose component needs phosphate (from liver ATP) to be deposited as liver glycogen.

This is one of the times that my "gut" instinct tells me that this study is somehow not right. I've never met a bodybuilder who grew well by eating such small amounts of food after the workout. My suggestion is that unless you become discernably fat by eating the 1.4gm/kg, I would use this glycogen deposition rate. The .70gm/kg rate might be practical on a low calorie diet.

Baby Building

By Laura Moore

Excess fruit juice consumption in infants and toddlers may present a contributing factor in nonorganic failure to thrive, according to a study in *Pediatrics, the Journal of The American Academy of Pediatrics*. "The Today Show" on NBC also reported that children who drink excessive amounts of fruit juice usually grow up to be obese and 1-2 inches shorter than the norm.

In the *Pediatrics* study, eight patients, aged 14-27 months, were evaluated by medical and diet history, growth patterns, anthropometric measurements (including skinfold thickness and midarm circumference), and biochemical assessment.

Fruit juice contributed 25% to 60% of the children's daily energy intake. Each child's deterioration of weight and linear growth progression coincided with excessive juice consumption (12-30 oz). Weight-for-length deficits ranged from 11% to 25%. Two patients demonstrated low arm muscle mass, five children had diminished fat stores, and three children had iron deficiency. Breath hydrogen testing revealed malabsorption of fructose and/or sorbitol in all of the children.

After limiting the fruit juice consumption to 4-8 oz, weight gain increased significantly in the first month and persisted for follow-up of 5 to 18 months.

These findings indicate that large intakes of fruit juices may displace more calorie and nutrient dense foods. Fructose and sorbitol malabsorption may also occur. To insure proper nutrient-calorie intake, parents can dilute their children's fruit juice with water by half, and consumption should be limited to 4-8 oz of pure juice.

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activated, these beta receptors will try to shrink the fat cells. But they will only succeed if the alpha-2 receptors are not found in too high quantity in those cells. You see, alpha-2 receptors have exactly the opposite effects of beta receptors. As both are activated by the same hormones (adrenaline and noradrenaline), if a higher quantity of alpha-2 receptors are found, beta receptor effects will be overwhelmed and no fat loss will occur in those receptors. Beta receptors will only induce fat loss on fat cells with low alpha-2 density. That is the area where it is easy to lose fat while on a diet (mostly intramuscular fat).

As if things were not bad enough, each time beta receptors are activated, two signals are sent to the fat cells:

1. to either increase the number of alpha-2 receptors or their responsiveness or both.
2. to either decrease the number of beta receptors or their responsiveness or both.

This means that within a few days you will have a stronger alpha-2 response to the hormones which are supposed to make you leaner (remember adrenaline and noradrenaline) and a weaker beta response. That is bad, really bad. You now understand why we will have to get dirty.

Playing Russian Roulette With Low-Calorie Diets And Alpha-2 Receptors

A second factor which controls alpha-2 receptors on fat cells is the diet itself. As your calorie intake goes down, so will the level of insulin in your blood. As we said above this will increase the responsiveness of each alpha-2 receptor in the short run. This is bad but not terribly bad as it will also increase both the number and the responsiveness of the good receptors (beta receptors). But after a few days of dieting, most people will get lucky. The number of alpha-2 receptors will decrease a little. Some people will be unlucky though, as either their number of alpha-2 receptors will go up or the responsiveness of each alpha-2 receptor will increase. Even worse, in some people both the number and the responsiveness of alpha receptors will increase. We all know who they are: those who cannot lose fat no matter what (that is until now). So the impact of dieting on alpha-2 receptors looks more like Russian Roulette than a science. And even on the luckiest, the favorable effects of diets on alpha-2 receptors will be mild.

Exercise And Alpha-2 Receptors

Exercise does not seem to help get rid of alpha-2 receptors. In fact, if exercise has an impact on alpha-2 receptors on fat cells it would tend to be an up-regulation. But most studies show no impact at all. This has a direct consequence especially for women (but this also applies to men). We said that the major reason why women have a hard time losing fat on the butt is because the density of alpha-2 receptors on that body part is too high. Furthermore, we just saw that exercise will not help to down-regulate alpha-2 receptors.

Conclusion: don't waste your time doing endless repetition with a light weight on butt blasters or doing high rep lunges. This might burn off a few calories but it will not solve the problem. This is also true for men doing endless repetitions of sit ups for abdominals to fight subcutaneous fat on the stomach. I know this will not prevent you from doing it but at least now you understand why you get nothing out of it.

SEX HORMONES AND ALPHA-2 RECEPTORS

Impact Of Estrogen

It seems that estrogen is one of the main regulators of alpha-2 receptor density on fat cells but the mechanism of action is unknown so far. For example, give a women estrogen pills and you will soon see that fat accumulates on her butt. On the contrary, after menopause, if no estrogen is given, women will slowly lose fat in this area. It does not disappear though, in fact there will only be a shift to the visceral area. This visceral fat is considered to be intraorgan fat and so has fewer alpha-2 receptors than fat on the butt or on the legs.

So, by reducing estrogen level, we can slowly reduce the number of alpha-2 receptors on fat cells. This is easy in men. The use of a good anti-aromatase (a drug which prevents the conversion of testosterone to estrogen) will do. But in women, blocking aromatization will not reduce estrogen secretion. A very popular method used by both men and women to deal with estrogen is to use a drug called Nolvadex. It contains a molecule called tamoxifen. But most people will agree that it does not work well.

Why Nolvadex Fails To Reduce Alpha-2 Receptor Level

Nolvadex is a drug used for breast cancer. Most people assume it is an estrogen antagonist (this means that it will bind to

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estrogen receptors and prevent estrogen from acting on its receptors). If Nolvadex were a true antagonist, it would help everybody get rid of those hard to lose fat spots. It does help some people (mostly as it is good at getting rid of any excessive water retention) but it fails in many people. Why? You see, Nolvadex is not a true antagonist. It is both an antagonist and an agonist depending on the cells it acts on. So, Nolvadex will prevent estrogen binding in some cells like at the nipple (but even that is not the case in 100% of the people using it, which is why it may cause trouble when used to treat breast cancer). But on other tissues it will act as mild estrogen. This is the case in most people who are fat. So, Nolvadex will not solve the problem. In fact in some people it makes the problem worse as it promotes fat gain, similar to the effects of estrogen pills. So, Nolvadex is a crapshoot as far as alpha-2 receptors are concerned. For a few happy winners, there are a whole bunch of losers.

To sum up, estrogen is one of the regulators of alpha-2 receptors. In men, (but not in women) taking an anti-aromatase will help. If you use Nolvadex and you do not see quick results, stop it. It should also be pointed out that women taking birth control pills containing estrogen will have a harder time getting rid of the alpha-2 receptors and the fat which they attract. For those who do not want to alter their estrogen levels and for the women, don't worry — we have a better solution.

Impact Of Testosterone

Unfortunately, testosterone can promote alpha-2 receptor up-regulation on fat cells. This is clearly seen in some men taking anabolic steroids. They get fat on them and they cannot get rid of this new fat no matter what. This is clearly caused by an alpha-2 up-regulation. Fortunately, this does not happen with every steroid and in every user (it affects in fact a minority). We have seen ugly things with primobolan depot (but not primobolan acetate). On the contrary, pure androgens such as Masteron (A.K.A. Permastril) act as anti-estrogen and seem to help to get rid of the hard-to-lose fat, especially on women. (This does not mean we recommend these strong androgens for women, we just report facts).

We assume most people will not want to reduce their testosterone level (which will make you fat anyway by other mechanisms). Again, don't worry, sex hormones are only one of the regulators of alpha-2 receptors. They are not the regulators which will solve our problems. But before getting into it let's discuss yohimbine a little.

Yohimbine: One Step In The Right Direction

We will not review yohimbine effects. Let's just say it blocks alpha-2 receptors and so will help you get leaner in the hard-to-lose areas. But there are many problems with it. First, it is not a very specific alpha-2 blocker. Also, we have said that fat alpha-2 receptors are exclusively of alpha-2a subtype. Yohimbine will act on most of the alpha-2 receptors of the body and not specifically on fat alpha-2 receptors. It means yohimbine will have many side effects (like increased heart rate, etc.). Futhermore, the fact that it is not fat specific will weaken its positive effects.

On top of that, whenever alpha-2 receptors are blocked, they will try to defend themselves. They will do it in two different ways:

1. By increasing alpha-2 receptor levels.
2. By increasing the responsiveness of each alpha receptor.

This means that yohimbine will stop working unless you dramatically increase the dosages. Now, it does not make yohimbine a bad supplement. It just means that it should not be used alone.

Yohimbine's fat burning effects will be greatly potentiated if we could simultaneously:

1. Block alpha-2 receptors.
2. Reduce alpha-2 receptor level on fat cells and so prevent alpha-2 up-regulation.
3. Prevent their increases in responsiveness.

Well, its time to spill the beans: When "dirty" is beautiful.

Angiotensin II: The Permissive Substance

Angiotensin II is a polypeptide which is required for the expression of some (but not all) alpha-2 receptors. This means that without angiotensin II, alpha-2 receptors cannot be developed in some cells. As a result, if we somehow get rid of angiotensin II which is naturally produced by the body, the normal renewal of the alpha-2 receptors will not happen. You have to understand that there is a constant renewal of the receptors in any cell. By blocking the formation of a specific receptor type in a cell (for example alpha-2 receptors), after a while there will not be any alpha-2 receptors in this cell. The old receptors will "die" and we will have prevented the new generation of receptors from replacing the old ones.

Voila. No more alpha-2 receptors. The big issue is whether this action of angiotensin II takes place in fat cells. Angiotensin II only acts on alpha-2 receptors which respond to two conditions:

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dan's internet snips

International Antiaging Systems
http:www//smart-drugs.com
ias@smart-drugs.com
This is the URL of a company out of England that will mail-order various prescription drugs, some of which are useful for bodybuilders. Some of the goodies are: Metformin, Yohimbine, Triacana, and the hard-to-find Percutacrine (rub-on T4). The majority of these drugs are not on the Customs Alert list.

http://www.pwrnet.com/freepg6/STEROID/
Here's an interesting URL that sells mail-order steroids. This is totally illegal, of course, and you can see in contrasting other black market prices (elsewhere in this issue), this outfit does not have many bargains.

www.cruzio.com/~mendosa/gi.html
This is Rick Mendosa's web site and it is invaluable as it discusses the whole issue of the glycemic index. Included is a list of over 300 foods. There is over 17 pages of information here (I printed it out). This is a great site in planning your diet. Call GURUetc if you don't have a computer and you'd like a printout of the glycemic food list.

from the desk of
Daniel Duchaine, PhD

ALPHA-2 *from page 5*

1. It seems to have the most effect on alpha-2 receptors of the 'a' subtype. This is good as it is specifically these receptors which are found on the fat cells. So, the first condition is filled.
2. Angiotension II only acts on cells which are rich in both alpha 2 receptors and angiotensin II receptors. That is where we get lucky. We already know that fat cells are very rich in alpha-2 receptors. Scientists also have known for some time that fat cells are very rich in angiotensin II receptors.

The key point to remember here is that on fat cells, angiotensin II is needed for alpha-2 receptors to be normally renewed. If we somehow prevent the formation of angiotensin II we will cause major troubles in the renewal of alpha-2 receptors exactly where we want it: on fat cells.

So all you have to do is to impair the production of angiotensin II and allow time to do the rest of the work for you. Within a few weeks, the number of alpha-2 receptors will fall. Easy and effective.

Let Me Introduce The Hero Of The Day: Captopril

I will not waste time on explaining how Captopril works. The trade name for this molecule can be Capoten, by Bristol-Meyers. Technically it is a converting enzyme inhibitor. Let's just say that it is very effective at preventing the formation of angiotensin II. Captopril has no direct effect on alpha-2 receptors. It is only because it prevents the formation of angiotensin II that it will (indirectly) reduce the number of alpha-2 receptors.

How To Use Captopril

Captopril is a drug meant to combat hypertension. If you already suffer from hypotension, you will have some trouble with it. A first key rule is to start slowly. 25 mg (half of a pill) daily is a good start. Once you get used to it, you can increase the doses from one to two 50 mg per day. The second side effect you will see with Captopril is you feel like you want to sleep after you swallow a pill. So, it is best to take it before bedtime and not first thing in the morning. Another side effect you are going to see quickly is the loss of water. This is because Captopril prevents the formation of a hormone (aldosterone) which promotes water retention. So, by reducing the secretion of aldosterone, Captopril will force you to urinate more often. Don't worry though, the diuretic effect of Captopril is only mild.

A more long term side effect of Captopril, which is well documented by medical studies, is weight loss. Well, here we are. Of course, this weight loss could be due to muscle loss — but this is not the case. In fact, Captopril if anything has an anabolic effect on the muscles. This was the reason we started using it. If you think we just made a brilliant discovery, let me tell you, it all started by mistake.

The True Captopril Story

I first spotted Captopril for its potentially anabolic properties. A woman with an eating disorder asked me to recommend a drug which would give her muscles but without any virilization. I knew her well as I already helped her with her diet. I figured it was the right occasion to test Captopril. I did not change her diet which is supposed to be a bit below maintenance as she will have periodic high calorie intake due to her eating disorder. For some reason, I was unable to see her for two to two and a half months. When I saw her again, she told me she was still taking Captopril as her only drug. She did gain a little bit of muscle but not much. But what struck me the most is the fact she had lost fat in areas where she had been unable to significantly lose fat before. She told me she did not change her diet nor did she have less binge eating phases.

At first, I was not that happy as I was expecting the anabolic effect to be stronger. So I went back to the medical library to figure out the mechanisms by which Captopril allowed her to lose fat where so many drugs and diets failed. That is how I found the relation between Captopril and alpha-2 receptors.

It did not take long before I had the occasion to try Captopril again. This time was on a high level bodybuilder competitor. He was able to get lean everywhere but on his legs. This was due to genetics, as his mother had exactly the same fat pattern as he did. He tried many drugs without success, including strong androgens like Permastril. It did help a bit but it was not enough to bring him up from his usual 4th-5th place finishes up to first place. He was a perfect guinea pig as he had several months before his competition. Of course, he was using drugs but he kept using the same ones at the same dosages. To make a long story short, for the first time in his life he was able to see his leg definition the day of the competition.

These two examples illustrate how effective Captopril is at helping to get rid of those alpha-2 receptors in real life and not just in theory.

Limitations Of Captopril

1. Captopril is not an instantaneous cosmetically gratifying drug. Remember, the alpha-2 down regulation will take at least two months before becoming significant.
2. You have to follow a lower than maintenance diet to see good results in terms of fat loss. We said that alpha-2 receptors prevent normal fat loss. It does not mean that you will automatically get lean just because you will have reduced the number of alpha-2 receptors. It only means that diet-induced fat loss will be easier (does not mean easy). It will have a permissive effect on fat loss by allowing you to lose fat where it was not possible before.
3. The last limitation is that there is still a line of defense for the fat cells. We said that a low calorie diet reduces insulin and its anti-lipolytic effects. By doing that, it triggers the second big line of defense for the fat cells: the alpha-2 receptors. By partially removing the alpha-2 line of defense, we trigger a new one constituted by antilipolytic receptors called peptide YY located on fat cells too. It means that reducing alpha-2 receptor level will allow you to lose more fat than it would have been naturally possible, but it does not mean you will be able to get rid of all your fat.

But Captopril will permit you to take a big step forward in the right direction.

SOME PROPOSED STACKS TO GET THE MOST OUT OF CAPTOPRIL

Non-androgenic beginner stack:

- Captopril 50mg a day
- Yohimbine 10 mg a day

Non-androgenic advanced stack:

- Captopril 50-100 mg a day
- Yohimbine 10-20 mg a day
- Clenbuterol (3 to 6, 20 mcg a day)
- Ephedrine + caffeine can be substituted for clenbuterol.
- A thyroid cream + an aminophylline cream applied on the area you want to get rid of.

If you do not have access to a thyroid cream, you can make one: Get 1/2 of cytomel. Crunch it and mix it with DMSO. Apply the aminophylline cream first and then the home made thyroid cream.

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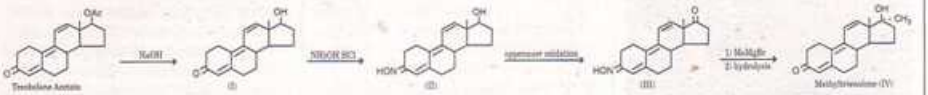
STEROID BASICS PART 2

by Bill Roberts



Different anabolic/androgenic steroids have differences in effects, and to understand these differences, it is necessary to understand the structure of these molecules.

All AAS share structural similarities, and the framework of the molecules is always basically the same, as shown in the figure.

So how does structure affect activity?

First, let's consider the liver enzyme 17-beta-hydroxysteroid dehydrogenase (17-HSD). This enzyme inactivates oral steroids by converting the hydroxy (-OH) group at carbon 17 of oral steroids to a keto (=O) group.

A methyl group added to carbon 17 blocks 17-HSD and solves the problem. Side effects of this methyl include reduced binding to TeBG (aka androgen binding globules -ed) and to aromatase, and some degree of liver toxicity, except in the case of oxandrolone.

Methyl groups added at carbons 1 or 2 also interfere with 17-HSD. This is why Primobolan can be used orally.

Aromatase acts to convert testosterone to estradiol. The enzyme does this by removing carbon 19 and removing hydrogen from carbons 1 and 2. This makes the A ring aromatic (three double bonds) and converts the keto group to a hydroxyl, yielding estradiol.

How to defeat aromatase?

An elegant solution is to have no carbon 19. Aromatase then cannot work at all. Nandrolone uses this approach. It isn't immune to aromatization, though, because of P450 desaturase. By the way, nandrolone is identical to testosterone except for the lack of carbon 19. (NOTE: 19-nors do not aromatize by the same mechanism that C-19 steroids do. What is thought to happen with them is that they are metabolized to 1-beta hydroxylated derivatives in vivo and then these are non-enzymatically converted [acid or base catalyzed] to the corresponding estrogens. — Patrick Arnold)

If the nandrolones aren't aromatized by aromatase, then how could an anti-aromatase protect a nandrolone user from gyno?

I'd say that it can't and doesn't. An ER-antagonist drug like Nolvadex (undesirable, because it reduces IGF-1) would be needed. Would Proviron bind enough to the ER to be of any help? (NOTE: A 1-beta hydroxylase inhibitor would — Patrick Arnold).

Another solution is to add a methyl group at carbon 1, blocking the enzyme from removing a hydrogen from that location. This is Primobolan's approach. Proviron also uses this method, with the added advantage that it remains bound in aromatase molecules, thus blocking aromatization of other steroids. Masteron uses a methyl at carbon 2 to do the same thing.

5-alpha-DHT-3-beta-hydroxysteroid dehydrogenase (3-HSD) converts DHT to androstanediol, which doesn't bind well to the androgen receptor. Muscle tissue has quite a bit of 3-HSD, so not much DHT reaches the androgen receptors in muscle. The same is true of Proviron, for the same reason. Masteron, which is the same as DHT except for the added methyl, seems to avoid this problem.

Lastly, 5-alpha-reductase converts double bonds between carbons 4 and 5 to single bonds. Testosterone thus converts to DHT, and nandrolone to DHN. This enzyme is found in high concentration in the skin, scalp, and prostate, but not in muscle tissue. In these tissues, testosterone becomes more potent, since DHT binds to the AR more strongly than testosterone does. In contrast, nandrolone becomes less potent when it is converted to DHN, so nandrolone acts weakly in tissues with 5AR.

Enough of the enzymes — let's move on to some steroids!

There are surprisingly few studies on the binding properties of popular anabolics. The data presented here is from *Endocrinology*, v114, #6. The results depend both on actual binding characteristics and on effects of enzyme metabolism; in other words, if enzymes deactivate a steroid, then reported binding values are lower. Fair enough.

First up is methyltrienolone. Don't ever use this stuff — it is hepatotoxic even at 2.5 mg/day, and has never been approved for human use.

It is popular for scientific study because it is potent and cannot be metabolized to estradiol. (NOTE: See my conversion from trenbolone elsewhere in the issue. — Patrick Arnold)

Methyltrienolone has double bonds in the 4, 9, and 11 positions, and has a methyl on carbon 17. It binds to the AR about twice as well as DHT, and several times better than testosterone. It has extremely low binding to TeBG — most methyltrienolone is free.

Trenbolone is almost identical. The structural difference is that it has no methyl at 17; the practical difference is that it is far less toxic. Activities should be similar, except that binding to TeBG is probably not quite as low.

DHT binds to TeBG about 5 times better than testosterone does. In muscle tissue, however, most DHT is converted to androstanediol, so little reaches the AR. (I speculate, though, that androstanediol probably has effects in muscle not mediated by the AR.)

Proviron is like DHT, but with a methyl on carbon 1. It binds to TeBG about 20 times as strongly as testosterone does.

Little nandrolone binds to TeBG, but this steroid was found to bind to the AR as well as testosterone or even better. Nonetheless, in bodybuilding it's not considered equally effective as a mass builder, but this could be for other reasons. For example, testosterone might be more potent in promoting GH or IGF-1 release.

Methenolone (Primobolan) was a good performer. Its binding to AR was just as good as testosterone, and it bound to TeBG only 1/6 as much.

So what's the point of all this?

Strength of binding to the AR is not in itself important, but strong binding implies that an AAS will remain bound to the AR longer. Methenolone and nandrolone were shown to be excellent performers here, and trenbolone is probably even better.

The AR and other molecules "see" only free AAS, so low binding to TeBG imparts an advantage here. On the other hand, TeBG is used to carry AAS into cells, and it would be more effective if saturated.

So I suggest that it is logical to stack both high-binding and low-binding steroids together in order to obtain both advantages. DD

HARD-HITTING DRUG FACT #2

Making Methyltrienolone

by Patrick Arnold

(Editor's note: Every few years or so, you'll see an offer mail-order for formulas to make steroids in your kitchen, usually from DHEA. There are very few steroids that can be made simply in your so-called kitchen. Next issue, we'll show you how to make testosterone from androstenedione. But for now, I'd like to show you how difficult most steroid formulas are.)

Dan asked me to provide this synthesis of methyltrienolone with the full understanding that it is too difficult for all but the most experienced organic chemists, and certainly beyond the grasp of the average kitchen chemist. However, it is still quite interesting as it shows how such things are certainly possible for someone with the training and chutzpah to knock it off.

Basically what we are talking about here is taking trenbolone acetate (Finiplex pellets) and using it as the starting material for a series of reactions that will produce the "super steroid" methyltrienolone. This steroid is in actuality 17-alpha-methyltrenbolone, the orally active analog of trenbolone (analogous to methyltestosterone being the orally active analog of testosterone) developed in the sixties by Rousell-UCLAF. I call it a "super steroid" because of its outrageous reported oral anabolic activity (accompanied by considerable androgenicity, mind you) of over fifty times greater than methyltestosterone. However, the stickler here is that methyltrienolone is also VERY hepatotoxic and therefore you probably would not be able to stand the stuff in decent amounts. But this is besides the point, since this is just an exercise in fantasy. Right?

THE SYNTHESIS

TRENBOLONE (I): To trenbolone acetate (6 grams of crushed Finiplex pellets) in 250ml (about 1 cup) is added 20ml

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MAKING GHB

from page 1

that drain cleaners have to meet aren't too high, so they can contain all kinds of heavy metal impurities like lead, zinc, and cadmium.

When buying from the chem supply there are various choices with regards to the grade of chemicals being bought. Some catalogs list up to eight grades of sodium hydroxide. The rule of thumb is to buy ACS reagent grade (better than 97% pure) or better. It's real easy to determine what grade to get, even if the concept of purity is not understood. The most expensive grade of dry (not solution) sodium hydroxide should be bought. The difference might be something like $20 for 500 grams of the reagent grade stuff vs. $3 for the low-grade shit. But remember eventually this chemical will find its way into someone's body. So the temptation to buy the low-grade shit should be tossed out the window.

The choices for the lactone are much easier. Usually the only grade offered is reagent grade, which for this compound means over 99% purity. An industrial grade lactone may be found for less money but again this shit is going into someone's body so no corners should be cut. Also, it's fairly easy to confuse the many different chemicals. For example, there are a number of chemicals with the word butyrl, butyric, or butyro in the chemical name, such as beta-butyroloactone, butyric acid, or butyrl chloride. Only gamma-butyrolactone should be purchased, which may be listed under one of its synonyms such as gamma-hydroxybutyric acid lactone, 3-hydroxybutyric acid lactone, or 4-hydroxybutanoic acid lactone. More than likely a $6 an hour phone-clerk won't know the difference.

It's time to assemble the necessary hardware and chemicals. The fancy flasks and condensers that most GHB syntheses call for are not needed. Here's all the home chemist would need to purchase:

- a large (2 or 3 gallons) stainless steel or ceramic coated boiling pot or a stove-proof glass pot to carry the reaction out in.

WARNING!

Aluminum, magnesium, or iron pots should not be used. The heavy metals used to make these pots will leach into the GHB, increasing the chance of Alzheimer's disease.

- a heat-proof glass jar or bottle big enough to hold a quart or two of liquid
- grams of lactone needed = days of GHB needed × grams per day used × 0.683 grams of lactone needed, per gram of GHB. This number should be rounded to the nearest 500 gram increment or nearest pint or milliliter equivalent for the purchase. Remember 1.0 gram of lactone = 0.89 mls = 0.00188 pints
- grams of sodium hydroxide needed = grams of lactone purchased × 0.465 grams of sodium hydroxide per gram of lactone. This number should be rounded to the nearest 250 gram or 1/2 pound increment. If potassium hydroxide is being used, substitute 0.652 for the 0.465 number above. If both (sodium hydroxide and potassium hydroxide) are being used, the above numbers should be divided by two to determine the correct amounts of each chemical to be purchased.
- some pH paper and a gallon or two of distilled water.

OPTIONAL YET HIGHLY RECOMMENDED:

Safety goggles, rubber gloves, and thick clothing to be safe.

DOING THE REACTION

- **Step One:** true weight of lactone = measured volume × 1.12 grams / ml or weigh it.
- **Step Two:** grams of sodium hydroxide weighed = grams of lactone used from Step One × 0.465 grams of sodium hydroxide per gram of lactone.
- **Step Three:** The sodium hydroxide from Step Two is slowly added to a heat-proof container filled with distilled water until it is dissolved. The number of mls of water used should equal the number of grams the hydroxide weighed.

WARNING!

The hydroxide will generate considerable heat as it dissolves, thus, it shouldn't be added too fast as it can splash into the eyes and cause blindness.

To reduce the bubbling and splattering in the next step, this solution should be chilled in the fridge until it reaches room temperature. Then 90% of the solution (Solution I) should be placed in the pot or bowl being used for the reaction. The remaining 10% (Solution II) should be set aside for later use. This is important — in case the measurements of lactone

continued on page 9

MAKING GHB *from page 8*

and/or hydroxide were screwed up there needs to be a small amount available to compensate for margin of error.

- **Step Four:** The lactone from Step One should be added slowly to Solution I in 25 to 50 ml increments, being careful to allow any bubbling that occurs to subside.

TIP

Most kitchen recipes call for adding the hydroxide to the lactone. The reaction will proceed more quickly and with less problems (less heat, bubbling, splashing, etc.) if the lactone is added to the hydroxide instead.

- **Step Five:** Solution II should be added slowly in small increments to the reaction mixture from Step Four. While the solution is being added, the pH should be checked intermittently with pH paper. When the solution gets close to 7, Solution II should be added bit by bit (a few mls at a time). Hopefully by the time solution II is all used up, a reading of 7 (neutral pH) will be present. Up to this step, no heating of the reaction mixture has been called for as the reaction between the lactone and hydroxide is exothermic (it generates lots of heat). When the last few mls of Solution II are being added, the reaction mixture should be brought to a low simmer (180 to 200°F — just below boiling) over a stove and stirred thoroughly with a stainless steel/chrome-plated spoon. This will speed up the time it takes to complete the reaction and assure that there are no unreacted pockets of lactone and/or hydroxide left in the pot. **This small detail should not be skipped.***

Note: I have observed that it is possible to get different pH readings from different spots in the reaction mixture when this detail is not followed. I have also seen batches of GHB where a pH reading taken a few days after bottling the solution was no longer at a neutral pH of 7. In all likelihood, if this happens, a pH reading of below 7 will occur, which indicates there is still unreacted lactone in the solution. This itself is no big deal as it is easy to dump the whole mess back into the pot and add enough hydroxide until a pH of 7 is reached (see Step Six). Actually the lethal dose for the lactone in rats is higher than that for GHB itself and their effects on the CNS are identical. While a little residual lactone probably won't hurt anyone, it's not a good idea to substitute straight lactone for GHB — gamma-butyrolactone is harder on the gut and appears to be responsible for the headaches many users of home-brewed GHB complain about. Besides, if anyone thinks liquid GHB tastes like shit wait 'til they swig some lactone.

*WARNING!

If a pH of greater than 7 is reached, the home brewer has problems (see Step Seven to correct this). This is indicative of excess hydroxide in the solution and if there is a sufficient amount there, it will do major damage to internals. The cases of wanna-be home chemists who have hurt themselves taking their own GHB are probably a result of making this mistake.

- **Step Six:** If the pH < 7 the mixture should be heated to around 180 to 200 degrees and stirred with a stainless steel spoon (a plastic spoon should not be used!) to make sure no unreacted pockets of lactone/hydroxide are present in the pot. The pH should be checked again. And more hydroxide added as needed.
- **Step Seven:** If the pH > 7 too much hydroxide was added or the lactone was misweighed. There are two alternatives to correct this: more lactone should be added until the pH is brought back down to 7. Or, some hydrochloric acid can be added until a pH of 7 is reached.

NOTE: A trick that some people have used to get rid of the petroleum-like taste that residual lactone imparts to the GHB solution is to intentionally add slightly too much hydroxide to the mix to assure that there is no unreacted lactone left over and then bring the pH back to neutral with some hydrochloric acid. If a lot of hydrochloric acid is needed to do this, however, the GHB will taste quite salty.

- **Step Eight:** the amount of GHB produced = starting weight of the lactone from Step One × 1.46 grams sodium-GHB produced per gram of lactone used.
- **Step Nine:** concentration of GHB = the amount of GHB produced in Step Eight ÷ volume of the GHB solution (measure it). If the amount of water used to dissolve the hydroxide is kept to a minimum, the concentration should be in the one gram of GHB per one ml of solution range. Conveniently, the usual three gram dose just happens to fit in a 3cc syringe.

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HARD-HITTING DRUG FACT #2

from page 8

1*N* NaOH. This is refluxed for 10 minutes and 250ml of hot water is added. The solution is allowed to cool to room temperature and then refrigerated overnight. The trenbolone crystals are then filtered off, washed with water, and dried.

TERNBOLONE OXINE (II): Trenbolone (1.52g), sodium acetate (4.35g), hydroxylamine hydrochloride (1.95g), water (19.5ml), and ethanol (50ml) are refluxed for 3 hours and then cooled. The mixture is then diluted with water (250ml) and extracted with benzene (100ml). This is then washed with water (3 x 100ml) and dried over magnesium sulfate. Removal of solvent under vacuum affords probably an oil.

4,9,11-estratriene-3,17-dione-3-oxine (III): To Trenbolone oxine (1.7g) in 270ml toluene and 40ml cyclohexanone refluxing under dry conditions (caCl trap or Nitrogen) is added dropwise a solution of 2g aluminum isopropoxide in 140ml toluene with simultaneously distilling off the solvent until about half is gone. Then another 8ml cyclohexanone is added followed by 1g aluminum isopropoxide in 100ml toluene and distillation is continued for another half hour. After the solution is slightly cooled, 5ml of water is added followed by vigorous stirring for 15 minutes. The precipitated aluminum hydroxide is then removed by filtration and washed with toluene. The combined filtrate with washings are then evaporated to dryness under vacuum on a boiling water bath. The resulting solid is then slurried with 20ml of hexane and refrigerated for 3 hours. The crude precipitate is then filtered and washed with a little cold hexane and dried.

METHYLTRIENOLONE (IV): To 0.3g of (III) in 18ml benzene was added dropwise under nitrogen 90ml 1.64*N* methyl magnesium bromide in ether. After addition the solution was refluxed overnight on a steam bath. The cooled mixture was poured into 100ml of ice water and acidified with HCl acid to a pH of 3.5, refluxed for 2 hours, and refrigerated overnight. The crude (IV) is then filtered and washed with water. If desired, it could then be purified by recrystallization or chromatography.

Dan's DEVIANT DELIGHTS

Video review:
PLAYING WITH FIRE
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LIKE FATHER LIKE SON
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One of the newer IFBB professional body-builders, Bruce Patterson, from Canada, has done two hard-core gay porn videos. For those who think that Bruce somehow was forced into this, I'd like to point out that the earlier of the two videos, PLAYING WITH FIRE, was shot in October of 1995. The other video, LIKE FATHER LIKE SON, was shot over a year later, in November of 1996. Patterson uses the screen name of Chris Thunder.

In **PLAYING WITH FIRE**, Bruce plays a firefighter. Bruce is not in the best of shape, not being very lean. He also has a moderate amount of acne on this back and glutes. So what does Bruce do in the videos? Better tell you what he doesn't do. Bruce doesn't take it up the butt, but he does get his butthole munched a lot, and he does get a load shot into his mouth and he swallows. As gay porn stars go, Bruce isn't very animate, and he doesn't have the most interesting or a very large dick.

In **LIKE FATHER LIKE SON**, Bruce is a little tighter, and the acne is gone. But, unfortunately, after another year of heavy steroid use, his gonads are gone too. In his first video, his balls are small, but discernible. By the second video, he looks like a eunuch. It also doesn't help that he has some co-stars that have some truly huge dicks.

MAKING GHB from page 9

- **Step Ten:**
  A: If the concentration > 3 gm/ml or a more watered-down solution is preferred, water can be added using the formula: volume of water to add = [weight of the GHB from Step Eight ÷ desired concentration in gm/ml] – volume present.
  B: If the concentration < 3 gm/ml or a more concentrated solution is preferred. The home brewer should boil off enough water until the volume present = grams GHB from Step Eight, desired concentration in gm/ml. The solution should then be evaporated off over low simmer (just below boiling). Or some time could be saved and the weaker concentration could be used.

WORTHWHILE MODIFICATIONS

One of the problems with taking the sodium version of GHB is that every three gram dose also has around a gram of sodium with it. GHB also has the nasty habit of lowering blood levels of potassium (by forcing it into the body's cells) at the same time it makes the user pee. The "GHB pump" and more cut appearance many experience from taking GHB results from these two effects.

The downside is that the user is getting an unhealthy amount of sodium and screwing up their sodium/potassium balance. This change in appearance is only transient and will more than likely be followed by a "dry" feeling the next morning.

The solution to this is to substitute potassium hydroxide for some of the sodium hydroxide used in the reaction.

Since potassium hydroxide has a higher molecular weight than sodium hydroxide, a greater weight of potassium hydroxide is used in the reaction; the gram weight of lactone being used should be multiplied by .652 to calculate the potassium hydroxide needed in Step One. A straight potassium-GHB formulation can be used, but this can create problems of its own. Large doses of potassium are irritating to the intestines and can lead to other symptoms of potassium overload like cramping and irregular heartbeats. Besides, some sodium is needed to facilitate the transport of ionic substances like GHB across the intestinal wall. The best way to go is a rough 50/50 mix between sodium-GHB and potassium-GHB. To make this as easy as possible, the quantity of lactone being used should be divided by two. This number should then be used to calculate the required amount of sodium hydroxide and potassium hydroxide. The proportion of sodium to potassium could also be changed by altering the amounts of potassium hydroxide and sodium hydroxide used. But it's probably not worth the effort. Remember K-GHB is about 10% less potent gram for gram than its sodium counterpart because the potassium component constitutes a greater percentage of the total weight of the molecule then does sodium in Na-GHB.

MODIFICATION 1:
100% potassium-GHB solution

- **Step Two:** grams of potassium hydroxide used = grams of lactone used from Step One × 0.652 grams of potassium hydroxide per gram of lactone used.

MODIFICATION 2:
Rough 50 / 50 sodium-GHB / potassium-GHB solution

- **Step Two:**
  A: grams of sodium hydroxide used = grams of lactone used from Step One ÷ 2 × 0.465 grams of sodium hydroxide per gram of lactone used.
  B: grams of potassium hydroxide used = grams of lactone used from Step One ÷ 2 × 0.652 grams of potassium hydroxide per gram of lactone used.

MODIFICATIONS OF QUESTIONABLE VALUE

Other modifications to the GHB recipe include using hydroxides of calcium and magnesium in addition to, or instead of, the sodium and potassium. On the surface the calcium version seems to be one worth trying as many people could use the additional calcium, nutritionally speaking. In this case .430 grams of calcium hydroxide would be used per gram of lactone. This reaction would be a little more difficult to carry out as calcium hydroxide is not very soluble in water. The home brewer could probably get away with adding the calcium hydroxide straight to the lactone once a little water was added, but this reaction looks like it would be a difficult and tedious one, so I'd say don't even bother. Magnesium salts are known to work great as laxatives so a magnesium-GHB would probably have you rushing to the toilet. Oops!

For users who hate the taste of liquid GHB and demand a powder there are two ways to do this. The water can be evaporated off by setting the liquid in stove proof glass pans on top of a boiling pot of water or hot plate (or any other reasonably safe scenario that can be imagined — the

continued on page 12

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ISO-OPUS ERRATA ... *from page 2*

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—Lyle McDonald, CSCS
Fitness And Sports Training, Inc.
Nashville, TN 37212

ALPHA-2 *from page 6*

Androgen stack:

To the above stack add an aromatase inhibitor (one and a half cytadren taken in 3 divided dosages throughout the day is a cost effective formula) + a strong androgen such as Masteron. For muscle mass, keep your favorite anabolic stack.

In any case, take the clen and the yohimbine before working out on an empty stomach.

(Editor notes: Capoten is the most potent of the ACE inhibitors. Unfortunately, it has the most undesirable side effects. There are newer, more benign ACE inhibitors. However, the Alpha-2 down-regulation research has been done only on Capoten. We do not know if the newer drugs will have the same positive effect. For example, because of my kidney disease, Capoten would be a terrible choice for me, so I use Zestril instead. It seems to be reducing my lower body fat, but it would be interesting to see if there is any better improvement with Capoten.)

from the desk of
Daniel Duchaine, PhD

Q&A

For Q&A questions send to: Dan Duchaine's Dirty Dieting Newsletter, 2533 N. Carson St., #2538, Carson City, NV 89706.

Q I NEED SOME PRICES OF THE BLACK MARKET STEROIDS AVAILABLE. CAN YOU GIVE ME AN IDEA WHAT I SHOULD BE PAYING SO I DON'T GET RIPPED OFF? AND HOW ABOUT SOME PIX?

A Okay. For 10cc injectables (these are the International Pharmaceutical ones).

	Average Retail	High Retail
MDV (multi-dose-vial)		
Nandrolone Decanoate (200mg/ml)	150	175
Testosterone Enanathate (250mg/ml)	80	110
Testosterone Cypionate (200mg/ml)	80	110
Stanozolol (50mg/ml)	80	90
Methandrostenolone (25mg/ml)	25	50
SUA (single-use-ampules)		
Omnadren (250mg/ml)	16	25
Primobolan Depot (100mg/ml)	8	25
Parabolan (76mg/ml)	20	28
Nandrolone Decanoate (200mg/ml)	14	20
Note: The Nandrolone is an IP product. Others are "legit."		
Injectables, various		
HCG 10,000 IU 10ml	75	90
Somatagen 4 IU 1m	70	125
Tablets, 100 tabs		
Methandrostenolone 5mg	65	150
(white is the IP, pink is the Thai Anabol)		
Stanazolol 5mg (IP)	80	150
Oxymetholone 50mg (IP)	180	300
Oxandrolone 2.5mg (SPA)	80	160
Tablets, various		
Primobolan S 25mg 50 tabs	75	110
Nolvadex 10mg 100 tabs	150	250
Triacana	70	110

MAKING GHB *from page 10*

heat should just be kept below 200°F or so.) The second option entails substituting alcohol or an alcohol/water mix for the water when dissolving the hydroxide. Everclear or even 100 proof vodka works fine for this step.

WARNING!

Denatured alcohol or any other alcohol like methyl or isopropyl should not be used, as they can leave behind some nasty little trace impurities.

The magic of this modification is that GHB is not very soluble in alcohol, so it tends to spontaneously crystallize out of the solution. Additionally the alcohol evaporates much more readily than straight water. The main disadvantage of using alcohol is that it will take more alcohol or alcohol/water to dissolve the hydroxide used (about 3.5 mls per gram of sodium or potassium hydroxide) and the vapors given off during evaporation are flammable and intoxicating.

FINAL WORDS

Due to the publicity GHB is currently getting and the rumors of the impending ban on gamma-butyrolactone, there has been a rash of would-be suppliers offering lactone for sale at ridiculously high prices. The following prices were pulled off the Internet newsgroup alt.drugs.chemistry:

BondTech Corp. offers kits to make GHB (potassium and sodium based) from ACR Research Lab, prices effective January 1, 1997 are as follows: retail US$175, wholesale US$125 (3 kits or more) for 180 to 200 grams of GHB (Bullshit!).

The following price was from tfreel3514@aol.com: 4 ounces (133 grams) of 98% pure gamma-butyrolactone for $35 plus $3.75 shipping and packaging.

Chemical Resale of Santa Barbara, 6 Harbor Way Suite #171, Santa Barbara, CA 93109-2353 wirehead@sb.net: prices for gamma-butyrolactone are: 500 grams $90 and 2,500 grams $310.

These prices are a fucking rip-off! If the home brewer looks hard (no, I'm not going to say where) they can find gamma-butyrolactone in the $15 range for a pint (approximately 535 grams) and around $60 for a gallon (approximately 4,200 grams). The sodium hydroxide and potassium hydroxide shouldn't cost more than $20 for 500 grams of the reagent grade. So open the yellow pages and save some money. DD

Number 2 • May 1997

Dan Duchaine's
DIRTY DIETING NEWSLETTER
MILITANT MUSCLE GROWTH AND FAST FAT LOSS

UPDATES FROM THE UNDERGROUND

(Editor's Note: One of our subscribers is a steroid dealer, based in the UK. His letter has so much valuable information, I thought it best to reproduce it in its entirety.)

Editor:

Your readers might want to know about the closure of the mail-order Greek pharmacies. Mougios and Skouvara were the two biggest players in the mail-order 'roid game, but it all came to an end at the beginning of '97.

Actually Skouvara are not going under as they chose to heed certain warnings, and they now request a valid prescription (which, according to Greek law, should have been the case in the first place).

continued on page 14

Contributing Editor's Note:

We apologize for the delay of this issue. We're working hard to provide you with information like none other on body manipulation, and getting the various articles together took a bit longer than anticipated. This issue, you'll note, has additional pages and a new column. We hope it's worth the wait. If you could, please take a few moments to fill out and send in the reader survey located on the inside back cover. We appreciate your comments. Again, sorry for the delay.

— C. JEFFERSON

IN THIS ISSUE

Glycerol As A Mild Diuretic

by Oliver Starr

Glycerol is well known for its action as an osmotic diuretic. In fact, due to its rapid effect on body water stores, glycerol has long been used to reduce intra-ocular pressure from glaucoma and cerebral pressure from head trauma. This is important since it's for this reason that glycerol has come under scrutiny as a potential aid for the competitive bodybuilder.

Dr. Paul Montner, one of this country's foremost authorities on glycerol and human hydration, was cautious in his assessment of the efficacy of glycerol for this purpose. Saying simply that he had "neither seen nor conducted any research on this topic and, therefore, would not recommend the use of glycerol in this regard."

Nevertheless, my discussion with him did confirm several of my suspicions. It also enlightened me to the fact that he felt his research confirmed glycerol isn't so much a diuretic, but a body water re-partitioning agent. While this might sound like the end of the story as far as glycerol's applicability to bodybuilders, that isn't the case.

As stated above, glycerol has been used medically to move fluid out of the brain and ocular compartments. It does this because glycerol doesn't easily transgress the blood brain barrier. It's apparent from the literature that glycerol in fact diffuses through cell membranes at varying rates and seems to enter plasma preferentially. This is why it can help bodybuilders get better cuts and possibly more vascular in appearance as well.

When glycerol is ingested it's rapidly absorbed through the intestinal wall. It quickly begins to diffuse through the tissues of the body. Since it moves more quickly into plasma than any other body compartment, glycerol causes an osmotic shift which results in fluid (i.e., body water) moving away from areas such as subcutaneous storage and into the plasma. This can have two desired effects. First, it will enhance the shredded look by pulling water out of the skin. Second, by increasing plasma volume, it may also improve vascularity. That's the theory.

As mentioned at the outset, this entire strategy is entirely experimental, so if someone elected to try this, he/she would be stepping into unresearched and uncharted territory.

The extremely sweet taste of glycerol is nauseating so it has to be mixed in some small volume of palatable liquid — orange juice works well and has been used most frequently during research studies. I'd say to use as little liquid as possible with the glycerol — 8 to 12 ounces should work for most.

Personally, I'd start with a dosage of around 1 to 1.5 grams of glycerol per kilogram body weight and see (the only way you'll be able to assess if this is working is

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Militant Muscle Growth and Fast Fat Loss

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GLYCEROL *from page 1*

by appearance because glycerol doesn't stimulate urinary diuresis) how I look. It will take up to 90 minutes for the glycerol to achieve peak plasma levels. The osmotic shift does occur fairly rapidly so one can expect to begin to see some effect within about 60 minutes.

Exceeding 3 grams of glycerol per kilogram of body weight may result in nausea and/or headaches. So it's wise to experiment with this practice at least once prior to attempting it before a show. This will allow for a determination of what dosage works best for different people (if it works at all). It also helps to determine when the peak water redistribution takes place so intake timing can be planned accordingly.

As far as measuring glycerol, one measuring tablespoon (no soup spoon — use a real kitchen measure) is equal to 25 grams of glycerol. It doesn't have to be exactly precise to the gram, but using 1/8 to 1 tablespoon increments allows for fairly accurate measurements without using a gram scale. I just discovered a good way to measure it — one of those children's medicine dosing syringes that have both tablespoon and "ml" lines — neat, clean and accurate. It's by far the best system I've discovered.

Interestingly, cyclists and other endurance athletes use glycerol with large quantities of water to achieve the exact opposite effect — that of hyperhydration. As a result, glycerol is available prepackaged through some high end cycling stores. I know this because the product they're selling — Glycerate™ — is a product I created. However, you should never buy this product. Why? Because for our purposes it's a rip off!

Glycerol is a commodity item. It can be found at even marginally complete pharmacies from coast to coast. It's regularly used as a moisturizer and as an emollient so it's fairly common. Vegetable glycerine at 99.7% purity is best, though 99.5% will work too. If it's not on the shelf, I ask the pharmacist for it. Oftentimes they have it behind the counter. If he inquires as to its desired use, I tell him I'm going to use it as a moisturizer. Some pharmacists I've encountered are reluctant to sell it to people who openly admit that they intend to use it for oral consumption.

It costs between $12 and $17 for a pint or more. This is enough to last several years. However, if not kept tightly covered, glycerol will absorb water from the air and become less effective. If for some reason glycerol can't be found in a local pharmacy it can be ordered from any of the chemical manufacturers or re-sellers. The only problem is they often only sell glycerol in 55 gallon drums. That's more than enough to supply every bodybuilder in California. One solution is to see about getting a "sample" which would generally be between 8 and 16 ounces. This requires a bit of deception, since they won't just send samples out to anyone.

WARNING!

Though glycerol has extremely low toxicity, as with anything, willful misuse can prove damaging. **DO NOT, I REPEAT, DO NOT INJECT GLYCEROL INTO YOUR BODY!** Injecting glycerol causes such a rapid osmotic shift that it will result in hemolysis (red blood cell bursting) and this will lead to renal failure and ultimately, even death. For a bodybuilding contest, glycerol should be used no more than once every 24 hours. There is no additive effect and side effects like headache and loss of equilibrium would be much more likely. If your show is the next day after the prejudging, a glycerol mixture ninety minutes before going on stage can be used, but only at about .75gm/kg.

And finally, since we haven't yet totally figured this out ourselves, we encourage comments as to experiences. So keep us posted with any results.

(Editor's Note: I bought a 4oz. bottle of Glycerin at my local drug store for about $3. It was the HUMCO (Texarkana, TX 75501) brand, and was sold as a "skin protectant." Most glycerins are sold with rose water added — something you don't want.) DD

STEROIDOLOGY

DIETING PARADOX REVISITED

by Michalovich Dharkan Greutstein (aka Dharkham)

(Editor's Note: Dharkham's submission is a response to my postulation that downregulation of T3 might be avoided by supplementing the diet with non-carbohydrate ATP substrates, most notably: pyruvate, taurine, and medium chain triglycerides. After I had finished my research, I was alerted to a late 1996 study that used commercial phosphate supplement [Redusan] as a liver ATP substrate. Redusan is: 537mg calcium phosphate, 107mg potassium phosphate, and 25mg sodium phosphate. The dosage was two Redusan, three times a day. This seems a more workable [and economical] solution than 36 grams of various pyruvate salts.)

You've probably read the recent *Muscle Media*® article about the thyroid problems induced by prolonged diets. It described how a diet will eventually stop working and how to deal with this problem. I would like to expand on the article. By solving the dieting paradox, Dan claims that "low-calorie diets never have to stop working." Is this statement correct?

Let's clarify that statement. I think that Dan would agree: if diets stop working, it's simply to protect our life. A diet which doesn't stop working will eventually bring you closer to my two bodybuilding heroes: Momo Benazzizza and Andreas Munzer. Eventually a diet has to stop working. My goal is to postpone the moment your diet is going to stop working so you can get closer to the body you want. You will not be able to reach your goal if you don't clearly understand mine. I am not promising that your diet will be easier or faster. I'm simply going to provide enough information so that you're able to go beyond what you did on your own.

Dan points out that the main culprit is the shrinking T3 levels. Of course, this is not the only reason why a diet stops working. *(Editor's Note: See my comments on UCP-2s at the end.)* It's rather simple to demonstrate. If low T3 was the key, adding Cytomel would overcome this sticking point and any obese person on the planet would eventually become lean. As pointed out in Dan's article, adding T3 is a messy solution at best. True, it will increase body temperature and hence your daily energy expenditure. You might even lose some fat. But if you try to artificially maintain a normal T3 level, you'll eventually sacrifice a portion of your muscle mass. There's no absolute guarantee that Cytomel will solve all your problems. So, T3 level is not the sole determinant of your chances of success.

Even more puzzling, many researches have found no correlation between thyroid output (or T3 level) and the fall in BMR (basal metabolic rate — a way of measuring daily caloric expenditure) associated with a low calorie diet. Recent research even points out that among several groups, the group which lost the most fat and the least amount of muscle had the lowest T3 levels! Other researchers did find a relationship between T3 level and fat loss. But only in the short run.

Only genetic factors, such as fat cell number, can help predict how much fat someone is going to lose in the long run. Thyroid hormones have no (detected) influence on how much fat you will eventually lose. In other words, if you want to get lean and stay that way you'll have to change your genetics, not your thyroid secretion. That's the bad news. The good news is it's not that hard to change your genetics but that's beyond the scope of this article. Nevertheless, I'll concentrate here on how to fix the thyroid problems occurring during a diet. Before getting into it, there's another point I disagree with Dan on.

Dan claims that all the thyroid problems are caused by a reduced T4 transport in the liver. To remind you, the thyroid gland produces mostly T4, an inactive form of thyroid hormones. T4 has to be transformed into T3 to produce its effects. The enzyme called 5'-deiodinase is responsible for the transformation of the inactive T4 into the active T3. This enzyme is found mostly, but not only, in the liver. By reducing T4 transport into the liver, T4 cannot reach this enzyme in significant amounts, so less T3 is made. I disagree.

Even the authors of this theory didn't claim it was the main cause of low T3 while dieting. They only say it's one of the several mechanisms involved. I believe the reduction of 5'-deiodinase activity is a very big problem while dieting. To be honest, no one knows exactly the respective participation of each pathway on the diet's T3 reduction. This is easy to explain; not many people are ready to sacrifice their livers so that researchers can look into it. Of course, we do have rat livers, but things are a bit different between rats and humans. *(Editor's Note: Rodents rely heavily on BAT to regulate*

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HARD-HITTING DRUG FACT #3

Making Testosterone From Androstenedione

(Hypothetically of course!)

Patrick Arnold

(Editor's Note: In the US, this chemical procedure is illegal. Additionally, the by-products of the various androstenediols may fall under the Federal Analogue Act, which schedules derivatives of any DEA-controlled drug [in this case testosterone] into a Control I substance. So in effect, this procedure outlined is an hypothetical exercise designed to show a possible conversion of androstenedione to testosterone.)

The procedure outlined below won't convert 100% of androstenedione to testosterone. But it will convert at least 60% of it, if the procedure is not screwed up too badly. The other percentage of finished product will contain a small amount of unreacted androstenedione and a larger amount of a (epimeric) mixture of 3,17-androstenediols, which, fortunately, are safe and considerably anabolic compounds in their own right.

MATERIALS NEEDED

Androstenedione (powder)
Methanol (wood alcohol)
Sodium Borohydride
(sodium tetrahydroborate)
Acetic Acid (ethanoic acid)
Distilled water
Litmus paper
Thermometer (Fahrenheit)
Also: Beaker or glass container for the reaction, a pot for salt water ice bath, a way to stir (i.e. spoon), filter, eyedropper (1 or 2cc size).

PROCEDURE

10 grams of androstenedione is dissolved in 400ml methanol and cooled to 32° Fahrenheit in a salt/ice bath (similar to chilling home-made ice cream).

2.5g sodium borohydride is added while the solution is stirred.

The solution is continually stirred for 45 minutes while the temperature is maintained as close to 32° Fahrenheit as possible.

After 45 minutes, acetic acid is added to the solution, while stirring, in increments of approximately 2ml at a time (hydrogen gas will evolve).

continued on page 5

from the desk of
Daniel Duchaine, PhD

PARADOX *from page 3*

body temperature.) But the scientists do have some indirect means of guessing which pathways are the most important to explain T3 problems. The researchers trying to quantify the participation of those respective pathways were not impressed by the transport theory.

The trouble is that dieting research is done usually short term with obese people. They don't react as other (non-obese) people do, as far as the thyroid axis is concerned. Things are very different in bodybuilder-like situations. Fortunately, I was able to look at how bodybuilding dieters responded to different drugs and different diets. So I've gotten a better understanding of what's going on during a long-term diet.

First of all, we know that thyroid troubles that occur during a diet can be somewhat fixed by taking either ephedrine or clenbuterol. Those drugs will increase the activity of the 5'-deiodinase enzyme in the liver. We know that reduced 5'-deiodinase activity is involved in the dieting-induced thyroid problems. Does that mean that thyroid transport in the liver has no role? No, on the contrary, it is very important and that is where I like to expand on Dan's article.

Dan points out in his article that adding T3 (Cytomel) is a solution, but not a good one. I always wondered why it was that bad of a solution. Well, I guess Dan gave us the clues to figure it out now. If reduced 5'-deiodinase activity was the only thyroid problem, adding synthetic T3 would be the perfect solution since we wouldn't have to care about that enzyme anymore. We'd have all that active T3 available to get leaner. The same reasoning would apply if reduced T4 transport was the explanation. No problem if T4 can't circulate in the liver either. With synthetic T3 we can lose body fat, however, we also can say goodbye to our muscle mass if the improper dosage is used. Why?

There are two ways of altering muscle mass. One is to increase or to decrease the rate of protein synthesis (anabolism). The second is to increase or decrease the rate of protein degradation (catabolism). True, a diet will increase catabolism, but this isn't all that bad. The real trouble is a diet will also reduce the anabolic drive. In other words, a diet will increase catabolism and prevent any increased rate of protein synthesis. If the latter was able to increase freely, as it usually does when anabolism is enhanced, we wouldn't lose any muscle mass while on a diet. No gains, but no loses either.

Several mechanisms are involved. The testosterone level will shrink. Unless oral leptin is available, it will be very hard to fix. Of course, taking steroids will solve this problem, but this is illegal in the US, and I'm assuming that most dieters will want to avoid this solution. Furthermore, use of steroids tends to reduce thyroid hormone level. Another obvious reason for muscle loss is from reduced insulin levels. Taking insulin will fix this problem. But it will also force the dieter to use other drugs which he might not be familiar with to combat the anti-lipolytic effects of insulin. So, again, it's not a good solution. Another reason for the negative nitrogen balance is IGF-1 levels are going down the drain. This is not normal. GH is the main stimulator of IGF-1 secretion and we know that GH level is going up while on a diet.

Do you know what a syllogism is? A syllogism is a wrong deduction coming from two correct statements. For example: whatever is rare is expensive. Cars are expensive. So cars are rare. Of course, this is not true. Bodybuilding magazines are full of syllogisms. Here is another classical one: GH is a strong anabolic hormone. GH level goes up during starvation. So far, so good. Now the syllogism: in order to get huge, thanks to the GH anabolic properties, you have to starve yourself. Of course, there is something wrong here. GH is indeed an anabolic hormone, but not while on a diet.

In order to be anabolic, GH has to be changed into IGF-1. This transformation takes place mostly, but not exclusively, in the liver. In order to stimulate IGF secretion, GH has to bind the GH receptors located on the liver. Unfortunately, two things happen to GH receptors on the liver while on a diet: 1) the number of GH receptors is reduced, and 2) available GH receptor activity is impaired.

This second problem is mostly caused by a shortage of high-quality proteins. It takes place even in bodybuilders. It is sad but true. Low-quality proteins are the dieters' number one choice. Look at how many dieters rely on tuna, fish or turkey proteins. To make a long story short, you should go with the very best proteins while on a diet. But, taking a high quality protein will not solve our anabolic problems if you have no more GH receptors in your liver.

(Editor's Note: Perhaps we should explore, in a future issue, what the "very best" protein would be on a low-calorie diet. I will not simply assume that whey protein is the ideal. It may very well be, but perhaps not. A high-quality protein may not supply the ideal amino acids.)

Most of you are probably aware of Dan's recommendations for GH users. GH works best if used along with insulin, T3 and of course anabolic steroids. Why? Because all those drugs will up-regulate GH receptors on the liver. But insulin, T3 and testosterone are all low during a diet. It's no wonder why our own GH has no anabolic property even though its secretion is high.

T3 alone is a potent up-regulator of GH receptors in the liver. So, in theory, taking Cytomel during a diet will:

- **Enhance fat burning**
- **Up-regulate GH receptors in the liver and allow GH to become a potent anabolic hormone**

True, T3 tends to be catabolic especially during a diet, but we're talking replacement only here. The big increase in IGF-1, which would follow Cytomel administration, should easily overcome any catabolic effect caused by T3. As pointed out earlier, Cytomel might increase fat loss but it has absolutely no anabolic properties. To up-regulate GH receptors in the liver, T3 has to be transported inside the liver and (everybody sing along ...) T3 TRANSPORT IN THE LIVER IS IMPAIRED BY THE DIET.

By following Dan's advice on restoring ATP level, you should be able to both restore (but not completely) the T3 level and improve (but not fix) the lack of GH anabolic properties. This is one more reason to follow Dan's advice, but I would also like to point out some further suggestions. I'll assume most readers will not follow this next suggestion but it will give us a better understanding of what is going wrong while on a diet. The best solution is to inject GH while on a diet.

Didn't I say GH was not that anabolic while on a diet? Well, I was talking about your own naturally-produced GH. Injectable GH is completely different from your own GH. It causes a huge elevation of GH in your blood. And this elevation will last longer than the natural elevations occurring at regular intervals throughout the day. The body will react by increasing the secretion of insulin. This insulin will not stop fat loss because elevated GH will oppose any bad effects of insulin on adipose tissue. This insulin will act on the liver to up-regulate GH receptors. Furthermore, insulin is able to up-regulate 5'-deiodinase activity. Insulin's effect will be potentiated by GH which is acting on its newly available liver receptors, and will synergize with insulin to further increase

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PARADOX *from page 4*

5'-deiodinase activity. As a result, normal T3 formation will be restored. It's possible insulin also restores normal T4 and T3 transports in the liver, allowing this newly formed T3 to further up-regulate liver GH receptors. In other words, GH injections will restore proper T3 secretion and so further enhance lipolysis while restoring normal anabolic functions, thanks to both insulin and IGF-1.

I would like to raise another interesting point. There's a very close relationship between liver GH receptor level and 5'-deiodinase activity. It could seem normal, as GH is acting on GH receptors, and up-regulating 5'-deiodinase activity. But I am wondering whether 5'-deiodinase could somehow regulate GH receptor level? That would mean the body would use the 5'-deiodinase level to gauge how strong anabolism should be. Starvation, by reducing the 5'-deiodinase activity, could reduce the anabolic drive. Overfeeding, which up-regulates 5'-deiodinase activity might indirectly increase IGF-1 production. If this speculation is correct, it would provide another reason why taking Cytomel will not really solve our problems while on a diet. Furthermore, it would mean that the whole thyroid axis, not just T4 or T3 transport, will have to be fixed while on a diet.

I concur with Dan's advice on restoring liver ATP levels primarily by using phosphate supplementation. You might want to add HCA and carnitine to it. If what's said about this stack is true, it also might help to maintain the ATP level in the liver. Glucose, and not triglyceride, is a better ATP substrate in the liver. I've never felt good when using HCA because of stomach problems. But if it works for you, fine. Just don't forget that a far higher dosage of HCA is required than what is recommended by the manufacturer.

This said, I would like to expand from here. Using ephedrine or clenbuterol, or any Beta 2 agonist, will partially restore the thyroid axis while dieting. Clenbuterol is best, but restricted in the US, so most people will have to make do with ephedrine. It doesn't mean that ephedrine is bad. In fact, it has been shown to enhance fat loss while preserving muscle mass during a diet.

I always wondered how ephedrine could spare muscle mass. Its main effect is to enhance the release of norepinephrine (NE). Once in the blood, NE binds receptors (called Beta-adrenoceptors) on muscle cells. Some people claim that NE is an anti-catabolic hormone. But as far as I am concerned, this direct muscle sparing effect of NE is far from obvious. Remember that muscle cells are composed of several different kinds of amino acids. Whenever NE acts on skeletal muscle it blocks the release of some amino acids, meaning it is anti-catabolic. However, it accelerates the release of some other amino acids which means it enhances catabolism. So, NE is both anti-catabolic and catabolic depending on the kinds of amino acids you refer to. It's hard to predict whether NE will enhance muscle mass or reduce it.

Some of the positive effects of NE are indirect. For example, we know that by releasing fatty acids from fat cells NE provides energy, which spares amino acids. But muscle, just like adipose tissue, is a source of energy while on a diet. Your body can use either fat or muscle calories to make up for the energy deficit caused by the diet. It's a fact that the body uses the two sources together. Not determined is how much of each is going to be used. When one has plenty of fat in the blood (due to lipolysis), the body will tend to use mostly fat and so those fatty acids will spare muscle's amino acids. This is good. When the level of fat in the blood is low, the body will use mostly amino acids as energy. This is really bad for two reasons: 1) your lean body mass will shrink; and, 2) those amino acids will spare our fat reserves.

This indirect effect of NE could at least partially explain ephedrine's muscle sparing effect. But if we use our newly acquired knowledge, part of the muscle-sparing effects of ephedrine could be mediated by the partial restoration of the 5'-deiodinase and T3 secretion and (indirectly) by the up-regulation of the GH receptors in the liver. If true, ephedrine effects should be boosted by the supplements aimed at increasing liver ATP level and hepatic T3 transport. Again, this is a speculation based on theory, not scientific proof.

As far as anabolism is concerned, clenbuterol is a better choice because it's more specific for the still-unspecified anabolic receptors located on muscle cells. Clenbuterol has roughly the same effects on the thyroid axis as ephedrine. This is probably why it has been shown clenbuterol enhances GH induced IGF-1 formation.

Yohimbine is also thought by some scientists to increase thyroid hormone secretions by blocking Alpha-2 receptors located on the thyroid gland. This is not proven in humans, but yohimbine is cheap and increases fat loss, so it has its place.

Now that we have our supplements, the next issue to explore is the timing of use.

continued on page 6

HARD-HITTING DRUG FACT #3

from page 3

WARNING!

Hydrogen gas can ignite from flame or spark.

After each addition of acetic acid, the pH of the solution should be checked.

When the pH just begins to turn acidic on the litmus paper, then the addition of acetic acid should be stopped.

The methanol solution is now concentrated by evaporation until the volume is around 50ml.

WARNING!

Hydrogen gas may still be present; it can ignite from flame or spark.

This concentrated methanol solution is then mixed with 700ml water.

The cloudy precipitate that forms is filtered off and the filter cake is washed extensively (several times) with water.

The filter cake can be air (sun) dried for several days or dried in an oven for several hours at a temperature no higher than 150° Fahrenheit.

The finished product, a fine white powder, should contain approximately 60 to 80% testosterone, with lesser amounts of unreacted androstenedione and a mixture of 3-alpha, 17-beta, and 3-beta, 17-beta androstenediols.

What should be done with this (now highly illegal) stuff once the procedure is finished?

Since it is highly illegal I would advocate getting rid of it.

Yet if someone wanted to take the risk and decided to use it, they would probably wonder if it should be taken orally or what?

I would not suggest that anyone take it orally because the purpose of the experiment would be defeated. Since testosterone is mostly deactivated in the gut, the ingestion of the finished product would be worthless. Instead, there are better alternatives, such as making sublingual liquid by dissolving the product in a solvent such as propyleneglycol and/or ethanol. If this is done, I suggest aiming for a concentration of around 30mg/ml and taking one-third of a milliliter (about 33 insulin IUs) under the tongue as a single dose (repeated throughout the day as often as wished by the user). Another alternative is to make a transdermal DMSO solution (messy and stinky). A third course of action (for the bold ones) is to make an injectable solution. There is a fine art to making such solutions (oil- and aqueous-based) and it is beyond the scope of this article for me to go into this. Perhaps that will make a future installment if we have a response to do so. DD

from the desk of

Daniel Duchaine, PhD

PARADOX *from page 5*

It seems logical to introduce them when your diet stops working. Or is it?

It's not truly the diet which stops working. It's your body's response, which fights the diet more and more efficiently as time goes by. It is crucial to understand this point. Saying that you failed to lose weight because your diet stopped working is a mistake. You're the mistake. Just like when you're bench pressing ... as the number of reps goes up the weight is feeling heavier and heavier. The weight has nothing to do with the fact that you failed to do another rep. The weight is still the same. It's YOUR muscles which are weaker. You're the one at fault. Your body and part of your brain is fighting another part of your brain.

I could point out many pathways the body uses to fight a diet. For example, within a few days leptin production is reduced. You feel angry and your daily energy expenditure is reduced. Of course, it's likely that some but not all of the effects of leptin are mediated by T3. But bringing T3 levels back to normal will not restore normal leptin levels. The point is, we face many feedback mechanisms. Reduction of T3 levels is only one of those mechanisms, even if it seems to have a key role. Restoring normal T3 secretion is not going to solve all our dieting problems. But it does solve some of them. The body will react to this restoration by telling you, you won a battle ... but you didn't win the war. And it's going to accentuate other feedback mechanisms in order to minimize your fat loss. What should you do?

When you start a weight-loss program, any type of diet will work. We want to take advantage of this situation. As long as you improve the protein quality — you will be fine — even with a junk food diet. *(Editor's Note: Perhaps better-off with junk food, if the carbohydrate sources are high-glycemic ones, which spike insulin secretion.)* **When your fat loss rate seems to slow down it's time to improve your diet and strictly adhere to it.**

The next step is to monitor your body temperature. As Dan pointed out, it's a rough but simple indicator of how your body is handling the diet. When your morning body temperature is reduced, it means that your body is starting to fight the diet. By the way, it's already too late to do anything. You need to act before this fall of temperature occurs. Therefore, only past experience can help you on that point.

Once you figure out when your body is going to start to fight the diet, take a little bit of ephedrine and increase your food intake slightly. Please note that when I am referring to ephedrine, I mean ephedrine and caffeine. I also assume you're using Dan's stack for restoring ATP level from the start.

Your body temperature should go up and so will your daily caloric expenditure. So, you'll keep on getting leaner even though you're eating more. How much more? It all depends on how you react to ephedrine. Some people seem to be insensitive to ephedrine, others react too much to it. In both cases, you're in trouble. You might want to consider the use of a Beta-agonist such as salbutamol or clenbuterol.

If you react well, try to eat 200-300 more calories a day (probably less for women). You should determine it according to what you see in the mirror. If it seems that increasing your food intake that much is stopping the fat burning process, too bad. You'll know it for the next time.

Eventually your body temperature will start to decrease again. Ideally, you want to react a little bit before that fall by increasing the ephedrine dosage. It's even better to add 30 minutes of aerobic along with the ephedrine. But please, do so only if you feel your muscle mass is not affected by the aerobics. Do the aerobics at maximum intensity on an empty stomach (except for the ephedrine) first thing in the morning.

It's also time to introduce the yohimbine. The big issue is whether or not it's time for some Cytomel. If you're able to get some, you might want to try it. If the ATP level in your liver is normal, you won't lose much muscle mass. But taking Cytomel will eventually deplete your liver of ATP no matter what natural supplements you're using. So, after a while, you are going to lose muscle. Thus, T3 replacement while on a diet should be limited in both time and dosage.

Whatever way you choose (natural or not), eventually, your body temperature will fall once more. It indicates your body is fighting very hard. You can choose to face it. But unless you're using the solution of injectable growth hormone, it's time for a break for both the low calorie diet and the ephedrine and yohimbine (and Cytomel). Increase your caloric intake a lot for a day and a half (eat mostly carbs). Then adjust your caloric intake to your old maintenance caloric intake with a little more aerobics (like 45-60 minutes a day). You can stop the aerobics after a few days.

Eating more from time to time is not only good to avoid fighting your body, it's also important to increase your muscle mass. This will not be pure muscle mass. By starving your muscles and then giving them plenty of carbs, you're going to load them with glycogen. This is also true of your liver glycogen stores, which will "hypertrophy." In doing so, it's going to take longer and longer for your liver to get depleted of its ATP while on a diet.

(Editor's Note: Muscle glycogen is best replenished with maltodextrin and a spectrum of minerals. Hepatic glycogen is best replenished with maltodextrins and protein. Alternatively, you could use a straight malt extract, which has everything you need already in it.)

Of course, once you feel like it after two or three weeks, you can resume your diet and reduce your bodyfat percentage a little bit more. And by the time you have gone through three or four cycles, I am going to tell you how to change your genetics and lose your last bit of bodyfat WITHOUT any diet.

To sum up, I concentrated on the diet induced thyroid problems. I do not think the solution here will allow you to diet forever without hitting a sticking point. But it will postpone that moment. Furthermore, I added one more reason why you want to prevent the ATP fall in the liver. DD

COMMENTS ON DIETING PARADOX

DRD — Additional Comments:

We should discuss the impact of the newly isolated uncoupling proteins in skeletal muscle and adipose tissue. The researchers feel that at least 40% of the thermogenic action of T3 is through this futile energy cycle. Perhaps the reduction of IGF-1 might be affecting (negatively) these uncoupling proteins. And we now want to know which substances stimulate UCP-2, which are resistant to most of the stimuli that activate UCP-1 in brown fat. Obviously, unsaturated fatty acids, having unstable proton bonds, affect all uncoupling proteins, as protons are lost between the ADP to ATP energy cycle.

MDG — Reply:

UCP-2 existence was just discovered. But recent mice studies revealed that animals lacking norepinephrine, epinephrine (so unable to activate UCP-1, which is responsible for most of the thermogenic effects of Brown Adipose Tissue) still experience a big elevation in metabolic rate as a result of overfeeding. This elevation was independent of any change in thyroid hormone level, UCP-2 activation or shivering. It stresses the fact we are still missing a major point as far as metabolic rate regulation is concerned. DD

PART III

ISO-OPUS ERRATA

RE: ALTERED THYROID ACTIVITY WHILE DIETING

LYLE MCDONALD

As has been previously discussed, the problem with all reduced calorie diets (whether high carb, Isocaloric or Bodyopus) is that they all stop working. This appears to be due in part by the lowering of thyroid levels.

Thermogenic agents like clenbuterol and DNP hasten this reduction in conversion due to the direct effects they have on the thyroid converting enzyme. The simple (but temporary) solution is to self-medicate with Cytomel (T3) or Triac (pseudo-T3). But using exogenous thyroid hormone has its own problems, since too much shuts down the thyroid gland leaving one in a worse state when they come off the diet. Alternatively, you can cut more calories, but this just causes more muscle loss. In the end, all of these strategies are only temporary and don't fix the problem.

So, the only real practical solution to the inevitable fat loss plateau is to simply come off your diet for some period of time (five days to two weeks) to allow metabolism, thyroid, etc., to up-regulate. Dan has suggested mini-cycles for years where you alternate periods of over and underfeeding to keep bodyfat at reasonable levels (10% for men and 12% for women) while stair stepping up in lean body mass.

More recently, an entire dietary approach (the ABCDE diet presented in *Muscle Media*®) has been proposed that uses very short two week cycles of acute calorie cycling in an attempt to force anabolism (with some fat gain) during the overfeeding phase. You then swing back into fat loss mode while keeping muscle loss to a minimum.

Admittedly, during overfeeding some very nice things happen. Certainly insulin comes up and so does IGF-1, thyroid, metabolic rate, testosterone and nitrogen retention (all of which promote muscle gain). One study found an increase of 4 lbs of lean body mass during three weeks of overfeeding in sedentary men (Forbes et. al., 1989). But, the individuals (who were not training) also gained 5.5 lbs of fat at the same time.

Another study using moderately active individuals found a greater gain in lean body mass versus fat during 12 days of overfeeding (Jebb et. al, 1996). **But, in all cases of overfeeding, some fat is gained.**

And, whether you're a bodybuilder or just a dieter, a fat gain — no matter how small — is distressing. So, it would be nice to find a way to at least minimize the inevitable fat gain that occurs. However, to determine how this is best accomplished, we have to delve into the pathways through which fat gain occurs.

The two main causes of fat gain during overfeeding are:

#1
STORAGE OF DIETARY FAT DUE TO INHIBITION OF FAT OXIDATION FROM HIGH CARBOHYDRATE INTAKE

When excess carbs are consumed, the body cranks up carbohydrate oxidation to compensate, but this means that less fat is used to provide energy. Additionally, all that insulin will effectively block fat mobilization from the fat cells as well as stimulate fat uptake into adipose tissue.

There really isn't much we can do about this one except for the use of an over the counter (or non) thermogenic agent. Obviously, DNP would prevent any fat gain during periods of overfeeding but most would be smart not to use it.

A short cycle of clenbuterol would probably help since it's known to re-partition calories away from fat cells and towards muscle. But this might cause problems with thyroid up-regulation. Even the good ole' ephedrine-caffeine-aspirin stack would be helpful during this phase to minimize fat gain.

Also, keep dietary fat to a minimum during this period (perhaps 10-15% with the majority coming from essential fatty acids like flax oil). This should help to minimize fat gain (overfeeding studies by Acheson have found that lipid oxidation drops to around 59 grams of fat per day so keeping fat intake below that level should avoid most of the fat regain).

Additionally, one study on rats found that vanadate (similar to vanadyl sulfate but far more toxic) pushed fat towards oxidation. But it stimulated fat synthesis at the same time. So, keeping insulin sensitivity high while avoiding too much insulin mediated fat storage with chromium, vanadyl or even phen- or metformin might be helpful.

#2
DE NOVO LIPOGENESIS (DNL) FROM CARBOHYDRATES

Normally, conversion of carbs to fat is relatively limited and DNL is thought to contribute a minor amount towards fat gain. But under certain conditions, mainly severe overfeeding, carbohydrates can be converted to fat. The great majority of the conversion occurs in the liver. One study (Acheson et. al., 1988) found a gain of 2.2 kg over five days of severe overfeeding (700-900 grams of carbs per day) following five days of low carb eating.

In those situations where you're super compensating muscle glycogen following training and consuming an excess of dietary carbs, some DNL will occur. During the conversion of carbohydrate to fat in the liver (a pathway mediated by an enzyme called citrate lysase), an intermediary substance called Malonyl-CoA is formed. One of Malonyl-CoA's main effects is to shut down fat oxidation by inhibiting the carnitine palmityl transferase (CPT)

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Baby Building

By Laura Moore

(Editor's Note: I thought you'd like to know how much weight gain is necessary during a pregnancy.)

Risks of gaining too much weight when pregnant:

a) gestational diabetes
b) preeclampsia (hypertension, with edema)
c) back strain and pain
d) harder time getting back into shape
e) infant will probably be big, thus a tougher delivery which increases the chances of a cesarean
f) possibility of stretch marks

Where do the pregnancy pounds go?

Maternal stores of fat, protein, other nutrients	7
Increased body fluid	4
Increased blood	3-4
Breast growth (yippee!)	1-2
Enlarged uterus (yucky!)	2
Amniotic Fluid	2
Placenta	1.5
Baby	6-8 (Tejay = 8 lb 11 oz)
Total	26.5 - 30.5 pounds

dan's internet snips

http://www.hotmail.com
http://www.geocities.com

Both of these web sites offer free e-mail. This seems redundant because if you can access these websites, in most cases you will have your own e-mail. However, some readers may not own a computer, but have access to the internet through a friend's or employer's computer. Additionally, most public libraries have computers with internet access. The cleverness of both hotmail and geocities is that you can create a private e-mail name, with a protected password, so you can access your e-mail from any computer online server, and your e-mail is private. Additionally, you can send and receive e-mail that might be sensitive, not to be looked at by a government agency, employer, or hacker (in the case of ordering steroids over the internet).

For example, once you enter the hotmail website, you can easily set up an e-mail account, with a name, for example, like NUMBNUTS@hotmail.com. You assign a password for access to get any e-mail people might send you. Then it would work like this: at your office, the business you work for might have a business e-mail name. So it wouldn't be cool for private e-mail to show up for you, as many other employees can access it. Through the business screen name, you would simply access the www.hotmail.com website, type your password, and send and receive e-mail. After office hours, you can drop by your local library and access the hotmail site for your e-mail. Or you could do this on a friend's computer. This will work with any internet provider, even out of the country.

http://www.dejanews.com

This seems to be an obvious and essential website, but I'm surprised how many internet users don't use it. The chief feature is a search and retrieval service that scans through all the Usenet newsgroups, looking for either title, subject, or screen names. Additionally, you can limit the newsgroups, if you wish. I say all of this because many newbies (computer users who are new to the internet) will broach a very broad fitness-related question on a newsgroup or private e-mail group, and the question has been answered and discussed previously. Buy plugging keywords of the question, dejanews will retrieve many discussions on the topic. For example, if you plug the word "steroid" and specify the screen name as "DDuchaine," you would find every response I've posted on the subject on steroids on the Usenet groups.

Additionally, dejanews has a newsgroup reader that allows you to read the current postings of, for example, misc.fitness.weights (the main bodybuilding discussion group). Although most internet providers have newsgroup readers, some of them have slow-to-list messages, or become temporarily out of service (very true with America Online). DD

FOLLOW UP

About ACE Inhibitors

by Robert Ames

(Editor's Note: Dharkham's previous article on Alpha-2 downregulation generated a high amount of interest. If you look at the PDR, you'll notice that the description of the various ACE inhibitors is unusually long, with many warnings. I've asked Robert Ames [with Dharkham's assistance] to expand on the topic of prescription ACE inhibitors. In the future, we'll introduce and discuss naturally-occurring ones, but this research is still ongoing.)

ACE means Angiotensin Converting Enzyme. *(Editor's Note: Angiotensin is a plasma protein acted on by the kidney enzyme renin.)* It transforms the polypeptide angiotensin I (an inactive form of angiotensin) into angiotensin II (the active form). Angiotensin II is bad news for bodybuilders. The harmful effects of this substance on our physical appearance greatly outweigh its beneficial effects. The point is, you will be better off with the least amount possible. Unfortunately, training and many popular bodybuilding and dieting drugs increase angiotensin II formation and exacerbate its harmful effects. So, if you combine training and drugs, you will benefit even more from ACE inhibition.

Here are the benefits derived from ACE inhibition, in reverse order:

10. It reduces arterial hypertension and blood pressure.
9. It has cardioprotective effects.
8. It improves the quality of sleep.
7. It reduces water retention by inhibiting angiotensin II formation, thereby mechanically lowering aldosterone secretion (aldosterone is a hormone which forces your body to retain water).
6. It reduces the release of training-induced catabolic hormones. Elevated angiotensin II will be one of the factors promoting the cortisol and vasopressin secretion seen after training.
5. It increases muscle blood flow and as a result increases oxygen and substrate supplies while working out.
4. It enhances insulin sensitivity and so allows easier fat loss. This is especially true for clenbuterol/ephedrine/ yohimbine users.
3. It spares proteins by:
   a. reducing amino acid transformation into glucose.
   b. reducing training-induced proteinuria (proteinuria is the scientific word to say that once you are done training, lots of amino acids will be transported into the bladder to be urinated, depriving your muscles of amino acids when they need it most).
2. It reduces the potential fat gains while bulking up by reducing the secretion of hormones producing fat hypertrophy.
1. It increases fat mobilization by reducing the release of hormones which prevent fat loss.

Actually, inhibiting angiotensin II formation has many more potential good effects but we're only concerned with the ones most beneficial to bodybuilders/ dieters. However, inhibitors of angiotensin II are not free of side effects. Here are the main ones:

ACE INHIBITOR SIDE EFFECTS

ACE inhibitors are relatively new drugs. Furthermore, as their actions are rather specific, they do not show many side effects. Of course, some people are unlucky and seem to experience the negative side-effects while others have none at all.

Here is a top five list in reverse order:

5. Skin rash and loss of taste. This has been reported by the scientific literature in some rare cases. We have never seen anything like this.
   (Editor's Note: Some people report itching and a loss of appetite.)
4. It can induce cough. It is reported by the doctors but we have never seen it in bodybuilders. Perhaps drugs like clenbuterol can prevent it.
   (Editor's Note: No it won't, but Stadol, will.)
3. Hyperkalemia. (Increased levels of potassium in the blood.) This can be dangerous in normal people. But this is good news for steroid and dieting drug users as both types of drugs tend to depress potassium blood level. So, those two side effects will tend to cancel each other.

   But bodybuilders preparing for a contest should be careful if they use potassium supplements and/or take potassium-sparing diuretics (i.e., Aldactone).

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DIRTY DIETING #2

FOLLOW UP *from page 8*

2. Somnolence. This is a common side effect, especially when one starts using ACE inhibitors. It can be avoided if one begins taking it at night with the lowest dose possible.

   Again, this could be welcomed by some drug users (like steroids, clenbuterol, yohimbine or ephedrine) as those drugs tend to prevent you from falling asleep.

1. Drop in blood pressure and hypotension. This is pretty common and could be troublesome if the ACE inhibitor is taken by itself. In chemically enhanced bodybuilders though, these side effects will be welcomed in order to counteract the increased blood pressure commonly seen with bodybuilding/dieting drugs.

   Overall, if you do not start with very high doses and do not use it alone, ACE inhibitors are relatively safe.

   Miscellaneous effects: women taking ACE inhibitors should discontinue if they become pregnant. Swelling of the face and tongue when beginning ACE inhibitors could be a sign of angioedema, a serious condition. If this happens, seek medical advice.

Shopping for the right ACE inhibitor!

Frankly this is easier said than done. There are many different ACE inhibitors. This is to be expected as the hypertensive market is very lucrative. All the major drug companies want to be present on this market with an original ACE inhibitor. The trouble is, there is almost no difference between them except the shape of the molecule. In fact, a very recent pharmacological review about ACE inhibitors concluded: "There is no clinically relevant difference among the various ACE inhibitors."

That is not going to help us. Until now, we've given you scientific facts. Now, I'm going to give you my personal preferences based on my experiences. I personally prefer Captopril (sold as Capoten) for several reasons. It is the oldest (launched in 1984) and best known as far as effects (good or bad) are concerned. In fact, most of the studies showing relevant positive effects for bodybuilders were done with Captopril.

But there is more. Captopril effects are short lasting. So, it is good to start with it. If anything turns ugly at least you will know it won't last very long. Furthermore, it is easier to fine tune the proper individual dosages with a short duration drug.

But Captopril is not trouble free either. First, it has to be taken at least twice a day. On top of that, it should be taken one hour before a meal. So, it is not a user friendly drug. To sum up, it is good to start with it and then shift to an ACE inhibitor which is more convenient. Common dosage for Captopril is 25-150 mg daily in divided dosages.

Enalapril (sold as Vasotec) was discovered shortly after Captopril (launched in 1985). It is easier to use as it can be taken once daily with a meal. Common dosage: 10-40 mg.

Lisopril (sold as Prinivil or Zestril) was introduced on the market in 1987 but does not show much advantage compared to Enalapril. Common dosage is 10-40 mg once a day with meal.

Same with Ramipril introduced in 1989 as Altace. Common dosage is 2.5-20 g at once or in divided dosages.

We could go on and on:

- Fosinopril (sold as Monopril)
- Benazepril (sold as Lotensin)
- Quinapril (sold as Accupril)

They all have the same posology: 10-80 mg at once or in divided doses.

- Spirapril (sold as Renmax). Doses: 3-6 mg once daily.
- Moexipril (Univasc). Should be taken one hour before meal. Dosage: 15-30 mg once or in divided dosages.

Overall, it is nice to start with Captopril. You can stick with it if you want a very precise dosage and don't mind the multiple, impractical intake. But as most will not find it convenient, you can switch to a more friendly ACE inhibitor which can be used once a day with food such as Lisinopril or Enalapril.

The new kid on the block.

Losartan (sold as Cozaar in the US) is not an ACE inhibitor. It simply blocks the angiotensin II receptors. It is specific for the AT1 subtype which are the angiotensin receptors located on fat cells. The dosage is 50 mg a day either all at once or 25mg both in the morning and in the evening. It is said to have fewer side effects than the classical ACEI but it is a relatively new drug, so let's stay prudent on that subject.

What is nice with Lasortan is it seems to go beyond what a simple ACEI can do. For example, ACEI does not seem to be able to completely abolish angiotensin II formation in fat cells. This is probably why it takes so long before cosmetic results become visible. By blocking the angiotensin II receptors, we are able to overcome this limitation. In theory, stacking Capoten with Cozaar should accelerate and perhaps enhance the Alpha-2 receptor down-regulation. Of course, that would make an expensive stack and the side effects are likely to add up.

A big problem with Losartan is the body will fight it in making more angiotensin II and more angiotensin receptors in fat cells. Stacking Cozaar with Capoten will solve the first problem but not the second. There is a way to down-regulate angio-tensin receptors in fat cells. Unfortunately, we do not know how to do that right now. But I am working on it. One last word on Losartan: it has been shown to prevent fat cell growth. However, only time will tell if it is more effective than regular ACEI.

Of course, we want fewer Alpha-2 receptors on fat cells, but our ultimate goal is to have both smaller and fewer fat cells. So, I am under the impression it is not the last time we are going to use the (dirty) words of angiotensin receptor blockers and ACE inhibitors.

SOME USEFUL STACKS

Converting enzyme inhibitors stack very well with muscle building and fat loss drugs (don't forget that both anabolic steroids and fat loss drugs, especially if done with high intensity workouts, will enhance angiotensin formation — an ACE inhibitor will take care of this).

But there is more:

ACE inhibitors + anabolic steroids.

Some of the side effects associated with anabolic steroids include increased blood pressure and cardiac damages. ACE inhibitors will reduce them both. Furthermore, by lowering aldosterone secretion, ACE inhibitors will fight steroid-induced water retention. ACE inhibitors will also enhance steroid muscle building effects. For example, steroids are not good at reducing training-induced proteinuria while ACE inhibitors are. So, these two drugs combine synergetically to enhance anabolism.

ACE inhibitors and dieting drugs.

By dieting drugs, we refer to either ephedrine, yohimbine or clenbuterol (or all of them at once). They too increase blood pressure and can cause cardiac damages. Futhermore, they all enhance training-induced proteinuria. ACE inhibitors will take care of all this. On top of that, ACE inhibitors and dieting drugs will promote fat loss by different mechanisms. By taking both we create a synergy while reducing the potential side effects associated with each when used on their own. DD

BUILD YOUR BODY

TIP #3

by Jack Giovanoli

HOW TO MAKE "CHEAP" CREATINE CANDIES

Those little Phosphagems™ from EAS® sure are great. Makes it easy to get plenty of creatine – especially in the loading phase. But they're pricey. So I've put together a recipe for creating your own creatine candies at a price that's less intimidating. However, if you're short on time, spending the few extra bucks for the Phosphagems® is probably the way to go (especially since they taste so damn good).

INGREDIENTS

6 Tbs. corn sugar (dextrose)
2/3 cup orange juice, strained of pulp
6 Tbs. light corn syrup
4 envelopes Knox™ unflavored powdered gelatin, softened in 1/4 cup water
1/4 tsp. orange food coloring
1/2 tsp. orange extract
36 grams creatine (about 1.25 oz)
confectioner's sugar

DIRECTIONS

1) Add the creatine to the corn sugar and mix to disperse.

2) Combine the creatine/dextrose mixture with the orange juice and corn syrup in a pan and heat them slowly, stirring with a wire whisk until the sugar mixture dissolves.

3) Continue to stir until the mixture just begins to boil.

4) Remove the pan from the burner and add the gelatin (when you combine the 1/4 cup water with the 4 envelopes of powdered gelatin it will turn into a big glob – don't worry, it will dissolve in the juice mixture). Continue stirring the mixture until the gelatin dissolves.

5) Add the food coloring and the orange extract and stir until the mixture is evenly colored.

6) Pour the mixture into a dampened, 6" square baking pan.

7) Let the mixture set in a cool place for at least six hours, or overnight.

8) When firm, cut into 36 squares, remove each square from the pan and roll in confectioner's sugar to coat.

Makes 36 candy squares, each containing:

28.75	calories
6.5g	carbohydrate
.67g	protein
0g	fat
1 g	creatine

These turn out a little different than Phosphagems® (one serving size):

Phosphagems™ (6 gems)	Cheap Chews (6 squares)
5.2g creatine	6g creatine
132 calories	173 calories
35g carbs	39g carbs
0g protein	4g protein
0g fat	0g fat

Cheap chews are all natural while Phosphagems™ do contain artificial flavor. The little bit of protein in the cheap chews probably doesn't help but it shouldn't hurt too much either. The consistency of these versus the Phosphagems™ product is a little different. The cheap chews are a little more tender, although they will firm up with age. The Phosphagems™ also seem to get more dense and sticky as time goes on. I personally like them less sticky. In any case, this is cheaper than buying the commercial creatine candies. And that's what some of you have asked about. If I really worked at it, I might be able to duplicate the Phosphagems™. I like these better though.

One more thing: Don't put them in the refrigerator because they'll really toughen up.

[Editor's Note: Corn sugar [dextrose, derived from cornstarch], is sold in beer-brewing stores. Someone might want to try a derivative using malt extract that contains glucose, maltose, and glucose polymers.) DD

NEW BREED MRPs

by Dan Duchaine

I know three MRPs (meal replacement powders) that contain moderate amounts of dietary fat. The one that we include in this newsletter is *ISO*^{3}™, at a special price of $34.95 for 37.25 ounces. This is the least sweet of the three. Thus the flavor is easily manipulated with additives. It also is the thickest, and will gel into thick milkshake consistency in cold water, or a pudding with skim milk. It mixes easily with a spoon.

The second one is the ***40-30-30 Balance***™ drink mix, based on the ingredients in the Balance bars. Discounted price of a 22.7 ounce container is $21.55. This is a sweet-tasting product (I bought the vanilla flavor for all three), can be mixed easily with a spoon, and (and this surprised me) was almost as thick as the *ISO*^{3}, even though the carbohydrate source is a simple sugar.

The third is from another (candy) bar company, the ***PR Powder***™, and has a discounted price of $19.95 for 18.2 ounces. I expected this powder to be better than *Balance*. It is the sweetest of the three, and dissolved with a spoon quickly, but with no thickening. The *PR Powder* is the only one that could be put into a water bottle, if that's any consideration.

The per ounce cost for each is:

- *ISO*^{3}: 96¢
- *Balance*: 95¢
- *PR Powder*: $1.10

In future columns, I'll discuss the technology we used in formulating the *ISO*^{3} product. Of the three products, the *ISO*^{3} is the most sophisticated, and has the costliest ingredients. (Note: It also has over 51% more protein per serving.).

For example, the main ingredients in the *Balance* product are fructose, a commercial vegetable oil powder, and a mixture of casein and soy proteins. Although fructose does have an acceptable glycemic index rating, fructose can raise blood triglycerides, lower active thyroid production in the liver, and increase hunger.

The *PR Powder* is slightly better compared to *Balance*. But *PR Powder* also used fructose as a chief carbohydrate source. (Note: This is particularly detrimental to a dieting bodybuilder.)

The *ISO*^{3} powder uses higher quality whey peptides and whey concentrates as the protein source. The carbohydrates come from amylopectin-based glucose polymer, as fructose was to be avoided. DD

NEXT ISSUE: Carbohydrate considerations in formulating a meal replacement powder.

DIRTY DIETING #2

ESTROGEN INFLUENCES ON SKIN THICKNESS

by Michael Zumpano

Have you ever seen a woman with cellulite cross her legs? Against the side of the thigh you can see bias lines form parallel to the lines of stress. You see this same thing across the rhomboids and lower traps of athletes without muscle separation. Dermatologists know this is caused by a depolarization of epithelial cells. The epithelial cells are normally aligned. An increase in the number of epithelial cells and a thickening of the epidermis is also present.

Some bodybuilders' skin is thick. Is it fat? No! I have compared the body composition of two athletes. One had great separation at 4% bodyfat. The other, more muscular athlete, had poor separation at 2.5% fat. Skin can range from 1 to 4mm in thickness.

What makes skin thick and how can you fix it?

There are a number of growth factors that control the thickness and integrity of skin. But the focus of this particular article is estrogen. Estrogen is not anabolic in muscle, but it's very anabolic in skin. The Epidermis is about 0.1mm thick, although estrogen can double this. Estrogen induces greater activity of fibroblasts in skin. These are the cells that make elastin and collagen. Extra fibrous proteins which can form beta pleated sheets are found in and around areas of cellulite. Estrogen increases the thickness of each histological layer in the skin. Estrogen is the classic depolarizer of epidermal cells. Although it works these feats by manipulating the activities of other hormones, estrogen is the light-switch for thick skin.

There are genetic factors involved with skin thickness as well.

Some people have thicker skin than others as a result of high estrogen receptor activity in skin. For those who have high activity, the most important thing you can do is get rid of estrogen. The dermis is composed of elastin and collagen proteins which, a number of studies show, respond to estrogen. This is why medical skin creams used to contain estrogen. It improves the structure of aging skin by increasing protein synthesis. This is why trans-sexuals' skin takes on a feminine look when estrogen treatments are begun.

Some mention has been made from a review of studies that were done in the fifties and early sixties which showed the histological impact of estrogen on protein synthesis in the skin. The review doesn't give the numbers, except to say that the protein content of the skin more than doubled in a short period of time.

With the popularity of straight testosterones at an all-time high, eliminating estrogen is difficult. Once skin has thickened it can take more than six months to involute. There are a number of compounds that inhibit the aromatization of testosterone and synthetic androgens to estrogen. Most of these compounds induce changes in the cytochrome P-450 of many enzyme systems involved in steroid binding. This means they can generally screw-up your anabolic state. But there are some that do not. Keep in mind that when you inhibit the aromatization you may up-regulate the enzyme system that degrades testosterone.

The turnover rate of skin tissue is 52-75 days, although the review noted above said that some improvement was still noticeable beyond this period. This leads me to believe that estrogen suppression must exist for about three months to have the desired effect. Testosterone is without stimulatory effect on elastin and collagen as per this review.

In the category of prescription drugs, Tes-lac and Arimidex are on the short list. Tes-lac became schedule-III along with steroids. Arimidex is not. Tes-lac must be taken about 5 times a day. Arimidex can be taken once a day. Tes-lac interferes with the binding of other hormones. Arimidex is reported to be inert in every sense except for its aromatase inhibiting effect (if you believe the drug companies). However, people who take Arimidex report the same thing we hear about every estrogen inhibitor: they feel generally less anabolic. We always thought this was due to testosterone displacement, but Arimidex isn't suppose to do that. So it may be that part of what we perceive as anabolic is just the extra water retention and resulting hydraulics we receive from estrogen. Arimidex is expensive. Plan on spending as much as $785 per 100. Ouch! One dose a day is said to reduce 80% of the estrogen conversion in your body. It's as good as anything.

(Editor's Note: Ciba is claiming that their letrozole is a better anti-aromatase than Arimidex. It is available in Europe as Femara.)

If you don't want to get into prescription drugs, there are a host of other possibilities for eliminating estrogen conversion.

These are a few available aromatase inhibitors in order of decreasing potency: dl-aminoglutethimide, 7,8-benzoflavone (not 5,6-benzoflavone), chrysin, apigenin, quercetin, 7,4'-dihydroxyflavone, alpha-napthoflavone, flavonone, and equol. Each of these compounds has numerous other effects (some toxic ones) which you should research thoroughly before experimenting with. The trouble in evaluating the potency of these compounds is that most of the data is from human placental and fish ovarian microsomes. These are different from the aromatase system installed in male bodybuilders, so you have to experiment. Nevertheless, many of these compounds are quite potent. Availability will probably dictate what you choose.

Quercetin, a flavone, is always available, as it's derived from Pagoda. You can buy it from botanical companies (like Sweitzall) and has about a 5 to 1 competitive effect on estrogen. It also is not reported to inhibit nuclear steroid binding. Quercetin is a monooxygenase inhibitor, like many flavones. This is why these compounds are effective on oxidoreductase systems like the aromatization of testosterones. Quercetin has the added benefit of being a potent cyclooxygenase inhibitor (virtually identical to monooxygenase). This means it will inhibit catabolic prostaglandin-E synthesis — a side benefit. On the other hand, dl-aminoglutethimide has a better ratio of about 19 to 1, but this was with fish guts, not bodybuilders.

Quercetin has almost one-third the activity of Cytadren. What makes it better is that quercetin has less affinity (there has been no documented activity I could find) on any class of steroid binding globules, unlike Cytadren. Quercetin is quite safe to take all the time. However, taking quercetin in individual doses greater than about 500mg active compound has been associated with chromatin changes in vitro. I'm not clear as to what the implications are for human subjects. It's just a statement that was thrown out in a review discussion I once read. These review discussions can be truly misleading sometimes.

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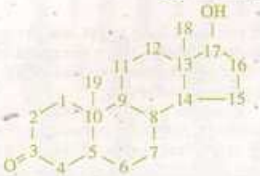
from the desk of
Daniel Duchaine, PhD

STEROID BASICS PART 3

by Bill Roberts



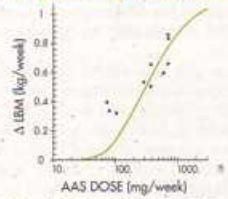
In Part I, we noted that in the normal male most androgen receptors (ARs) have androgen bound to them at any given time. This is because of the high binding affinity of testosterone and particularly DHT to ARs.

Why, then, is increased anabolism seen when anabolic/androgenic steroids (AAS) are taken as drugs?

One reason is that two ARs must join together to form an activated dimer, and both must bind a molecule of AAS. This means that if, say, 71% of receptors are binding steroid, only 50% of the dimers will be activated. Thus, there is room for improvement.

Nonetheless, anabolism increases even as the dose becomes more than sufficient to ensure virtually complete binding. Why?

Another piece of the puzzle is if an effect is dependent upon the activity of a receptor, then the response should follow a sigmoidal function. A graph such as the one below will be of an "S" shape: nearly flat both at low and high doses, and approximately linear at moderate doses.



We don't have good data for this type of graph. The data points are compiled from many different studies, the subjects were not eating adequately for bulking cycles, and there is no high-dose data. Nonetheless, it's clear that a sigmoidal function doesn't describe the response to increasing doses of androgen. If the sigmoidal fits the data points in the linear region, it underpredicts response in the low-dose region.

This is typical of a drug response in which there are at least two mechanisms of action. One or more mechanisms are responsive at low doses and are quickly saturated, and one or more are responsive to high doses.

There is ample evidence that this is indeed the case with AAS. Certain mechanisms are clearly not mediated by the ARs. For example, neuronal effects have been observed in vitro which occur far too rapidly to be mediated by the ARs transcription-factor mechanism.

In muscle tissue, androgen has been observed to activate the immediate-early gene zif268 in a process not involving the ARs. This activity is almost certainly related to muscle growth, and it requires high doses.

Testosterone is observed to increase the efficiency of mRNA translation of cellular proteins, and this may be mediated by a mechanism independent of the ARs.

In what other ways might high doses increase activity?

As discussed before, an increase in the number of androgen receptors is more important than an increase in binding. Androgen is known to up-regulate the production of ARs. We've all heard otherwise, but such claims are based on flawed experiments using aromatizing androgens on tissues containing high levels of aromatase.

If you doubt this, and believe that AAS down regulate the receptor, then I believe you will have a difficult time indeed explaining why bodybuilders and powerlifters who use high dose AAS continuously have a lot of muscle. They should be very small according to that theory!

Besides androgens themselves, there are other factors that up-regulate ARs production. Weight training is one example, although it's not known how much is required to achieve optimal results. Nor what style of training is most effective. But it appears that more sets than Mentzer would advocate are required.

Obviously, one is going to be training with weights anyway. So what other factors will up-regulate or improve the activity of ARs?

cAMP promotes the activity of ARs, and so drugs which increase cAMP will be of benefit. This includes ephedrine. Perhaps this is the reason for the observed value of ephedrine or clenbuterol in dieting phases. However, the effect is clearly not of great importance in bulking phases when cAMP levels are high anyway.

Growth hormone up-regulates ARs production. Prolactin also exhibits this property, but overly-high levels probably won't be desired. Unless, of course, one wishes to breast-feed.

Not only are the number of ARs important, but also their efficiency of operation. ARA70 is a protein which can improve the activity of the ARs by ten times! Perhaps this protein is up-regulated by high doses of AAS — I wouldn't be surprised. ARA70 is a new discovery, and the regulation of this protein is not understood. Unfortunately, it would not be possible to increase cellular levels of ARA70 by taking it as a drug.

RAF is another helpful protein. It enhances the binding of the ARs to DNA by about 25-fold. GRIP1 and cJun also improve activity. Although it's not clear how to increase muscle levels of these proteins, we can understand that the body may at differing times have high or low responsiveness to AAS depending on the levels of regulatory proteins.

Not everything is good news, though.

ARs mRNA does have suppressor elements that can be bound by proteins. This means that the body could produce proteins that would reduce the production of ARs.

Nuclear factor kappa B is another enemy of the ARs, acting to negatively regulate its gene. CFos, RelA, and calreticulin are also inhibitors of transcription or transactivation.

The activity of the ARs itself can be modulated by phosphorylation, but this is unlikely to result in low activity because highest activity results from complete phosphorylation. Severe dieting, however, might result in less activity.

And now for some practical applications.

First, recognize that some activities of AAS simply are not going to occur at low doses. People seem to believe that the scientific research showing that AAS did nothing for the athletes in the studies was bogus, but I don't. The science was correct. 100 mg/week or so of AAS will not do anything significant.

No study has ever shown much results with anything less than 300 mg/week of

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ORAL STEROID DOSING

It would be very desirable to have high anabolic activity from an AAS with no adverse effects on natural hormones.

The Lancet (1976, v2, p699) reports usage of 100 mg/day Dianabol by 11 athletes for six weeks. Despite the fairly high dose, LH/FSH was not inhibited at all.

What was different about this study? Instead of dividing the dose through the day as most bodybuilders do, these athletes received the Dianabol in single daily doses.

Similarly, Michael Mooney has reported that oxandrolone in divided doses is strongly inhibitory of LH/FSH. While Alexander Filippidis reports that this isn't so when the drug is taken all at once each day.

Other evidence suggests that there are also benefits for the liver with this dosage pattern.

We'll be talking more about the best ways to stack orals in future issues. **DD**

STEROID BASICS *from page 12*

testosterone. I'll grant that in the case of the occasional athlete who suddenly devotes himself to hard training and big eating, 250 mg/week of testosterone can be effective. This is because such a person does not need the full potential effect of anabolic steroids. He could make large gains without any drugs at all.

Second, recognize that increasing the number of ARs is of prime importance. Receptors, once produced, have a lifetime of weeks. The most logical plan is to up-regulate receptor production early in the cycle with potent steroids which are probably most effective for this purpose.

Trenbolone (Parabolan) is likely the king of anabolics for this purpose. Testosterone's effectiveness as a mass builder despite weaker binding properties than many other AAS implies that it's effective also. On the other hand, AAS such as methenolone (Primobolan) and nandrolone (Deca) are perhaps not very effective in up-regulating the ARs.

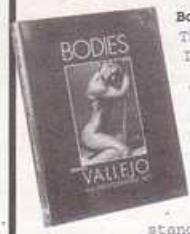
Can the highly anabolic state induced in the first few weeks of high-dose use continue forever? Can one gain 100 lbs of muscle per year? Of course not. Recall that there are negative mechanisms, as discussed, and furthermore we must consider effects on the natural hormonal axis.

Next issue we'll consider what to do after the first few weeks of the cycle. **DD**

Dan's DEVIANT DELIGHTS

Denise Masino's Private Collection
10, 8" x 10" color photographs
$15, or $150 for the collection
(add $2.50 for postage)
from: Denise Masino
Box 409
13300-56 S. Cleveland Ave.
Ft Meyers, FL 33907

Denise Masino is a professional female bodybuilder. She turned pro at the Nationals in 1995 by winning the lightweight division. The reason I bought her pictures is because she has, in my humble opinion, the best tits in bodybuilding, unless you have something against implants (which I do not). If you like those Farah Fawcett big-always-erect type nipples, the ones you can hang your weightlifting belt on, this is the naked girl for you. In her collection, you'll find four pictures of her on a chair with her tits hidden, but with her ass hanging out. The rest of the pictures have her wearing only combat boots (I kid you not), while sitting on an ab bench in a gym setting. My girlfriend (who is a semi-expert having 700cc's after numerous boob jobs) mentioned that in most of her shots, Denise is either holding tits, or her arms are raised overhead. She says that this is usually an indication of droopy boobs. I have no way of knowing. All I can say is that her implants are so large and tight against her skin, they look like they might burst out of her skin (Denise is reasonably lean in all the pictures). Personally, this is a real turn-on for me; others might think it to be painful-looking. Beyond the tits, other than Denise having a slightly short torso, this woman is really stunning, having dark, seemingly Indian-Hispanic exotic looks. I wish that she had done the tits-front shots in a non-gym setting — it would have been more erotic. For those on a budget, I'd recommend the following 8" x 10"s: Pictures D and C are the stand-outs, with the best tit shots. Another photo from her regular "Collection," Photo B, that has beach-sand smeared over her ass, is the prettiest picture that shows her off best.



Bodies, Boris Vallejo, His Photographic Art
Thunder's Mouth Press, $39.95
ISBN 1-56025-126-3

Vallejo is the fantasy illustrator, specializing in big titted-and assed women. The reason I bought this book is because Vallejo is the only photographer I know who will pick flat-chested women for nudes. As much as I appreciate big tits, most of them are from surgical enhancements, and it is a terrible standard to impose on women, which is sexist, demeaning, etc. On an intellectual and artistic level, I appreciate the fact that Vallejo finds these under-endowed (and the adjective is not a put-down) women, and photographs them to their best advantage. **DD**

UPDATES *from page 1*

Mougios, on the other hand, presumably was more reckless. The result: he (along with collaborators and/or relatives) was arrested by the Salonica police on charges of smuggling "narcotics" out of the country and having set up a bogus pharmaceutical company importing and exporting illegally. Very large amounts of "letters" packed with blister packs (the plastic-and-foil strips that tablets are packaged in) were confiscated, as well as "several hundred million drachmas" ($1 = 240 dr) in cashier's checks, postal money orders, cash, bonds and bank accounts.

The Vipharm/Skouvara businesses stocked Restandol, Testoviron, Primobolan Depot, Proviron, Deca-Durabolin, Anabolin, and Pregnyl. From this selection it would appear that they genuinely only carried products approved by the Greek Health Ministry, and were unwilling to supply products which don't legally circulate in Greece.

The Mougios pricelist included: Spiropent, generic clenbuterol, generic nandrolone, Pregnyl, LIV-52, Proviron, Methandros, Anapolon-50, SPA Oxandrolone, Sustanon, Testoviron, Primobolan, testosterone cypionate, testosterone propionate, Trisoralen, stanozol, growth hormone, etc. Their range of products was much bigger and showed a high amount of knowledge of the bodybuilding market. Large orders (e.g., 1000 ampules) were only filled by unapproved products, such as the Karachi products (Karachi Organon is a privately owned Organon license holder in Karachi, Pakistan), indicating that Mougios was aware that large volumes would attract unwanted attention from the Greek government and his legitimate Greek suppliers. The stocking of LIV-52 proves that they were involved in direct purchasing from India. Three months of orders went missing at the time of closure of Mougios.

Generally speaking, the seizure rate from the various mail-order steroid businesses is one mailed out, but only one package in every 100 in tablet packages.

The big mailorder pharmacies that we all know and love were far more involved in the world 'roid biz than many of us may have realized. Greece was being used as a hub for products manufactured outside the EEC, to enter the EEC. Due to its weak importation laws, massive amounts of steroid products were shipped from third world manufacturers to Greece, the deals being arranged by the top level UK 'roid bosses. The Greeks were allowed to sell product retail to the US and European market because the sales had little effect on the rest of the market. It is not unusual for an entire year's worth of third world factory output to be purchased in one go, which is one million or more ampules.

Testosterones from the Middle East cost as little as 25¢ in batches of 1,000,000. The price of smuggling into mainland Europe is approximately $1 per ampule. Next level down purchases from importers start at 5000 ampules. I know the guys that buy the CID Primoteston pay approximately 75¢ per 250mg ampule in batches of 200,000. They pay a legit shipping company to smuggle them, who charge about a pound (UK£) per unit to ship direct to the safe house. A unit could be a one box ampule with full inserts, or a box crammed full of loose ampules. For example, ten Primoteston ampules will fit into a box, so 1,000,000 would cost £200,000 to have shipped. It may sound high but the demand is high, and the stuff will shift near-instantly for at least £2 per ampule, resold at £5+ for retail. The smuggling trail is: India > Greece > Ireland > Mainland England.

According to the "Kathimerini," an Athens daily newspaper, a (US) DEA probe was in Greece just recently, trying to put an end to the whole scheme. "Cooperation by the Greek authorities was ensured, and some technical details were smoothed," said the paper. Make your own assumptions.

This doesn't really matter. They (Skouvara), along with Mougios, don't use return addresses that can actually be read. All products on Skouvara and Mougios are already on the US Customs Alert Bulletin list.

One of my regular customers told me this: "Four weeks ago I ordered some generic clenbuterol from Mougios and I received the shipment divided into three envelopes exactly 13 days later." Clenbuterol is available in Greece as a pediatric syrup. The generic clenbuterol is probably Bulgarian or English "black" imports. I find it impossible that these tablets were imported officially.

Another customer of mine mentioned that they have ordered stuff like Extraboline (2ml), Retin-A, Nizoral, Proscar, etc. ... for hair loss or skin care and the Greek pharmacies have put their real address on the envelope. Extraboline (this is a nandrolone) is made by Genapharm in Greece, so the name is real. But Extrabolines come in 1ml/50mg ampules. I've never seen the 2ml version, and it's not listed in the Greek pharmacists' drug book. But some drugs produced in Greece are for exportation only and have different quantity and packaging.

I've heard the Skouvara is definitely still in business, but prices are about 33%. And I've heard the Mougios is back also.

New operations are springing up all the time, or so it would appear, but what's really happening is that the names and addresses of these operations are simply changing. It's an unstable business and you can easily get caught with several weeks of back orders, so what could be simpler than disappearing with a nice sum of cash ready to buy stock for a new operation? There are plenty of excuses to cover your ass, namely: you got raided; the money never arrived; the stuff got seized; etc. The average customer won't realize that he's dealing with the same guy again, especially if the guy claims he doesn't speak English and communication is only by e-mail or fax.

If you look at the well known operations you will see a pattern, e.g. anything in the Netherlands or the UK is likely the work of Paul Masters (it's nice to see a boy and his dad working so closely together). Ironically there are plenty of father and son teams in the business. Maybe the sums of cash involved make it hard to trust anyone outside the family. In the Far East it's the same story, with all the pharmacies selling Anabol being run by the Chinese mafia.

Pricing is another interesting issue. Most of the mail-order services will charge $9-15 per ampule of the common products, e.g., Sustanon, Testoviron, Primobolan, etc. Genuine Deca-Durabolin from Organon will cost a good deal more, e.g. $22 per 200mg ampule. An average order is normally 10-15 ampules, so the profit is not that great. To make decent money you would need at least ten orders a week. This may not sound like that many, and compared to the amount of users in the US, it isn't. But reaching these potential customers is a real problem. The safest method is to obtain mailing lists of the bodybuilders and send them a flyer. The next best is to run an ad in the classified section of one of the big bodybuilding magazines such as *MuscleMag*. **Please note that all the English companies who have advertised in MMI have been raided in the past three months.** And then we have advertising on the Internet. The problem with this is that everyone who accesses the Internet seems to think they know everything about counterfeits, prices, etc.

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UPDATES *from page 14*

Even worse, they only believe negative comments about a product or service.

Running one of these operations might seem like a dream come true to the average consumer, but let me tell you, it isn't the case. Sourcing your stock is a nightmare; one bad purchase can finish you off. Try getting a refund on $5k's worth of fake deca. Once you find a decent source, you then have to figure out what to buy. Simple, you may think? It is, until someone decides that they haven't gained enough weight from your product and tells the world it must be fake, even when you know it's good. You're then stuck with hundreds of ampules that you end up shifting to friendly customers at close to cost just to recoup your original investment. You also have to avoid products that your competition can buy for less than you because they live in the country of the manufacturer.

Negotiating the right price is a battle in itself. Just because you know the smuggler is paying approximately $1 per ampule doesn't mean he's going to let you have it for anything near that low. You might be able to pick up 5000 ampules for $4 a piece, but that's as good as it's going to get. And prices are only going to go up. Now you are thinking to yourself that you can buy 10,000 ampules and sell them retail over six months. But now you have a huge amount of stock to store. And if you get caught, not only will you be in a heap of trouble, but you'll lose all your money. If you don't have the money to buy thousands of ampules, the only way you'll able to compete is to import the products yourself, which means you have to start smuggling and risk unwanted attention, which may lead to your mail-order business being discovered.

Then we have the CN22, the green customs declaration sticker. These are the most loathsome pieces of paper on the planet. What you write on these affects the chances of the customer receiving his products. So you try and come up with the most creative and least suspicious thing possible. You name it; I've used it. The problem is that the other guys have probably used it as well, thus lowering the odds of mine being successful. The CN22 has to be filled in by hand and signed. You have to make sure to vary the handwriting style and change the name used each time. **No one really knows how the customs agents choose a package to investigate.** From trial and error we have assumed they must use some kind of x-ray based scanner. So a product that might appear similar to ampules or tablets is often used on the declaration.

The sensible operator will never leave his prints on his products or packaging; this means wearing gloves. Doesn't sound too bad, does it? Well, just try wearing gloves and performing a simple task like removing an ampule from its box. It goes without saying that you shouldn't store any products at home. Many a door has been kicked down to reveal a pile of cash and a bunch of packages ready to be posted.

What advice would I give to the consumer? Don't assume everyone who advertises as a pharmacy really is one. All the suppliers have realized that the customers like to think they will avoid counterfeits by buying from a real pharmacy, rather than a mail-order operation. Don't trust anyone with a huge product range. Don't pay by using checks or wire transfers from your accounts. You don't want to leave a paper trail. Never trust someone who claims they have never had any shipments seized. DD

ESTROGEN *from page 11*

Remember Flavone-X? That's chrysin. Well, now it's available. For comparison it has about a 10 to 1 activity. Pretty good. The price of chrysin has come way down and its availability has suddenly become infinite. So you will be seeing it on sale in the next couple months. It may be that quercetin is the best choice based on cost to benefit ratio. High doses of quercetin — up to 500mg — are tolerable (but not recommended) which allows for a very potent inhibition. Some other flavones are not tolerated at doses less than 100mg. Even though they may be stronger on a molar basis, quercetin can achieve a more potent effect in use.

I recently received a shipment of chrysin. I'll be using this with a group of local bodybuilders over the next few months along with a few other compounds. Anyone who wishes to do their own experimentation can contact me with their results at mzumpano@msn.com. Please keep your communications brief and I will respond quickly. DD

ISO-OPUS ERRATA... *from page 7*

system which carries fat into the mitochondria to be burned.

So, if we can inhibit Malonyl-CoA in some fashion (either by lowering the amounts made or by keeping it from affecting the CPT system), we should be able to at least minimize DNL as well as keep CPT activity high to sustain fat burning in the liver.

There's a readily available supplement that, at least in rats, inhibits activity of citrate lysase which should lower the conversion of excess carbs to fat. It's called Hydroxycitric acid (HCA— trade name Citrimax™) and it might be useful during deliberate carbohydrate overfeeding. Human dosages are unknown but may range from 750 mg three times daily up to several grams per day. HCA should be taken 30 minutes prior to eating because it has to get to the liver before your food.

Oleate (or oleic acid found in olive, peanut and safflower oils) has also been shown to inhibit Malonyl-CoA formation — and stimulate fatty acid oxidation and ketone body formation. It promotes fat oxidation. High amounts of oleic acid are present in Dan's ISO[3] (over 7 gms a serving).

Additionally, one study of a new anti-diabetic drug called pioglitazone (which improves insulin sensitivity) decreased the amounts of liver Malonyl-CoA in rats. Whether one of the biguanides (metformin/phenformin) would do the same thing is unknown but might be worth trying. Plus, keeping insulin sensitivity high with metformin (or even a combination of magnesium, vanadyl sulfate, and chromium picolinate) might help to prevent fat storage.

Finally, the anti-hypertriglyceridaemic drug Gemfibrozil (trade name: Lopid) has been shown to lower blood lipid levels. It's also been found to act as an inhibitor of Malonyl-CoA and might prevent some of the overfeeding fat gain.

In conclusion, a hypothetical list of substances (in order of importance) to prevent some of the fat gain during overfeeding periods would possibly include:

1. Hydroxycitric acid: 750 mg - 4.5 grams three times per day.
2. Low fat (less than 60 grams per day of a combination of flax and olive oil), moderate protein (1g/lb bodyweight), high carb diet (lots).
3. The Ephedrine/Caffeine/Aspirin stack or another thermogenic agent such as clenbuterol.
4. Vanadyl sulfate: up to 120 mg/day OR metformin (up to 2000 mg per day) or phenformin (up to 150 mg per day).
5. Magnesium: 1000 mg/day.
6. Chromium picolinate: 800 mcg/day.
7. Gemfibrozil: 600 mg twice per day. DD

from the desk of
Dan Duchaine, PhD

Q&A

For Q&A questions send to: Dan Duchaine's Dirty Dieting Newsletter, 2533 N. Carson St., #2538, Carson City, NV 89706.

Q More and more I'm seeing weird packaging of European steroids. Either there are no boxes, or the tablets are in bottles, not in strips. What's going on?

A As each year goes by, it becomes harder to smuggle things into the country, as Customs and the DEA learn from their past seizures. And you should know that steroids are not high-profit items. A smuggler can make more money from other drugs (this was obvious), but other items like exotic bird eggs (or the birds themselves), or, of all things, freon, can command more profit than steroids.

To give you an example: injectable steroids, notably Parabolan and Esiclene, have bulky packaging materials (boxes and inserts), so many smugglers throw the packaging out. Additionally, these two particular injectables are highly breakable, as the ampules are both large and have very thin glass. This means that the breakage rate is much higher than a Primobolan or Sustanon ampule. This breakage means lost sales, so the cost is passed onto the next purchaser. This is why an amp of Parabolan can hit $28, while other smaller, sturdier ampules, (having the same wholesale price in Europe), will be $10 less. Esiclene usually retails for almost $80 for six ampules, simply because this injectable gets broken the most. And remember, broken ampules can alert Customs to a steroid shipment, as oily-cardboard boxes have been a signature of steroid shipments for decades.

In years past, we would get tablets in strips, along with all of the original packaging. In the beginning of the nineties, the steroid smugglers pretty much started shipping just the strips. And lately, because of the trend of trying to get more steroids in less space, the smugglers are having the tablets removed from the strips in Europe, smuggling loose tablets, and bottling these tablets when they hit the states. I have seen Primobolan, the 25mg tabs, this way, and I've heard that the SPA oxandrolone will be repackaged this way also. The American steroid dealers love the Thai methandrostenolone, as it is packaged in 1000 tablet plastic jars.

Q I went to the glycemic web site. Pretty depressing. I usually snack on rice cakes, or bread, both of which are high GI. And rice and potatoes didn't look so swell either. And I'm sick of yams. Is there any compact low or moderate GI carb source that I can travel with?

A What you want is a starch that resists water so it doesn't swell up, which allows the digesting enzymes to have more area to work on. I recently came across a cracker, though not officially tested, that might be a good candidate. Years ago, the best-selling cracker in the New England area (where I grew up), was Nabisco's Crown Pilot Chowder Crackers. Chowder cooks liked Crown Pilots because you could throw crumbled-up ones in hot milk, and they didn't get soft and fall apart. This is an excellent indicator that water was not swelling the starch granules, so the starch molecules were reasonably compact. However, Nabisco took the Crown Pilots off the market. After a CBS Sunday Morning segment, featuring irate Mainers lamenting the Crown Pilot's demise, Nabisco received thousands of letters telling them to bring the cracker back. Which they did, but only in New England. However, you can telephone Nabisco at 1-800-622-4726, and order a case (12 boxes) of Crown Pilots, and they will ship them just about anywhere you want. The box of 12 will cost $43. I charged mine on my charge card, but I imagine that they will send them UPS COD. I'm estimating that the GI of the Crown Pilot is around 55-60 GI. Oddly enough, some cookies having soluble fiber and fructose may have a lower GI. But they won't taste as good with peanut butter on them.

Q I'm going to Mexico for some juice. In which issue of MM2K was it that you did an article on Mexican steroids?

A There is something better to buy. PHYSICAL ENHANCEMENT WITH AN EDGE is a Canadian-published book, written by a woman (Shelley Hominuk), that is a complete guide to Mexican steroids and accessory drugs. It's over 500 pages, with a retail price of $39.95. You can call the publisher at 888-797-7729. GURUetc also sells it. DD

Number 3 • July 1997

Dan Duchaine's
DIRTY DIETING NEWSLETTER
MILITANT MUSCLE GROWTH AND FAST FAT LOSS

UPDATES FROM THE UNDERGROUND

(Editor's Note: Our UK steroid dealer subscriber gives us a refresher course on steroid counterfeiting.)

Every cycle you do using under-dosed or counterfeited products is a complete waste of time. It will take you about three weeks to realize the products you're using aren't right and that you'll have to increase the dosage. For most people these three weeks will be about a third of their cycle.

For three weeks you'll be blowing a fortune on extra food and supplements. Three weeks you'll be purposely training extra heavy. Straining your joints because you think the juice is protecting you. To make matters worse, you're also given a false impression of just how effective a product is.

When you do manage to get the real thing you end up using far too large an amount and then suffer the consequences.

It's hard enough planning a perfect cycle when you know exactly what you have. Planning one with fakes is a complete nightmare. No wonder the pros are trying to take two grams (and more) a week. They

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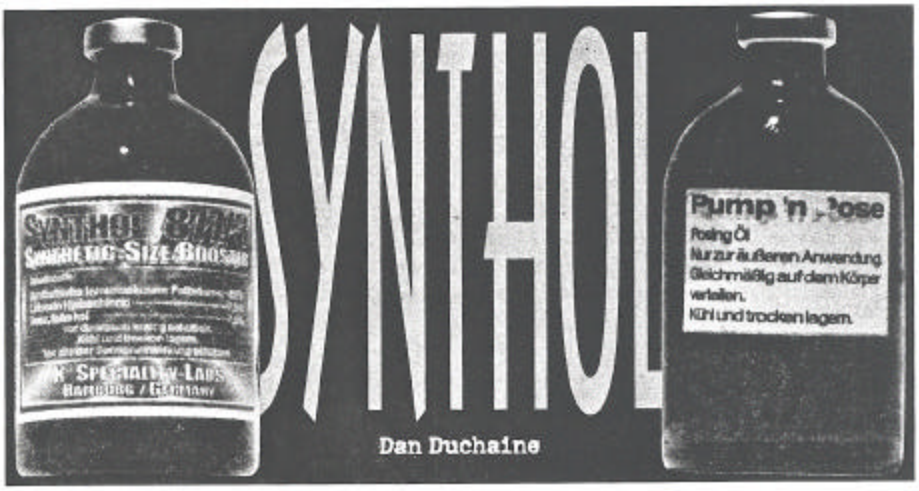
IN THIS ISSUE



The label reads:

SYNTHOL 877/3
Synthetic Size Booster

[translated from the German]
Ingredients:
Synthetic Intramuscular Fatty Acids.....85%
Lidocaine Hydrochloride 7.5%
Benzyl Alcohol 7.5%
Shake before using
Store in a cool and dry place
Protect from direct sunlight

X^3 Specialty Labs
Hamburg / Germany

Additionally, to be able to get this product into the country (the US) legally, Another label is used:

Pump 'n Pose
Posing Öl
Nur zur äuBeren Anwendung
GleichmäBig auf dem Körper verteilen.
Kühl und trocken lagern.

Contained in the brown-glass multi-use vial is approximately 50cc of the oil. The oil is clear, light textured, and has a distinctive benzyl alcohol smell. In this country, the retail price of a bottle is between $300 to $600, depending how gullible the potential buyer is. One seller graciously supplied a sample at his wholesale price of $250.

Supposedly, this is the main reason for some bodybuilders suddenly gaining inches in some of their bodyparts. The favored bodyparts are biceps, triceps, and calves. Unlike the injectable Esiclene, which can add up to a half-inch for a few days, **Synthol** injections can add up to two inches onto a bodypart, and this size will stay for at least six months.

The protocol (word of mouth) is to inject 1cc of the oil in each body part every day. This is somewhat deceiving because the calf has two distinct heads, the implication being that 1cc should be done in each head, so that would be 4cc's each day. In the (arm) biceps, I imagine that a 1cc injection is to be put into each head of the biceps. I have not heard of anyone using 1cc in each triceps head, though.

The injection is supposed to be painful for a few days afterward, with calf pain being worse than the arm pain. There have been no reports about infections.

The size could be from a variety of mechanisms. Some fatty acids (more on this later) are resistant to mobilization in the body. And the body might be trying to encapsulate the bolus with collagen, which many steroid users experience but on a much smaller scale. Even myself, who has-

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Dan Duchaine's DIRTY DIETING NEWSLETTER©

Militant Muscle Growth and Fast Fat Loss

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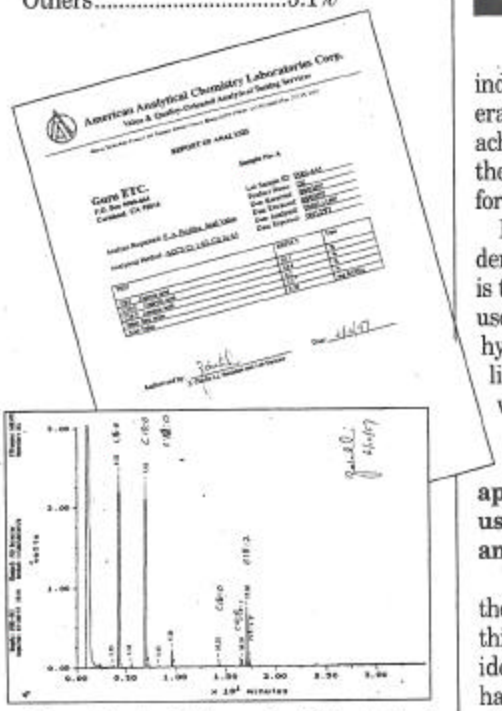
SYNTHOL *from page 1*

n't done a steroid injection for eight years, still has scar tissue and collagen lumps in glutes from numerous past injections.

Additionally, some fatty acids can be caustic (well, technically it would be more acidic than alkaline) and cause inflammation and fluid in the area. Either way, something remarkable is happening. But I can't tell you if this will accrue into a health hazard in the future. In the coming months, we'll try doing some ultrasounds in individuals who have, and still do use **Synthol**, hoping to see any tissue changes.

Below you'll find the laboratory analysis on **Synthol**. As you can see, it is made of:

C8 (caproic acid).....45.3%
C10 (caprylic acid).....43.4%
C18:2 (linoleic acid).....6.2%
Others.....5.1%


American Analytical Chemistry Laboratories Corp.

We estimate that much of the "other" fatty acid is C12. Basically we see medium chain triglyceride, mostly C8 and C10. The producer of MCTs in Germany is Dynamet Nobel (yes, the explosives company). I do remember that both food-grade and medical-grade MCTs have been available from this company.

In the next issue, I'll have a follow-up on my talk with the American company that sells the MCTs here. I have no idea (and no practical way of knowing) if the MCTs in the **Synthol** are sterile. But with that much benzyl alcohol, most organisms should be inert. In discussing this substance with other chemists, one remarked that both coconut oil and MCTs are resistant to mobilization once inside the body. And many

Greg Kovacs

individuals who consume MCT oil in moderately large amounts do complain of stomach pain, and researchers have identified the C8 component as causing the discomfort.

Every bottle of Synthol has a white powdery sediment on the bottom (perhaps this is the reason to recommend shaking before use). I believe that the solid is the lidocaine hydrochloride, which is odd, as a straight lidocaine (without the hydrochloride) would be oil-soluble.

One of the current rumors floating about is that Greg Kovacs, the disappointing IFBB bodybuilder, tried using Synthol in his rear delts only and the results were ... unexpected.

I've reproduced the picture of him with those weird-ass delts as he stood on stage at this year's Night of Champions. I have no idea who took this picture, and we'd be happy to pay the photographer for the use of it.

Some writers have excitedly proclaimed that, "**Synthol** will change bodybuilding forever!" We'll have to see at the upcoming pro bodybuilding shows. No doubt about it: two inches within four weeks is remarkable.

As you might imagine, I don't think that the price of **Synthol** is worth the $5 of ingredients involved. Next month, I'll show you how to make your own version (along with sources for all of the ingredients).

One more thing: laboratory researchers routinely swab rat skin with a mixture of benzyl alcohol and lidocaine to bring up the surface blood vessels for injections. Maybe **Pump 'n Pose** really could be used as a posing oil. I'd sooner use it this way, rather than inject 50cc's of a suspect substance. DD

The Art Of Spot Reduction

Michalovich Dharkam Greutstein (aka Dharkham)

(Editor's Note: Dharkam is doing some interesting work on a new method of fat reduction. Adipocytes are not long-lived in the same way that nerve cells are, so they must be replaced when they expire. By blocking the transition of pre-adipocytes to adipocytes, we can lower the number of fat cells in the body, which is a more effective way to reduce fat than reducing the size of the cells. I held back this article initially because I wasn't sure that there really were Beta2 receptors on fat cells. But Dharkham supplied the references showing that this really is the case.)

Why do we always have that last spot of fat when everything else is gone? If you learned all the food markets in your area were going on strike, what would you do? You'd probably stockpile and hoard food, wouldn't you?

In a similar way, the body holds onto calories in case you're not able to find anything to eat anymore. For women it is even tougher because they need to have enough calories in the form of fat to bring a pregnancy to term.

The surface of the fat cells contains two kinds of biochemical receptors:

- The ones which open the door and empty the warehouse (let's call them the good receptors)
- And the ones which do anything to keep the door closed and prevent the emptying (let's call them the bad receptors).

Needless to say, the last spots of fat contain a lot of the latter and few of the former.

Our strategy is to activate the good receptors while putting the bad ones out of order. We don't want to do this everywhere in the body. Only in very specific last spots of fat. We're going to make a surgical strike, but instead of smart missiles, we're using hypodermic needles.

The goal is to increase the level of a substance called cAMP (cyclic Adenosine Mono Phosphate).

cAMP is good stuff. The more of it we have in the fat cells, the quicker we can get rid of them.

Our weapon to accomplish this is called a beta agonist. The most popular is injectable clenbuterol. But injectable albuterol is as good for fat loss and easier to find. What you do not want to use is an injectable beta agonist which is not specific for the beta receptors such as epinephrine or norepinephrine.

Once injected, the beta agonist will increase cAMP in fat cells and will start to slowly open the lock. Unfortunately, there is an alarm on the lock. Once cAMP level is increased in fat cells an enzyme called phosphodiesterase (PDE) will appear. This PDE is the first enemy we meet, as PDE will reduce the level of cAMP by destroying it.

So now that we've softened up the enemy with beta agonists, we have to defeat the PDE reaction force.

Our weapon for this battle is called a phosphodiesterase inhibitor. There are many on the market — the best being Amrinone and Milrinone. But they're hard to find. In the event a PDE inhibitor cannot be found, less specific ones can easily be found. *(Note: pentoxifylline [Trental] is a phosphodiesterase inhibitor but it is the wrong kind and will not help fat loss — which does not mean it cannot be useful for other purposes, like building muscles).*

So in this case we're left with either theophylline or caffeine. Remember, only when injected locally can those two drugs reach the critical concentration needed to effectively destroy PDE. Taken orally, one will never benefit from these properties of the drugs. So, injecting a beta agonist and a PDE inhibitor will greatly accelerate fat loss where injected.

First, the alpha 2 receptors. There are drugs to destroy the receptors themselves but this requires a few weeks. So, we're going to use the next best but quick solution — block them. We're going to lay down a mine field with the help of an injectable drug called atipamezole. This is the perfect tool for the job but ... it's not easy to find.

Most will have settle for second best, which is yohimbine. Easy to find for oral use, but not for injections. Well, that doesn't matter much. If don't want to use it mixed with DMSO, ingestion is not that bad (but not best).

The last of the enemy's forces are called adenosine receptors. The more the cAMP rises, the more adenosine will be found in the fat cells. This is because when cAMP is degraded it produces adenosine. It's a negative feedback used by the body to make sure you are not losing fat too fast. We need to take care of the adenosine receptors by blocking them. Theophylline or caffeine will do the trick. If Amrinone is used, then theophylline will have to be used along with it.

We have to burn the fat otherwise it will be redeposited. This is called re-esterification. And when the fat is re-esterified it's always in the wrong place.

The classic non-dieting way of ridding fat is weight training and aerobics.

When we say aerobics, we do not mean slow speed, 60% of your heart rate. We mean maximum speed. Enough energy is in the blood to stand it. Aerobics should be done first thing in the morning, on an empty stomach. During the night, the body will have wasted all its carb energy and will already rely on fat calories for energy. Absolutely NO eating beforehand. For two reasons:

1. Eating will bring in calories and so will spare the fat calories we have in the blood
2. Eating while taking a beta agonist and an alpha 2 antagonist will result in a huge boost of insulin.

The insulin is like superior artillery which would defeat all of our armament, especially if we're not using the phosphodiesterase inhibitor (amrinone).

If you're tired that means your body doesn't know how to convert the fat calories into a useable form of energy.

It will learn the hard way if you are waging TCW (total chemical warfare). Train as long as you can. When you cannot stand it any more, have a protein drink (Designer Whey seems to be the best). But remember no carbs. It will make you feel better and you can resume training.

Each day your goal will be to postpone the moment you take the drink. It will mean your body is learning how to use its fat for energy. Afterwards eat protein only. Try to postpone carb intake as much as possible. And remember, you're on a diet, don't stuff your face.

The key is to start very low with only the beta agonist. In a few days add the PDE inhibitor, then the alpha 2. Remember the doses should build up slowly. Too low a dose start won't hurt. For example, 1/2 a ml of clen will probably have no discernible effect. Next day 1ml. See what happens.

Obviously, the injections go into the fat. But you do not want to go too deep. This is where insulin needles (without the insulin) are useful. Furthermore, you don't want to inject in the same place everyday. One day high in the right buttock, and next day high in the left; the next day low in the right and so on.

Divide the area you want to spot reduce into several squares. Use a different square everyday. You will need several injections to have all the drugs in place. Space those injections a little bit in your square. DD

Number 4 • September 1997

Dan Duchaine's

DIRTY DIETING NEWSLETTER

MILITANT MUSCLE GROWTH AND FAST FAT LOSS

UPDATES FROM THE UNDERGROUND

(This issue, our UK steroid dealer elucidates on: the UK veterinary steroid scene.) All prices converted to US dollars.

ANDROJECT
by Intervet: Testosterone phenylpropri-onate; 10mg/ml, 10 ml. Price is $32. At the price of $32 for only 100mg, the black market wholesale on 10 amps of 100mg Virormone (another TP) is only $60. It's the better buy, so there is no way that Androject will ever be available.

CHORULON
by Intervet: HCG; 1500iu per 2ml ampule, 5/box. Price $37 ($180 for 25 ampules). HCG is becoming harder to find. The wholesale price is currently about $24 for a 3 pack, so it doesn't seem too bad for a

continued on page 4

IN THIS ISSUE

The Anabolic/Catabolic Diet

Dharkham

Dan asked me to comment on the ABCDE diet. We are told to keep changing our training routine constantly in order to surprise our muscles and force them to grow. But we keep eating the same way and the same food year in and year out.

Why not surprise our muscles by radically and constantly altering our diet?

This is what diets such as the Rebound diet, the BodyOpus diet and the ABCDE diet are trying to do.

The ABCDE diet is great for people who do not easily gain fat or who easily lose fat. But what if you rapidly gain fat and cannot seem to be able to get rid of it? What will happen is that by feasting for 2 weeks you will accumulate a tremendous amount of fat.

This accumulation will occur in two forms:

1. Each fat cell will hypertrophy. Once some fat cells have reached a critical size, they will secrete growth factors which will build up new fat cells. This hyperplasia will occur in a matter of days if your adipocytes are already big enough, i.e; as you are fat in the first place. OK -- you will diet down the fat hypertrophy. But there is no way of losing the new fat cells within 2 weeks. As you repeat the cycles you will add new fat cells. Intense training (because of cortisol) plus a high carb and high fat diet is a great way to induce adipose tissue hyperplasia in people who are prone to gain fat.

Your body fat percentage is the reflection of the size of the fat cells multiplied by their number. You can shrink fat cell size but only up to a point. You're not be able to lose fat cells. In other words, as you start a low calorie diet, you will only have a single variable to play with (reducing fat cell size). But once you have reduced fat cell size up to a point (which is quickly reached) they won't shrink anymore. So you will not have any variables left.

The diet stops working and you are stuck at a high bodyfat percentage. The only ways to lose fat cells is to use weird

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Dan Duchaine's

Dirty Dieting Newsletter®

Militant Muscle Growth and Fast Fat Loss

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The Anabolic/ Catabolic Diet
(continued)

drugs, or to undergo liposuction. I **think the concept of the ABCDE diet is valid but not appropriate for fat people or drug users.** For those people I propose a variation of this diet that I have been using for some time. I call it the anabolic/catabolic diet (let's call it ACD diet).

It is based on the fact that there is a constant turnover of protein in our muscles. We are constantly adding protein (anabolism) but also losing protein (catabolism). It is the balance between the two which determines the size of our muscles.

So whenever someone complains of only gaining 5 pounds a year, this is not strictly true. He gained maybe 200 pounds of muscle but he also lost 195 pounds. This (contrived) example points out that we are able to gain a tremendous amount of muscle every year. As we cannot hold on to those 100 pounds, it means we are doing something really wrong. As its name implies, the ACD diet is composed of two distinct phases.

An anabolic phase lasting 10 to 20 days and a catabolic phase lasting 5 to 10 days. When I say a catabolic phase, I really mean it: we do whatever it takes in order to lose muscle. Our body will detect something is very wrong and will attempt to stop this rapid muscle loss. It will deactivate the ATP-dependent proteolytic pathway. So far, almost no drug can do this.

The more catabolism you seek, the more your body will fight it. After the catabolic phase, we will be in a position where both anabolism and catabolism are very low. But anabolism is ready to be accelerated to catch up growth while catabolism is reduced to a minimum for a while no matter what.

During the anabolic phase, we will try to boost anabolism as much as possible. During the first week, catabolism will stay very low. So, there will not be an anabolic catch up, there will be an overshoot. How do we do that? During the catabolic phase, the goal is to lose as much fat as possible no matter what the cost is for the muscle mass. I mean a low carb, low fat and low protein diet.

If you are using creatine stop it. Oral anabolics will be stopped 48 hours before the beginning of this phase while 5 days before the injectables are stopped. Thyroid medication will be started at day one. Clenbuterol will be used at day one and maybe on day four or five at massive doses (15 to 20 tablets). Of course, if you are new to clenbuterol do not use that much. At that dose, used acutely you should feel sore all over which will only enhance catabolism.

Overtraining is a must and don't forget about the daily (at least) one hour of aerobics. During your first few catabolic cycle, please do not play with so many variables. Avoid too much aerobics and don't be too hard on the diet. Make it last only 5 days. As you get use to it,add days and variables. The following anabolic phase will start with either a carb load or a fat load lasting only one and a half days. But be careful about not eating too much. Many people get sick whenever they try to load on food after starving themself. On the other hand, one or two days sick in bed is a good way to end up the catabolic phase but only if you are very advanced. The fat load consists of the following: on the last day of the catabolic phase do 2 hours of low intensity aerobics. I like the rowing machine since it involves the upper body as well as the lower body. Repeat on an empty stomach before the fat load.

As its name implies the fat load consists of eating fat with some proteins but no carbs at all. This will direct the fat inside the aerobically trained muscles not in the subcutaneous adipocytes. Whenever you are eating carbs along with fat, this fat will tend

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Steroid Basics Part 5

Bill Roberts

Designing a cycle that will produce good gains is not difficult. We could almost say, "More is better," and leave it at that. Designing a cycle that will produce good gains which shall mostly be retained after the cycle is a more challenging problem. The most basic solution is simply to avoid using the strongest anabolic/ androgenic steroids, and to stick with moderate doses of the mildest steroids, such as Primobolan.

The more advanced bodybuilder, however, has gone beyond the stage where such a cycle would be effective. His maximum genetic potential at, say, 400 mg/week of Primobolan, might be only negligibly bigger than his current size, and well below his maximum potential with a more serious stack.

The problem is that the more "serious" the stack is, the more problems will be had with inhibition of natural hormone production after the cycle is over. We will look at the problem in reverse order. The testes are responsible for producing testosterone. They are stimulated to do so by luteinizing hormone (LH) receptors, which also can be stimulated by human chorionic gonadotropin (HCG.) No other hormones are required. However, if the testes have not been producing testosterone for many weeks or months, and are atrophied, time as well as stimulation by LH or hCG will be required: perhaps weeks or months.

LH is produced by the pituitary. Now this is a considerably more complicated thing. Just as muscle has androgen receptors, which if supplied with androgen will cause cells to produce more protein, and the testes have LH receptors, which if supplied with LH will cause them to produce testosterone.

The pituitary has gonadotropin-releasing-hormone (GnRH) receptors. If supplied with GnRH, also known as LHRH, the pituitary is stimulated to produce LH as well as another hormone, FSH, which is involved with spermatogenesis. However, the amount of LH produced is dependent on many factors. **First, the number of GnRH receptors is subject to regulation. And secondly, responsiveness of the pituitary is affected by androgen, estradiol, progesterone, prolactin, and melatonin, as well as thefrequency and intensity of stimulus of GnRH.**

Too much GnRH will shut down LH production. GnRH is produced by the hypothalamus. The amount that is produced is dependent on the amount of androgen, estradiol, progesterone, melatonin, corticosteroids, opiates, and stimulation by certain neurons.

To review: the hypothalamus controls the pituitary via GnRH, and the pituitary then stimulates the testes via LH. However, both the hypothalamus and the pituitary are affected by a variety of factors, including levels of steroids. And generally, both are inhibited by heavy use of anabolic steroids. Most people probably feel that they know the effect of estradiol (estrogen.) It is bad! And this is why drugs such as clomiphene

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Hi-Tech MRPs

Dan Duchaine

The foundation of all the present meal replacement powders started with Michael Zumpano and myself at OEM Publishing in 1982. Michael stumbled across a powdered form of carbohydrates called glucose polymers trade-named Maltodextrin and recognized it as an exploitable carbohydrate supplement. He formulated a complete powdered food consisting of maltodextrin, caseinates, and liquid medium chain triglycerides. Joe Weider thought it was too odd and too expensive. On my kitchen table, we logged Ultimate Orange and the straight maltodextrin (called Instant Polysaccharide). Eventually, Unipro introduced the maltrodextrin as Carbo-plex. After Unipro backstabbed Zumpano by not fulfilling their contract to him, Champion Nutrition was formed to produce Metabolol, the first meal replacement powder. Since the mid-eighties, Zumpano was friendly with Scott Connelly, who also had a MRP that he was using on some hospital patients. I couldn't really tell you who influenced who, but eventually Scott's MRP became Met-Rx.

Beyond the hype that was used to sell it initially, repeat sales of Met-Rx happened more because of its convenience and superior taste. Although Metabolol was skillfully flavored (especially the chocolate), Met-Rx's strength was its creamy and thick texture. The chief aspects of the flavor and texture are generated from the balance of carbohydrate sources, chiefly from the various starches in the form of modified maltodextrins.

This starch technology was borrowed from General Foods Jello Instant Pudding. It couldn't be exactly used, as those specific modified starches have patent protection. What Met-Rx and almost everybody else does, is to follow the patent without infringing on it. If you want to reproduce most nonfat MPRs, simply put 1.6 ounces of whey protein with a packet of fat free, sugar free Jello Instant Pudding (each package has 210 calories of carbohydrates), into 16 ounces of water, and blend. I particularly like the vanilla pudding with Designer Protein in praline vanilla. This simple (and very cost-effective) combination will stand up to every no fat MPR, and will usually beat them all in terms of thickness and smoothness. You could add a tablespoon or so of your favorite oil, and you'd have a poor-man's ISO^3, minus the vitamins and minerals. I imagine that most of you would use flax oil, but my favorite is macadamia oil, found in most health food stores. My ISO^3 has the benefit of mixing well with a spoon, which isn't the case with the poor-man's versions.

UPDATE FROM THE UNDERGROUND (continued)

product of guaranteed quality, and the idea of a monster 25 amp box makes my dick hard.

DURATESTON by Intervet: Testosterone proprionate 6mg, phenylproprionate 12mg, isocaproate 12mg, decanoate 20mg (in each millimeter). Price $22 for 10ml. You would need to take the whole 5mls to get the effect of a human 1ml Sustanon 250. Price wise it sucks, but the 5ml amp looks awesome.

LAURABOLIN by Intervet: Nandrolone laureate; 25mg/ml. Price is $36.50 for10ml, $66 for 50mg/ml strength. This is probably the only veterinary steroid available worldwide. It's no bargain by our black wholesale prices, but for a novice who is paranoid about using a fake, it might be appealing. Laurabolins are the #1 steroid for making counterfeit injectables.

NANDORAL (tablets) by Intervet: Ethylestrenol; 0.5mg per tablet, 500/tub. Price $96. I was very excited when I heard this veterinary Maxibol was available until I saw the tablet strength. You would need to take 50+ per day just to get a 25mg daily dose. And even then it's a very weak steroid.

NANDROLIN (injection) by Intervet: Nandrolone phenylproprionate; 25 or 50mg/ml. Price is $30.50 for the 10ml, 25mg, and $125 for the 25ml, 50mg bottle. Durabolin has always been a desirable product due to its lack of availability. Here is a product offering the same strength as human versions. The 50cc bottle is a work of art and puts human products to shame. This is the only UK veterinary product I'd buy but I'm not sure if I'd bother to use it myself; I'd probably find an old man or a lady to give it to.

ORANDRONE (tablets) by Intervet: Methyltestosterone; 5mg, 500/tub. Price is $107. With this product name, I was hoping I'd found oxandrolone but it's only methyl test. The price is about the same per hundred as black wholesale, but this would be a very nice container to ship it in.

RETARBOLIN by C-Vet: Nandrolone cyclohexylpropionate; 10mg/ml, 10ml Price is $30. This is a rare form of nandrolone and at this strength a complete waste of time and money.

SESORAL (tablets) by Intervet: Methyltestosterone 4mg/ethinyl oestradiol 0.005mg, 500/tub. Price is $76.50. This oral is cheaper than Orandrone but slightly weaker and contaminated by a tiny amount of estrogen.

VENTIPULIN (oral granules) by Boehringer Ingelheim: Clenbuterol; 16mcg/g, 500g/tub. Price is $41. This is equivalent to 4000 tabs (!!!) of human clenbuterol. This is the bargain of the veterinary world. I have a sneaking suspicion that this is the clenbuterol powder being found to manufacture home-brew tablets. I'll be trying to purchase some shortly as soon as I've made sure there are no undesirable additives.

Steroid Basics Part 5

(continued)

(Clomid) are used. Supposedly, blocking estrogen receptors in the hypothalamus and pituitary will cure all.

It is true that estradiol will cause the hypothalamus to produce less GnRH, and in this regard, clomiphene is helpful, so is reduction of serum estradiol levels by aromatase inhibitors such as aminoglutethimide (Cytadren.) And estradiol does reduce the transcription of mRNA encoding LH, which is a bad thing.

What is not generally realized, however, is that estradiol also greatly upregulates GnRH receptors and dramatically improves response of the pituitary to GnRH. So, therefore, while clomiphene can result in more GnRH being produced, it also results in lower response to GnRH. Estradiol can in fact improve the response of the pituitary to GnRH by eight times or more. My recommendation, then, is that one should not imagine that estradiol activity should be reduced to levels far below normal. Doing so may actually impede recovery. There is no point in continually trying to stimulate the hypothalamus to produce more GnRH by blocking estradiol when the pituitary can't use it efficiently because of a lack of the same steroid.

Progesterone or other progestins act in an interesting way on the hypothalamus. Over the long term, they downregulate production of GnRH and severely inhibit response. Also, progestins decrease the pulse rate of GnRH, which tends to shift the pituitary's production away from LH and towards FSH. And like estradiol, progesterone reduces the

continued on page 5

Steroid Basics Part 5
(continued)

translation of LH mRNA, thus causing less to be produced. But if used in the short term, progesterone improves production of GnRH and LH for a period of several hours after treatment.

One mechanism for this, is steroid's regulation of the enzyme which converts glutamate, an excitatory amino acid, to GABA, which is inhibitory. Androgens downregulate the production of LH in the pituitary, and inhibit the hypothalamus. This is most unfortunate, but it's true.

Perhaps it would be wise for the last anabolic employed in the cycle to be an oral with once a day dosing, thus allowing the hypothalamus and pituitary to be androgen-deinhibited for much of each day.

Up to now, and probably for some time into the future, there has been no science to recovery. About all that was usually said was,

"Use Clomid and maybe Cytadren, and use HCG for the testicles if genuine, always-refrigerated pharmaceutical product is available, and hope for the best. Taper down the anabolics slowly, leaving the weakest ones for last."

We hope to get beyond this. However, the real world results aren't in, and some of the most promising drugs aren't readily available.

First, for reasons of conservatism, safety, and almost certain good effect, is using epitestosterone. This steroid is naturally produced by the body in amounts fairly comparable to testosterone itself. It is a stereoisomer of testosterone; it has the same atoms connected in almost the same way, except that if viewed from the side, the -OH on carbon 17 is pointing downwards instead of upwards.

Epitestosterone has virtually no effect on most tissues of the body. It doesn't seem to matter to muscle tissue. It does

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DAN'S DEVIANT DELIGHTS

Sunset Bull $49.95
Palm Drive Video
800-736-6823

Chris Duffy, for those who don't remember, is an IFBB professional bodybuilder who stopped competing and started doing gay hustling and performing in gay porn videos. Under the screen name of Bull Stanton, Chris generated some minor excitement in the gay porn industry because of his size and conditioning (and unlike Bruce Patterson, has a decent-sized dick). Additionally, he supposedly is able to do sexual tricks, like being able to accept unusually large fists and dildoes up his butthole.

Before I go on, I'd like to mention that Chris has been one of my closer friends, and he and his wife lived with me in Venice in 1985. So I have seen Chris through various transformations: bum, personal trainer, gym owner, Compton slum landlord, and his return into bodybuilding competition that finally netted him his pro card.

When Chris turned to hustling and porn as a career, it didn't surprise me, simply because he was easily bored, and he had done almost everything else he had dreamed of doing. In one of our numerous telephone conversations, he said the whole motivation of getting into the gay sex biz came about from his extreme use of injectable methamphetamine. Chris's father was an alcoholic, and not a fun one, so he avoided all recreational drugs throughout his early life (other than steroids). Around about 1990, he became enraptured with LSD, its derivatives and its current guru Terrance McKenna. After the been there/done that phase, Chris restricted his drug use to marijuana. The speed thingy must have happened when I was in prison the second time. In Chris's favor, he seems to be a functional speed freak; he maintains his bodyweight, still trains regularly, and obviously, still able to get his dick hard. On the debit side, when he starts talking, he won't shut up.

He mentioned that his early videos didn't get wide distribution, because many states have laws against pornography, and some of the acts that would qualify seem to be: (male) homosexual sex acts that include ejaculation, along with fisting, and sodomy involving dildoes over a certain size. I believe that the current offering, Sunset Bull, is a sex video that could be acceptable in all states, as Chris is all alone in it, and in all the time that he is jacking off, never ejaculates. Additionally, when he finally whips out a dildo to stick it up his butt, he seems careful to hold it up in front of the camera, between his two hands, kind of a fisherman, making sure that his catch is within the legal limit, otherwise he'd have to throw it back in.

Frankly, Chris looks like shit in this video. Shaven head, unkempt mustache and goatee, untanned body, fully covered with body hair (expect his scrotum and asshole). And he isn't in the best of shape. Chris was considered the least worthy professional bodybuilder, now that Ray McNeil is dead, as he always had small arms and shoulders, regardless that he weighed, in contest shape at about 260 pounds. Chris is a tall man, well over six feet, and he has some pretty meaty thighs. I point this out, because it puts his cock in perspective. He claims a ten-incher, and he very well may be at, or near that, but contrasted to his overall girth, it doesn't appear exceptionally large. Put the hose on Lee Priest, and it would look Guinness-sized.

I wish I could reproduce some stills from the video, but Palm Drive has put some anti-copying safeguards and my Snappy frame grabber informs me that it's performing an illegal operation and the software shuts itself down. The best I could do, is snap a few digital stills off my big-screen television.

I can't tell you if Sunset Bull is a good gay porn video. One big hairy guy beating off for 90 minutes tends to be boring. At least in other Palm Drive solo jack-off videos, other guys keep my attention better, like the guy that slipped an enormous hollow dildo over an Olympic bar racked on a flat bench and then impales himself on it.

from the desk of
Daniel Duchaine, PhD

Steroid Basics Part 5
(continued)

have a beneficial effect on the pituitary, however, enhancing the production of LH. Unfortunately, when anabolic steroids are used, natural epitestosterone levels fall. It should be beneficial to replace epitestosterone at least to normal levels, or perhaps to the normal ratio with testosterone.

For those who might wish to make it, this may be done by inversion of the -17 hydroxyl after tosylation. It is, however, a controlled substance, even though it is not an androgen.

A second drug to be considered, not in the taper itself but during the heavy phase, is RU486 (mifepristone.) This anti-progestin blocks the long-term inhibitory activity of progestogenic androgens such as trenbolone. It is unknown, though, whether the full anabolic effect would remain or not.

Nalbuphine (Nubain) in certain instances could be very valuable in recovery. Where GnRH secretion is at an excessively slow pulse frequency, nalbuphine should correct this. Naloxone is the drug which has been studied for this, but nalbuphine, a delta opioid antagonist / kappa agonist, should work about as well or better. LH release can be increased by progesterone, as mentioned before, but also by deoxycorticosterone (cortexone, descortexone.) This drug is a mineralocorticoid, like aldosterone. Triamcinolone acetonide also increases LH release; this drug is a fairly commonly used glucocorticoid.

To renormalize the pituitary at the end of the cycle, these drugs would not be used continuously, but in a pulsatile fashion. Sublingual delivery would probably be best.

Tetrahydroprogesterone seems promising. This metabolite of progesterone increases the release of GnRH from the hypothalamus via action at the GABA type A receptor. It is quite possible, but not yet known, that it may not have the long term inhibitory properties which progesterone itself has, or not to the same degree. Melatonin, in a low dose, such as 2 or 3 mg before bed, might be of some value and would not be of harm. More would not be better, however.

All too frequently, a bodybuilder will begin dieting heavily at the same time that he ends his steroid cycle in an effort to lose fat gained during bulking up. This can be a serious mistake. Recovery of natural hormone production is considerably more difficult with a low calorie diet.

Yohimbine should probably be avoided at the end of a cycle. This alpha-2 adrenergic antagonist can reduce the pituitary's ability to produce LH surges. I know of no evidence that this has any relevance under normal conditions, nor that it definitely can be a problem for bodybuilders at the end of a steroid cycle. But nonetheless it is probably wise to minimize all possible obstacles to recovery of the natural hormonal axis.

Comments DD: A few more facts about progesterone. Progesterone increases the amount of 17B hydroxysteroid dehydrogenase, which is the enzyme that converts androstenedione into testosterone. It also increases sulfotransferase, which sulfates estrogen, making it less active to the estrogen receptor, and increases its excretion from the body.

Progesterone also down-regulates estrogen receptors. It is thermogenic, as it acts as an uncoupler of oxidative phorsphorlation (like DNP does). Unfortunately, progesterone causes hyperglycemia, from lowering insulin senseitivity. The result is bodyfat deposition, and for some reason, it happens in the belly area. The synthetic progesterone, Megace, was the first drug used to treat wasting syndrome. The increase of bodyweight is called among PWAs as the "pot-bellied" look. Patrick Arnold has postulated that 100mg of oral pregnenelone would be converted mostly to progesterone.

The Anabolic/Catabolic Diet *(continued)*

to be esterified in the subcutaneous adipose tissue not inside the muscles. I know the ABCDE diet tells otherwise, but oh well... Even though you are not eating carbs, don't forget the creatine. It will find its way in the muscles without insulin. **The oral anabolics will be started during the first day while the injectables will be used as late as possible the day before.** Along with the carb up, use creatine and a sulfonylurea (I like Glipizide: 2.5 mg before the meal if you are new with it). In the first few days, eat a high carb, low fat and moderate protein diet. The protein should be of the highest quality possible to make up for the moderate amount. The more protein eaten the sooner the ATP-dependent proteolytic pathway will recover.

Of course the thyroid medication, the clen, the aerobics and the overtraining are stopped. In fact, it is better to reduce training to a bare minimum in the first few days. During the remainder of this phase, do not eat too much. In fact, you should just eat over maintenance. ..unless you want to gain fat cells! I do not think overfeeding builds up muscle. My belief is that the measuring techniques are biased.

Whenever you eat too much and gain weight, they are going to tell you have gained muscle mass along with the fat. But whenever you diet it down, the bias will take place the other way round. The apparatus will tell you you have lost muscle along with the fat. Your overfeeding gains just evaporated.

The length of the anabolic phase is determined by the length and the severity of the catabolic phase. The shorter the latter is, the shorter the former should last. Of course, this kind of radical cycling is much more appropriate for steroid users than for drug free bodybuilders. It allows anabolics to keep working for a long time as it is frequently stopped during a short catabolic phase. This will fully restore their potency. In case you are a drug free bodybuilder, it is better not to go to the extreme. Shoot for a moderate catabolism not an extreme one.

Number 5 • November 1997

Dan Duchaine's

DIRTY DIETING NEWSLETTER

MILITANT MUSCLE GROWTH AND FAST FAT LOSS

UPDATES FROM THE UNDERGROUND

(This issue, our UK steroid dealer elucidates on a follow-up about buying steroids by mail order.)

Most people are more concerned about the legal ramifications of their steroid purchase than they are about the quality of their products. This, combined with the minimal effort involved and their impatience, leads them to buy from their local black-market dealer.

The chances of getting busted during this kind of locker room transaction are relatively low. The main risk is getting pulled over during the drive home; so make sure your purchases are well out of sight whenever they are in your possession. It's quite hard to find real products in

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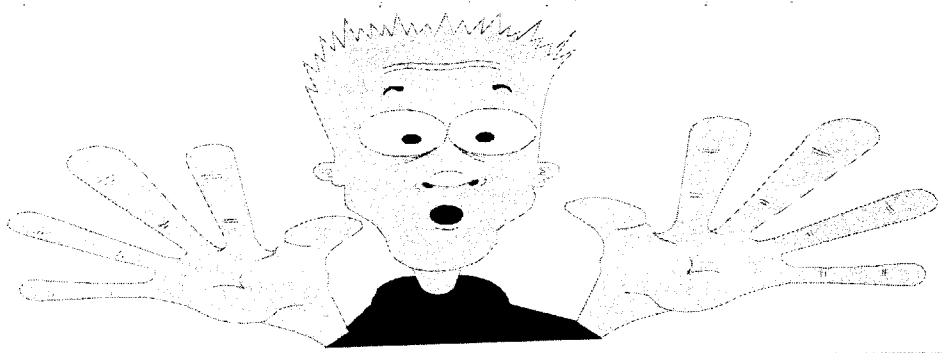
IN THIS ISSUE



HAIR LOSS TREATMENT PART ONE

Philipides & G. Gionis MD

About 90 percent of a person's scalp hair is continually growing, a phase that lasts between two and six years. 10% of the scalp hair is in a resting phase that lasts between two and three months. At the end of its resting stage, the hair is shed.

Shedding 50 to 100 hairs a day is considered usual.

When a hair is shed, a new hair grows from the same follicle. Scalp hair grows about one-half inch a month. Natural blondes typically have more hair (140,000 hairs) than brunettes (105,000 hairs) or redheads (90,000 hairs). The rate of hair growth slows down with age. The building material of hair is a form of protein, the same found in nails. **Thus protein builds not only muscles but hair too.**

Abnormal hair loss can be due to many causes.

- **Childbirth.**
- **High fever, severe flu**
- **Thyroid disease. Both an over active and underactive thyroid can cause hair loss.**
- **Inadequate protein in the diet (bodybuilders are excluded for obvious reasons).**
- **Cancer treatment drugs.**
- **Birth control pills.**
- **Low serum iron.**
- **Medications (some: blood thinners, some drugs used to treat arthritis, depression, heart problems, hypertension, high doses of vitamin A, AND... most of the anabolic steroids used in high doses).**

Hereditary thinning or balding. It's the most common cause of thinning hair. The endency can be inherited from either the mother's or father's

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from the desk of
Daniel Duchaine, PhD

Dan Duchaine's

Dirty Dieting Newsletter®

Militant Muscle Growth and Fast Fat Loss

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HAIR LOSS TREATMENT
(continued)

side of the family. Women with this inherited trait develop thinning hair, but do not become bald. Steroids accelerate this process.

Androgenic alopecia (AGA), the pathogenesis of AGA involves increased scalp follicle sensitivity to androngens. Scalp follicles in AGA contain increased levels and activity of 5a-reductase, the enzyme that converts testosterone to dihydrotestosterone (DHT).

DHT shortens the hair cycle and miniaturizes scalp follicles. Recently, two isoenzymes of 5a-reductase have been identified in human tissue. The type 1 isoenzyme is found in scalp skin, whereas 5a-reductase type 2 is the predominant form in prostate. Differences in pH optima, substrate Km (it regulates the rate of an enzyme-catalyzed reaction) and sensitivity to inhibitors can distinguish these isoenzymes.

Hypogonadol men do not become bald, and hair loss can be induced by testosterone (T) in these individuals. Male pseudohermaphrodites with 5a-reductase deficiency do not exhibit male pattern baldness, suggesting that DHT is the active androgen in the development of hair loss. Increased information of 5a-reduced metabolites of T has been shown in bald scalp compared to that in hairy scalp. Topical administration of the 5a-reductase inhibitor and antiandrogen N,N-diethyl-4-methyl-3-oxo-4-aza-5a-androstane-17B carboxamide prevented the development of baldness in the stumptail macaque, an animal model of male pattern baldness.

PROSCAR (finasteride) was introduced in 1989 and has demonstrated few adverse effects. Such effects occur in fewer than 5% of patients and usually relate to decreased libido and impotence. Up to a 25% reduction in semen volume can be expected in some patients without any changes in sperm counts, motility, or morphologic features. Finasteride lowers serum levels of prostate-specific antigen, a laboratory test used to monitor benign prostatic hypertrophy and screen for prostate cancer, since elevated prostate-specific antigen levels occur in both benign prostatic hypertrophy and prostate cancer.

Finasteride is a potent inhibitor of human 5-alpha-reductase type 2, yet devoid of antiandrogen activity so the circulating levels of testosterone are not affected. Finasteride, manufactured and marketed by Merck Pharmaceutical as Proscar, has been shown to be effective in the treatment of benign prostatic hyperplasia. At the therapeutic dose of 5 mg/day, finasteride lowers serum dihydrotestosterone levels in men by 65-80% compared to baseline levels and decreases intraprostatic levels of dihydrotestosterone by 85% compared to placebo.

Long term therapy with finasteride may progressively inhibit the type 1 isoenzyme, as suggested by in vitro studies showing high dose finasteride inhibition of type 1 isoenzyme. Although type 1 5a-reductase is the dominant enzyme form in the scalp, the fact that AGA does not develop in men with congenital 5a-reductase deficiency (defective type 2 5a-reductase only) suggests a role for type 2 5a-reductase in AGA.

It is not known if inhibition of both

5a-reductase isoenzyme types are necessary to promote hair growth. Finasteride was shown to be nearly as effective in animal models of AGA as N, N-diethyl-4-methyl-3-oxo-4-aza-5a-androstane-17B carboxamide, an inhibitor of both isoenzyme types. Prolonged exposure to finasteride may be necessary for clinical effectiveness; 6 months of treatment is required for maximal response in benign prostatic hypertrophy, and a similar gradual response pattern may occur in AGA.

The effect of finasteride on lowering of scalp skin DHT could be mediated through one or a combination of mechanisms. The DHT concentration in full thickness scalp samples may be dependent in part on circulating levels of DHT, because scalp skin is highly vascularized. Finasteride may exert part of its effect on scalp levels by lowering serum DHT. Residual DHT in scalp after finasteride treatment may represent the local activity of the 5a-reductase type 1 isoenzyme, which is less effectively inhibited by this drug.

Alternatively, it is possible that finasteride can effectively inhibit 5a-reductase type 1 in vivo after chronic treatment. Although finasteride is a more potent inhibitor of 5a-reductase type 2, inhibition of type 1 is seen at higher concentrations in vitro.

From a study (in 1995), designed to help determine the optimal dose of finasteride for use in male pattern baldness, we have some interesting results. A six-week trial measured concentrations of scalp-skin and serum DHT, using oral finasteride at doses of 0.01, 0.05, 0.2, 1 or 5 mg per day or placebo. Finasteride at doses of 0.01, 0.05, 0.2, 1 or 5 mg per day suppressed scalp-skin DHT. In men taking 0.05-5mg per day of oral finasteride, scalp-skin DHT was reduced between 56 and 69 percent from baseline. In men taking placebo, scalp-skin DHT decreased by 13 percent. Other than the group taking 0.01 mg, no significant differences between the doses were seen; however, significant differences were seen between the groups that received finasteride (not including 0.01), and the group that received placebo. Effects on hair growth were determined in a 6-month trial using doses of 0.01, 0.2, or 1 mg per day or placebo.

New data from three studies in Australia show that Propecia (the Australian brand name, finasteride 1 mg), Merck & Co. Inc.'s investigational oral treatment for men with male pattern hair loss, prevented further hair loss in treated men and increased hair growth in both frontal and vertex areas of the scalp in many men. **In those studies, 86 percent of men maintained or showed an increase in the amount of their hair based on hair counts during the course of the studies, compared with 42 percent of men receiving placebo.**

The application for Propecia is under review by the U.S. Food and Drug Administration. Upon clearance, Propecia would be the first oral treatment taken once-a-day for the prevention of further hair loss and for regrowth in the most common sites of hair thinning in men.

Upon marketing, it will be indicated for use in men only.
Side effects were infrequent and occurred in a small number of

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Dan's Internet Surfing

Shelly Hominuk

http://www.geocities.com/Colosseum/Field/4992/weights.html
a.k.a (The Iron Dungeon).
Similiar to adult sex links, I had a hard time leaving this site. Extensively informative, the site is totally awesome in ALL aspects pertaining to the bodybuilding industry, and then some.

http://www.elitefitness.com/Anabolix/fakes.html
This is one of the very few links with balls to address the counterfeit steroid issue; and will even extend identifying assistance to those in dire need. All aspects pertaining to steroids are nicely covered here.

www.cyberiron.com
Here's another great link to gather a shitload of info in aspects of drugs, newsgroups, supplements, training, books, yada, yada, yada.

http://heml.passagen.se/daho1000/
a.k.a (David's Steroid Page).
If you're new and ever hungry for steroid info, you may be orgasmic over this link. Nice pics, nice descriptions. Even those who may yawn might pick up a thing or two.

http://www.moth.com/statement.html
a.k.a. (Men of the Hour - MOTH)
Little faggots will be stoked to know that this link got both thumbs up (my ass of course!!).

Number 6 • January 1998

Dan Duchaine's
DIRTY DIETING
NEWSLETTER

MILITANT MUSCLE GROWTH AND FAST FAT LOSS

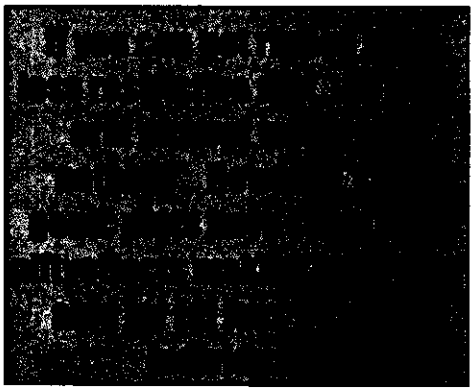
Anticatobolic Effects of Ketogenic Diets

IN THIS ISSUE

Oliver Starr

Last issue we examined the physiology behind ketogenesis (ketonebody production) during a zero carbohydrate diet. To briefly recap, when liver glycogen becomes depleted (as a result of carb restriction and intensive exercise), insulin drops, glucagon increases, free fatty acids mobilize from fat stores where they are burned in the liver producing acetyl-CoA which is then condensed into ketones which float around in the bloodstream providing an alternate energy source for the body since glucoseis not available.

Physiology aside, what benefits does a cyclical ketogenic diet such as Bodyopus have over a normal moderate carb, high protein, low fat diet?



The biggest, and the one we'll discuss in this issue, is the possible anti-catabolic effects of a ketogenic diet.

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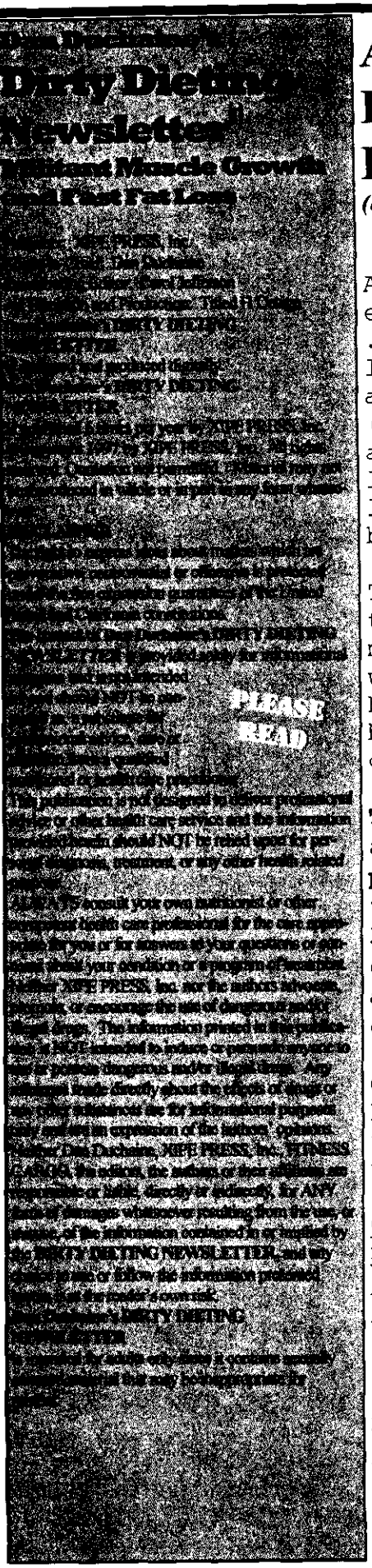

Anticatobolic Effects of Ketogenic Diet

(continued)

All diets are inherently catabolic .Calorie restriction lowers levels of anabolic hormones (insulin, IGF-1, etc.) and increases muscle loss, especially if large amounts of aerobics are done.

The best we can hope to accomplish is to minimize muscle loss while dieting (or rebuild what we've lost but I'm getting ahead of myself).

This is generally accomplished with super high protein intakes or supplements like the ephedrine/ caffeine/ aspirin stack or clenbuterol.

Of course, many bodybuilders use steroids to prevent muscle loss while dieting.

Other than the hormonal reasons, what is the cause of muscle loss while dieting? In very general terms, it's due to the breakdown of protein to make glucose(through a process called gluconeogenesis which literally means "the making of new glucose". Why would this occur though? Why would the body deliberately break down its own muscle?

As far as the body is concerned, the brain (which is a major glucose hog, using up to 25% of the total glucose used in the body) is a more important tissue to maintain than muscle.

So, when calories are lowered (and carbs decrease by extension), there is less glucose in the bloodstream. But, the brain still wants its glucose.

The difference between what's available from the diet and what the brain needs is made up by breaking down protein and converting certain amino acids to glucose.

What about fat? Why won't the body just use bodyfat to fuel the brain? Because fats are *unable* to cross the blood brain barrier. Simply, the brain can't use free fatty acids (FFA) for fuel on a diet.

So where does ketosis come in? As mentioned in the last article,

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from the desk of
Daniel Duchaine, Ph.D

LOOK IT UP YOURSELF YOU LAZY BASTARD

Some of the below resources are free, some are on a per use basis, some require memberships or subscriptions. Prices can vary from as little as $12 for the complete text of a specific document to as much as $1200 per year for a subscription to current contents.

Medline found on the internet at:
http://www.healthgate.com/
This is an enormous database of medical research studies that have been published in peer reviewed journals. It has (at last count, over 14 million studies online) searches for titles and abstracts are free, but you pay between $12 and $24 for each article for which you order full text. A service then copies and mails or faxes you the requested pages. Many medical libraries have free access, and you only have to pay photocopying charges for the full text.

Chemical Abstracts found on the internet at:
http://www.cas.org/
This is in some respects the holy grail of chemically oriented research. Formerly we had to go to the medical libraries to use this resource, but thanks to the miracle of the internet, we can peruse it at our leisure. Searches are on a per "hit" basis, and also based on usage time. Just guessing but this is probably Pat's personal favorite. One benefit to using this web site is that it also has a dozen or so links to other research oriented sites.

Current Contents: This service requires an annual subscription costing roughly $1200. Despite its cost, it has numerous advantages. Subscribers receive an index CD ROM which is updated every few months, and a weekly CD containing new abstracts from several hundred journals. This is by far the most up-to-date information source. These same abstracts will eventually be uploaded to Medline, but generally that requires 4 to 6 months, so if I'm onto something really hot this is where I go first. Again, complete text must be ordered via internet or fax at a per document charge of $8 to $12.

The Merck Index: This is the most complete book of chemical compounds anywhere. Items are listed alphabetically and include virtually all pertinent information about every compound. If you want to know the melting point of androstenedione, this is where you should look first. You can find the Merck at any good bookstore, or at your library. Cost: about $75.

Dialog: found on the Internet at:
http://www.dialog.com/
This is one of those enormous databases that covers virtually every subject. Dialog can help you locate information about pharmaceuticals, chemicals, FDA regulations, you name it. The two major drawbacks to Dialog are refining your search strategy and the cost. Dialog is a fee based service and costs $24 per hour of use. I tend to go here as a matter of desperation when my other searches are yielding little useful information.

The Physicians Desk Reference: Another comprehensive text devoted to pharmaceuticals. It contains reasonably complete drug information including prescribing guidelines, contraindications and known conflicts with other drugs. It's available at bookstores nationwide, you can sometimes find it for as little as $39 but it's usually upwards of $50. The PDR is revised every year. You have the option of keeping the older version and ordering the updates, or buying the current one. Many bookstores will sell the previous year's PDR for as little as $10.

The Sports Science Web Site:
http://www.sportsci.org/
This site is primarily devoted to the discussion of sports science related issues. It does occasionally

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Anticatobolic Effects of Ketogenic Diet

(continued)

ketones are primarily brain food, especially after the first few days of the diet.

But you contend, aren't ketones just an end-product of fat metabolism?

Why can they be used in the brain while FFA's can't?

This is the magic of the state of ketosis. Ketones are essentially water soluble fats which CAN cross the blood brain barrier.

Ketones are made available as an alternate energy source when carbs aren't available for the brain (but only when the metabolic shift takes place).

Think about this for a second. Under normal dieting conditions, the body breaks down protein to provide for the brain's high glucose requirements. Under ketogenic dieting conditions, the brain no longer wants glucose, it has plenty of ketones which it can use for fuel. Also, the high rate of FFA breakdown produces glycerol which can be converted into glucose as well.

The end result? Less bodily protein will be broken down to make glucose for the brain. Which means less muscle loss while dieting.

There have been several studies that do not support the anti-catabolic role of ketones in the body. Upon close inspection, these studies have flaws.

In one, ketones were infused via an IV, not generated in the body. This means that blood glucose was normal and the body was still using glucose, not ketone metabolism. So, ketones wouldn't be expected to spare protein.

In other studies, generally using Very Low Calorie Diets (VLCD) of 400-800 calories per day, ketogenic diets didn't do any better at sparing muscle loss than a high carb diet (don't forget that insulin is also anti-catabolic).

However, extrapolating from a 400 calorie starvation diet to a slightly below maintenance calorie level is inaccurate.

In a VLCD situation, some muscle will be lost no matter what the diet.

A study comparing a 10% deficit ketogenic to a 10% deficit carb diet would be most illuminating but good luck on ever seeing it. Other studies have shown that muscle loss is related to inadequate dietary protein intake.

A value of 1.5 grams protein/kg body weight has been established as the minimum for a ketogenic diet to spare muscle loss, especially during the first weeks when adaptations are taking place. But that was for sedentary individuals. Most bodybuilders consume 2 grams/kg or more of protein (about 1 gram/lb.) anyhow so it's not a huge issue.

Finally, ketones appear to have a slight insulin-like effect themselves. This is not entirely understood, simply realize that ketones in high concentrations may exert an insulin-like effect on muscle cells, helping to prevent muscle loss.

LOOK IT UP YOURSELF YOU LAZY BASTARD

(continued)

have interesting information about doping and drug controls.

It also has a somewhat more useful list of links that often yield bits of otherwise unknown data.

Search Engines: These do not have any specific sports or drug oriented information, however they can prove quite useful if you are willing to devote the time to checking out the dozens of "hits" listed for any given key word. When I have the luxury of extra time (which is rare) I will occasionally do a keyword search on, for example, steroids. This yields some 427,000 odd "hits" listed in order of relevance. It's then up to you to go through and determine which ones are likely candidates for having the information that you are seeking. The popular ones are:

Yahoo:http:/www.yahoo.com/
Lycos:http:/www.lycos.com/

Webcrawler:
http://webcrawler.com/

Infoseek:
http:/www.infoseek.com/

The Food and Drug Administration Home Page:
http://www.fda.gov/

Another fun one (for good stuff on what's about to be banned) Science and Engineering News:**http://www.ari.net/senn1.html**

This has miscellaneous science and engineering related information. Rarely useful for my purposes, but your needs may vary, so I thought I'd include it.

Trademark Searches:
http:/www.questel.orbit.com/
This one can come in handy when you are looking to name a magazine or a product and don't want to end up in court because you ripped off someone else's trademarked name.

The Herbalist: CD ROM. This is an amazing compilation of facts, pictures and scientific and historical information on herbs. It's one of my favorite tools. It was available in CD ROM, but it's my understanding that it may now be unavailable. Keep your eyes peeled for a re-release, though, I expect one out soon. $75.

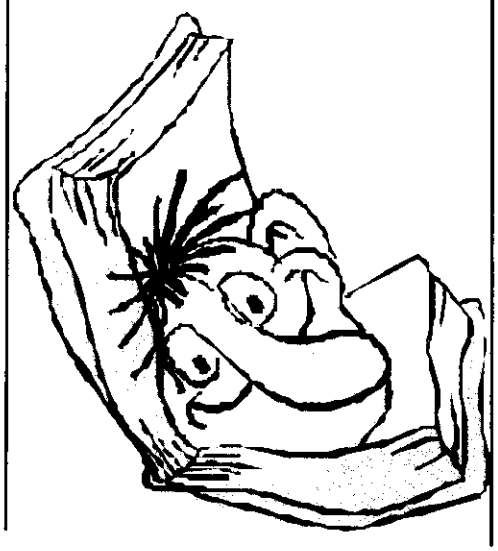
The Material Medica:
This book detailing numerous herbs and their current and historical use. This can be difficult to find, but worth the $45.

Your Local Library! My least favorite, because it means that I have to get in my car and then waste 45 minutes trying to find what I want, only to discover that it's either lost, checked out, or they no longer subsribe to that journal. Nevertheless, for those of you without a computer or without subscriptions to a number of the major journals, this is both free and better than nothing. An added bonus is that the research librarians in the medical libaries can be helpful if you reach a dead end. And I might get a quick bang in the stacks.

This is not every available way to get data, but I'll wager that if you can't find at least one good solid lead using the above resources, you're either barking up the wrong tree or you need to check your spelling.

(Editor's note: The US patent web site has been useful to me, especially since the supplement industry has a new trend of using use-patent to make claims for products.) The URL is: ***http://patents.uspto.gov/pto/classes.html.***

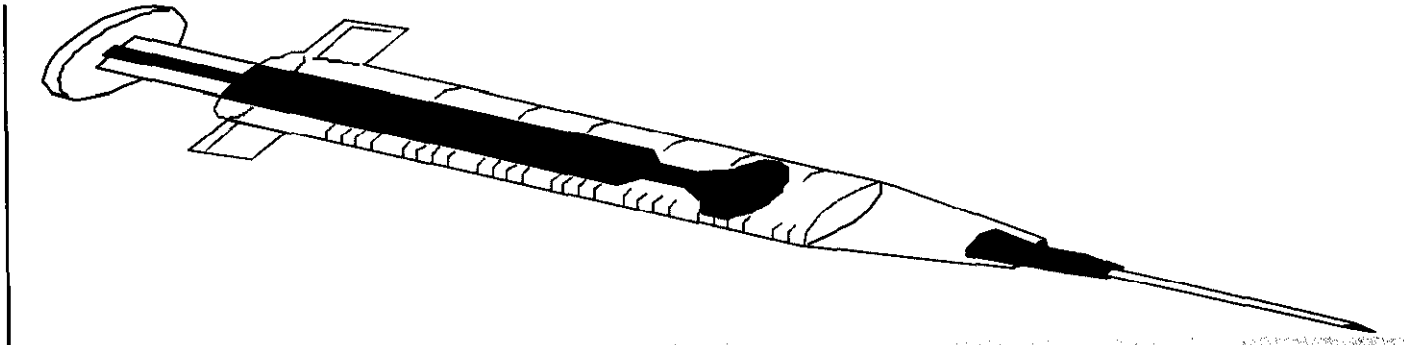
As Oliver mentioned, some of the just-published research doesn't make it to Medline immediately. Usually the major newspapers in the country will mention noteworthy health and medical advances. Lately, the most interesting medical stuff has been appearing in NATURE and NATURE GENETICS. If you want to cover all the bases, most yellow pages will have a listing for "Newspaper Clipping Services"; these companies clip and mail specific subjects from numerous newspapers.



from the desk of
Daniel Duchaine, PhD

Number 7 • March 1998

Dan Duchaine's
DIRTY DIETING NEWSLETTER
MILITANT MUSCLE GROWTH AND FAST FAT LOSS



Androgen/Insulin Synergy

IN THIS ISSUE

Michalovich Dharkam Greutstein

Should anabolics be used with insulin or is it best to use insulin while off steroids in order to hold on to muscle mass?

We are going to demonstrate they have to be used together. We will also try to provide some clues about their respective contribution to the synergy both hormones create. This will help us to handle both drugs better.

Here are some general observations:

Anabolics work much better while on a high calorie diet. Anabolics do not work very well while on a low calorie diet even if protein intake is high.

It is safe to conclude something else is needed to uncover the full anabolic effect of steroids.

The hormone which is the most affected by a high calorie or by a low calorie diet is insulin.

Also, heavy steroid users know that past a certain amount of steroids, adding insulin will make a big difference as far as muscle gains are concerned.

Insulin is thus a strong candidate as a potentiator of anabolic steroids (which we will indiscriminately refer to as androgens, steroids or anabolics).

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from the desk of
Daniel Duchaine, PhD

Dan Duchaine's

Dirty Dieting Newsletter(r)

Militant Muscle Growth and Fast Fat Loss

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Androgen/Insulin Synergy

continued

Furthermore, studies performed in trained dogs have shown a lack of insulin completely negates the anabolic effects of steroids on protein synthesis.

There are some easy hypotheses such as a possible androgen receptor upregulation, a stimulation of androgen secretion, an anti-aromatase effect arising from insulin. But there is still something missing.

Using anabolics plus insulin will not make you much bigger unless you weight train. The synergy can only be realized if insulin + steroids + training are present. What is the link between those three factors?

A very likely candidate is an enzyme called insulinase. As its name implies, it is an enzyme responsible for the destruction of insulin. But we are going to see it does much more than that.

It is found inside many tissues of the body, particularly in muscle. What science is telling us is that insulinase is essential for insulin to provide its anti-catabolic effect on our muscles. It is also likely that insulinase is able to multiply the anabolic effects of androgens.

It's worth repeating: insulin cannot stop protein catabolism without insulinase and the effects of steroids are potentiated by insulinase. It sure looks good.

Androgens are very powerful stimulators of the muscle protein synthesis rate. On the other hand, the muscle gains provided by androgens do not match this elevation in synthesis. Steroids promote anabolism to a much higher rate than they make our muscles grow.

The reason for this discrepancy is that they also stimulate protein degradation. I know many people think they are anti-catabolic but it is not the case. **Anabolics stimulate protein turnover.** This means they increase both synthesis and degradation of proteins. They are simply more effective at stimulating synthesis than degradation, which is why they make our muscle grow but not at a superfast rate.

Look at how long it takes to grow huge muscles. If androgens were stimulating synthesis while inhibiting degradation, one would grow very, very quickly.

This is where insulin comes in. As we said, it mostly reduces protein degradation rate. It might stimulate protein synthesis right after training but this effect is very limited in duration.

Ideally, using insulin along with steroids would allow us to accelerate synthesis (thanks to anabolics) and reduce degradation (thanks to insulin). This is the best way to grow muscle fast.

Unfortunately, as both insulin and anabolics need insulinase to work better, they will compete against each others for this enzyme. For natural athletes, the supply of muscle insulinase should roughly meet the demand. Now if you add anabolics, there will be less insulinase for insulin. If you do not take too high a dose of steroids, the level of insulinase should still be sufficient to allow a fair insulin-induced anti-catabolism.

But as you take more steroids, the insulinase available for insulin will be lower and lower.

Insulin will lose its anti-catabolic effect. As it will still bind some insulinase, the enzyme availability for steroids will not be optimal either. Anabolics will lose some of their potency.

What is important to understand is that past a certain dose, anabolics will provide their own antidote against muscle growth. The only solution (beside using less steroids) is to increase insulinase level.

At least two factors can accomplish this feat:

The first one is insulin itself. The higher the insulin level is in a target organ (muscle for example) the higher the insulinase level will be. You would expect that the body would detect the shortage of insulinase for insulin and so produce more insulin (or more insulinase).

Unfortunately, this does not seem to be the case. While insulinase is crucial for the anti-catabolic effect of insulin it does not seem as important for glucose disposal.

Insulin's main function is not to assist in muscle growth but to control glucose homeostasis. As a result, it is likely our body does not really care about a relative shortage of insulinase. In any case, we are left with a less than optimal equilibrium. It is up to the bodybuilder to react to this imbalance.

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Dan's Internet Snips

Shelly Hominuk

http:/www.geocities.com/hotsprings/4039/gymdata.htm

"World Wide Gym Database". Planning a business trip or vacation and want to know which gyms are worth checking out within the vicinity? This is an excellent idea of a database of gyms around located the world. This site offers several names of gyms, how they rate on a scale of 1 to 10, the address, city, state, country, phone number, hours of operation, cost of visits, quality of equipment, atmosphere, type of crowd, other services such as tanning, bodyfat checking, juicebar, aerobics etc. Suggested to also report personal gym visiting experiences to help add to this site's database.

http://www.kuai.se/~jbartoll/iron.html

"Thorax's Iron Page" This is a noncommercial site located in Sweden where you can find "uncensored hardcore bodybuilding" information. Not only can you download the glycemic index from here, but you can also retrieve various articles featuring information such as steroids, prices, tradenames, training, supplements, recipe s, etc. Of particular interest is the fact that you can find listings of some discovered companies, mail order scams,

continued on page 4

Dan's Internet Snipets
(continued)

dealers, and individuals, which are uncovered counterfeit organizations.

http://www.bypass.com/~twilbur/moves.html

Here you will find an illustrated guide to exercises with free weights.

This is an extensive list of several (not a few) but many, many exercises that can be performed for the abs, back, biceps, calves, chest, forearms, neck, shoulders, thighs and triceps. Covers practically every exercise known to mankind.

Reacquaint with old techniques, learn new ones, or simply just to add some variety back into your slacking training regimine.

Androgen/Insulin Synergy
continued

One way of increasing insulin secretion is to eat more but you can only do so up to a point. You cannot increase your carb intake in parallel with the amount of steroids without getting too fat. Another solution is to use drugs to add or to stimulate insulin secretion. This way you get the insulin without the excess of calories.

In any case you now understand why steroids work better while on a high calorie diet while they lose their potency during a diet or a shortage of insulin.

Here is a way of artificially increasing insulin level: One dose of long acting insulin first thing in the morning (this is the only injection). Before each meal (except the pre-workout one), take a sulfonylurea (an oral anti-diabetic drug which will boost food induced insulin secretion). I like Glipizide because of its short half-life. In case you experience hypoglycemia, you know it will not last. This is the main problem with the long acting sulfonylureas. When you are hypoglycemic, you try to compensate by absorbing carbs. But the drug will make your pancreas secrete even more insulin before the carbs can hit the blood. It makes the hypoglycemia worse not better.

In case of problems, make sure you get some ready-to-inject glucagon (sold as "insulin emergency kits" in drugstores). An additional the benefit of Glipizide is, it induces the release of GH on top of insulin which is beneficial for non diabetics.

This is a nice way to fix the reduced anti-catabolic property of insulin. Unfortunately, this will not yet provide the optimal amount of insulinase to have steroids work better.

We said that training was the third key ingredient in this synergy. This is because training can stimulate insulinase activity. **Not any exercise will do. The traumatic ones inducing muscle soreness are the most effective.** It is the factors inducing soreness which will trigger this increase in insulinase.

On the other hand, you do not want to create too much soreness as it will temporarily reduce the effects of insulin and androgens by

impairing their effects at the level of their respective receptors. What you want is mild but frequent soreness along with some very frequent pumping sessions.

Do not forget both androgens and insulin circulate in the blood. The more blood you get into the muscles (and the longer it stays), the more your muscles will "drenched" in those two hormones.

Please note that insulinase is produced locally in the trained muscles only. It does not circulate into the blood.

We cannot stress enough the need of frequently training each muscle while on steroids.

Q&A

Q In DD #2 you printed Pat's formula for making testosterone from androstenedione. If I substituted norandrostenedione, would I get durabolin (nandrolone) instead?

A Yes.

Q What's up with that yohimbe cream I saw in the classifieds last month?

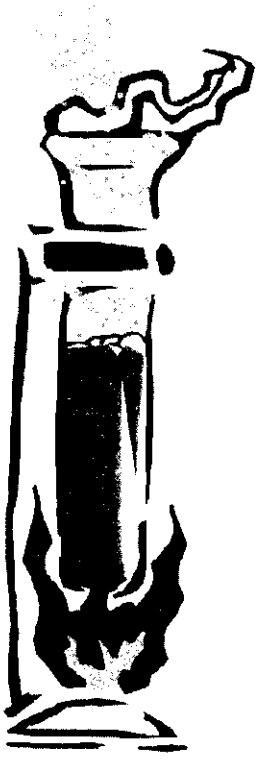
A I always wondered why nobody came out with a yohimbine cream after the Greenway study, showing that some topically applied drugs can reduce thigh girth. All the thigh creams in years past were based on aminophylline, which seemed, in the study, to best reduce thigh circumference from all the drugs tried. However, the user feedback from the fat chicks that used the aminophylline creams, was that as soon as they stopped using the cream, the thighs got fat-looking again. I wouldn't be surprised that the reducing effect might have come from a local diuretic effect, expelling some of the water out of the skin and fat cells. And from a marketing standpoint, it's just good business sense to use something that requires continued use for its effect.

Yohimbine did show some significant thigh size reduction, and I feel that it probably is true fat loss, from a mobilization of fatty acids from the fat areas.

At least the research shows that this does happen with injection. Oral Yohimbine isn't ideal for this, as fatty areas don't have much blood supply (which is why the fat is not reddish-brown looking).

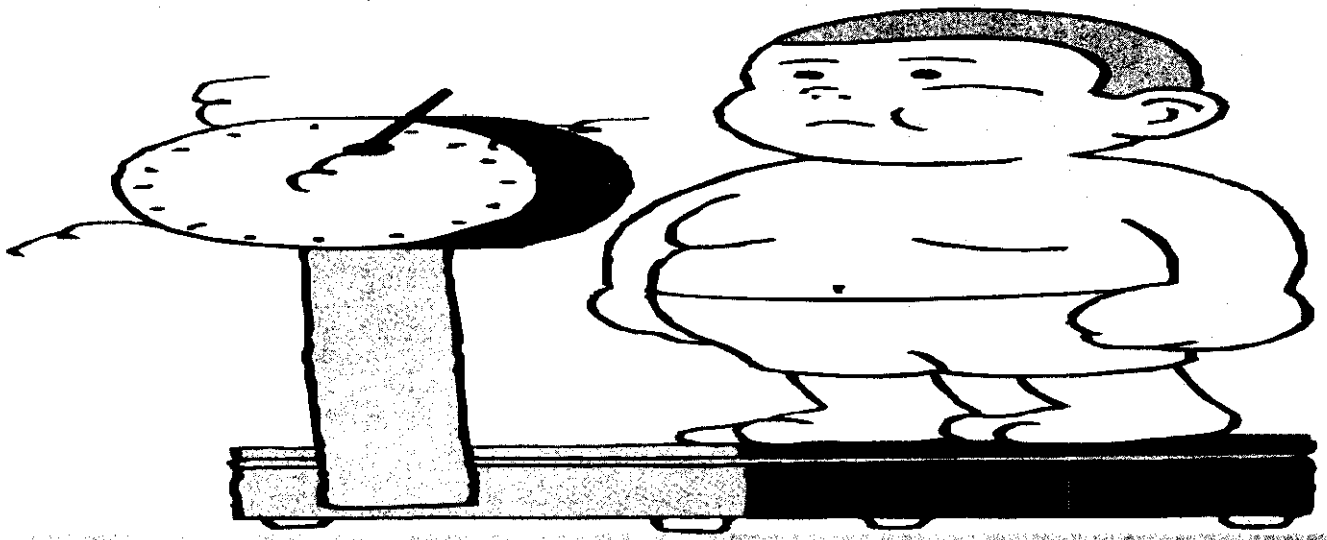
When I wrote my BODYOPUS diet book, I put a yohimbine cream on my wish list. And eventually, an European cosmetic company responded, giving me some examples of a Yohimbe extract cream. Every person using the cream responded positively about its spot reduction effects. Some of the users are men who used the cream on their love handle areas.

I haven't the time to sell the cream myself, so I arranged for my girlfriend to import the cream and sell it. The yohimbe extract cream now has a name: Yo-Be-Lean. We've estimated that one jar would last about 30 days, with a daily application. So far, most of the initial buyers of the Yo-Be-Lean seem to be reordering the cream, which is usually an indication that the user is satisfied with the result.



Number 8 • May 1998

Dan Duchaine's
DIRTY DIETING NEWSLETTER
MILITANT MUSCLE GROWTH AND FAST FAT LOSS

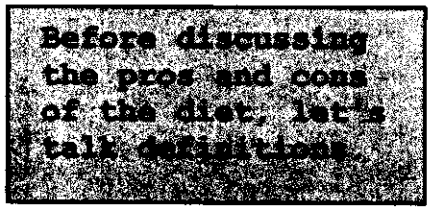


ISO-OPUS ERRATA PART VI

Lyle McDonald

We have discussed some of the metabolic goings-on of a ketogenic diet (including a basic sketch of ketone body production as well as why ketones may spare protein loss while dieting).

What other things must be considered before embarking on a ketogenic (zero carb, moderate protein, moderate to high fat) diet?



Many individuals are familiar with the Atkin's diet or others of it's like (i.e. Protein Power) which are strict ketogenic diets. By strict, we mean that carbs are restricted indefinitely to low levels.

While these diets are appropriate for non exercisers, individuals performing any amount of high intensity activity (i.e. weight training) will lose muscle since training intensity cannot be maintained without dietary carbohydrate intake. Most lifters should consider the

continued on page 2

IN THIS ISSUE

from the desk of
Daniel Duchaine, PhD

Dan Duchaine's

Dirty Dieting Newsletter©

Militant Muscle Growth and Fast Fat Loss

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ISO-OPUS ERRATA PART VI

(continued)

BodyOpus type of diet (generally called a Cyclical Ketogenic Diet or CKD) which alternates 5+ days of zero carb eating (the ketogenic part of the diet) with 1-2 days of high carb eating (which serves to load the muscles with carbs, allowing training intensity to continue).

So, let's look at the pros and cons of the CKD.

Pros of a Cyclical Ketogenic Diet

1. In many people, ketones blunt hunger. It's nice to have a diet where you're not hungry all the time. If there's a problem with keto diets it's that you get less food volume wise because so much of it is dietary fat. So, having hunger blunted is a nice side effect. Amazingly enough, one of the early criticisms of the Atkin's diet was that it blunted hunger too much. Apparently, the powers want individuals to be hungry and miserable when they are dieting.

Tip: Taking the ephedrine/caffeine/aspirin stack with a keto diet is almost guaranteed to shut down hunger.

2. It seems to help women maintain their menstrual cycle. I can't explain this one with research, it's just an anecdotal observation. One female bodybuilder I helped prep kept her period all the way through her contest (she came in at about 7-8% bodyfat). The other girls in her gym had been without a period for months on a low fat, low calorie diet. I imagine the dietary fat helps keep the hormonal axis normal.

CKD also seems to help women lose fat in their lower bodies. The exact reason is unknown at this time, but lowering carbs definitely seems to makes a difference.

3. It helps to maintain sex drive while dieting (which can never be a bad thing). I have a hunch this is because the dietary fat is pushing testosterone production (or, more accurately, keeping it from dropping so much). In fact, many women report an increase in sex drive on the diet (which is definitely not a bad thing), again probably due to

higher testosterone. Add in yohimbe, androstenedione and DHEA to the dieting stack and hang on for the ride.

4. It's Simple to do. The ketogenic diet only allows a hand each day (it can get a bit bland though). One study put the Bantu Indians on the diet and they were able to follow it with no problem. **Some studies found that subjects remained on the diet after the study was over because it was so damn easy to follow.**

5. It allows (hell, requires) a major cheat day once a week. The cyclical ketogenic diet inserts a 1-2 day carb-up period where you can overconsume carbs and not get fat. The bodybuilder mentioned above was much mellower than the other girls in her gym because she could eat her blueberry pancakes and have a margarita every weekend. Tied in to this, the CKD shouldn't cause the post-contest food binges seen in bodybuilders. **Simply, you don't feel deprived since you get to pig out once every 7 days.**

Cons of a Cyclical Ketogenic Diet

1. Expect everyone you know to give you shit about the diet, citing health dangers, dehydration, cholesterol levels, etc., etc., etc.

The misconceptions about the ketogenic diet abound and are a product of ignorance and a lack of research. There is ample human research on the benefits of the ketogenic diet, but it's just not widely known (we are working to correct this problem). Simply accept that you will get funny looks at the store when you buy ground beef, heavy-cream, cheese, oil and Diet Coke during the week and go in on Friday night to get Alpha Bits cereal. Or when you go to a restaurant and order a double cheeseburger without the bun.

2. Along with 1#, don't expect restaurants or anybody you know to make it easy for you to avoid carbs. **Trying to explain to a server why you want some cheese on the side of your meal (but no croutons in your Caesar salad) gets old fast.** Of course, it's ultimately no worse than trying to avoid every ounce of fat they might be used to prepare your meals.

But, most people can understand low fat

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ISO-OPUS ERRATA PART VI
(continued)

diets. Low carb? Not any time soon.

3. The diet does get boring during the week. The limited amounts of foods you can consume during the ketogenic diet get really old really fast. It's basically meat and fat, meat and fat. Did I mention the meat and fat? Of course, you get to eat whatever you want during the weekend carb-up phase and most bodybuilders eat the same foods while dieting anyhow so it's probably a moot point.

4. Expect your training to suffer the first few weeks. **While adapting to the diet, your energy levels will crash big time (at least it does in some people).** After that, you'll actually have MORE energy during the lowcarb week and feel shitty during the carb-up. Go figure.

5. We don't honestly know the long term effects of the diet. The longest any study has examined someone on a ketogenic diet is about a year (epileptic children are frequently kept on the diet for up to 3 years but they aren't a great model for dieting bodybuilders). Most health problems (i.e. heart disease) take years to show up. The CKD should probably be used as an event, to prepare for a contest or a pool party (even a big date) but don't stay on it forever.

6. It's not ideal for mass. **Contrary to the views of some authors, the CKD is not the ideal mass building diet for most people.** Depletion of liver ATP will negatively affect hormonal profiles (as detailed by Darkham a couple of issues ago) limiting growth. The CKD should be considered a fat loss diet only by most people.

Conclusions:

So, is the CKD for you?

- **If you are a woman with lower body fat problems, a reduced carb diet will help with fat loss.**
- **If you're a male who loses too much muscle dieting, the CKD is worth trying since it seems to reduce muscle loss.**
- **If you have the ability to eat the same foods day in, day out (and don't mind everything tasting greasy), the CKD is probably for you.**

from the desk of
Daniel Duchaine, PhD

STEROID BASICS Part 6

Bill Roberts

(Editor's note: Bill wanted to address some accruing question from the last five installments.)

Q: People say that some steroids are androgenic and some are anabolic. They say that androgenic steroids are dangerous, or that the androgenic ones give the best growth. What's the deal?

A: This word is very misused. The parts of the word, put together, mean "making into a man." The word itself is defined as, "Of the nature of a male sex hormone." So this includes all anabolic steroids. Wrong usage of the word may be intended to mean that a drug causes strong side effects of any type, that it is effective, that it causes water retention, or that it is virilizing,that it causes increased aggression, or almost anything. It is vague and almost useless.

Q: But you know what I mean! Aren't they the strongest steroid because they aromatize.

A: No. If this were true, then adding a little estrogen to, say, Primobolan would make it a very strong anabolic. This is not so. And if aromatization really helped growth much, then we'd see much less growth when aromatase inhibitors are used with testosterone. Not so. Excellent gains are possible while estrogen is low.

It is true that water retention does help strength, and very high doses of added estrogen have assisted powerlifters. However, this is at the cost of appearance. <u>If the pear-shaped look is what you want, then just take estrogen; an aromatizing steroid is not required.</u>

Lastly, and conclusively, trenbolone is as strong as anything available, and it does not aromatize significantly if at all. So aromatization is not the key to the effectiveness of a steroid. It is just a usually-undesired side effect.

Q: Actually, what is aromatization?

A: If the ring on the left end of the molecule has three double bonds, as estrogens do, it is called aromatic. If certain groups are removed from an androgen, for example, the #19 methyl and a #1 hydrogen from testosterone, it can become aromatic and become an estrogen. For some steroids this is possible. For others, it is not.

Q: They also say that the androgenic steroids are the ones that convert to DHT. True?

A: No. There is one and only one anabolic steroid that converts to DHT, and that is testosterone, or its precursors.

DHT is the same molecule as testosterone, except that it has a single bond between carbon atoms #4 and #5, where testosterone has a double bond. Primobolan, for example, can't convert to DHT because it has an extra methyl group that cannot be removed. And it has its double bond in a different position anyway. A similar situation applies for all other anabolic steroids.

Q: What? But many books say that some steroids convert to DHT,and some steroids are DHT derivatives. They even base their theories on this!

continued on page 6

STEROID BASICS Part 6 (continued)

A: They are wrong. Their conclusions might be wrong also. As to the derivative issue, it is true that some steroids, for example oxandrolone, are most cheaply synthesized from DHT. But after the chemical synthesis, they are no more similar to DHT than to testosterone.

Q: What is meant by "anabolic index" or "androgenic index?"

A: Years ago, this data was obtained from testing on rats. Skeletal muscles did not grow much in rats from anabolic steroids, but the prostate and a sex-specific muscle called the levator ani did grow enough to allow easy measurements.

The so-called "androgenic index" simply refers to how much the prostate grew in the rats. It ought to be called the prostatotrophic index instead. It has nothing to do with many other "androgenic" effects we might be concerned with such as virilization in women or anything else. And the so-called "anabolic index" refers to the growth of the levator ani, which actually is very dissimilar to human skeletal muscle and doesn't really help predict the value of a drug to bodybuilders.

Q: If all this is useless, then what do we do?

A: Just look at each individual effect you are interested in and evaluate each drug on that basis. Does trenbolone grow muscle effectively? Yes. Does it tend to increase aggression? Yes. Does testosterone (without drugs to block estrogen) give water retention? Yes. And so on. Forget trying to pigeonhole everything into an anabolic/androgenic spectrum.

Q: Why do people make good gains for some number of weeks, and then the gains stop? Doesn't that prove that something bad happens to the androgen receptors?

A: No, it doesn't. It simply is hard or impossible for the body to grow rapidly for any sustained length of time. The fastest the body grows naturally is during puberty. Even five lbs. per month, if sustained, would be 60 lbs. per year. Certainly that is not normal. Such a rate of gain is not sustainable. Sure, you can get a spurt of growth and add 10 lbs. of muscle in a month, or 25 lbs. in a cycle. But that just is not going to continue uninterrupted. The body will pause.

Q: Why?

A: I don't know the specific mechanism. But this does take place. The more that one has grown recently, the harder it is immediately thereafter to grow further. This is another reason to cycle steroids. After adding some significant amount of muscle mass, some time is needed before the body is ready to grow more.

Q: Take a steroid novice. Let's say he has pretty much reached his genetic limit with natural training. He will do well with a fairly small dose of anabolic steroids in his first cycle. But after he has added say 25 lbs., he finds that he needs bigger doses to make any further gains. Why?

A: I think it is a question of homeostasis the body's tendency to maintain some equilibrium. Without anabolic steroids, but with certain training and certain genetics, the homeostasis point might be say 200 lbs.

in cut condition. If the trainer is only 150 lbs., he is far from the homeostasis point, and will rapidly approach it. By the time he gets up to 190 lbs., he is close to it, and the approach will be slower. Of course, with different genetics, different numbers would be needed. With a low dose of anabolic steroids, the homeostasis point is shifted.

Now, with appropriate training, 225 lbs. for might be the new maximum muscular weight. Does this mean that now his body is not responding to anabolic steroids as well as before? No! With low dose steroids, his homeostasis point is 25 lbs. higher. But if he wants to get bigger yet, he will need a dose that is sufficient to shift this point yet higher.

This is simply a theory based on the typical behavior of biological systems and observed effects of anabolic steroid use. The homeostasis point I refer to is not something that can actually be measured. But it can be observed that a certain maximum is achieved beyond which one cannot go without changing the circumstances. That might be done by using higher doses of steroids or adding in other drugs such as insulin and GH.

Q: What are anti-estrogens? And what are estrogens, anyway?

A: Estrogen is a generic term which includes all-hormones that act like estradiol, the most potent natural estrogen. Other natural estrogens are estrone and estratriol. There are synthetic estrogens too.

There are two means by which the effects of estrogen can be reduced. The first is that a drug can inhibit aromatase, the enzyme which converts testosterone to estradiol. Cytadren and Arimidex are anti-aromatases. These result in lower actual levels of estrogen. Proviron and DHT are effective antiaromatases also. The second is that a drug can bind to the estrogen receptor, but without activating the receptor. Estrogen is then blocked from the receptor. Nolvadex and Clomid are hormone antagonists of this type.

Q: There's a lot of talk about cortisol, and that one should keep cortisol levels low. It has even been claimed that anabolic steroids really work by blocking the cortisol receptor. Is this true? How should cortisol considerations affect a cycle?

A: It isn't true that the blocking of cortisol receptors is the key to how anabolic steroids help build muscle. If this were true, then people with Addison's disease, who are cortisol deficient, would be huge. Not so. And cortisol blocking drugs would be great anabolics. They are not.

Nonetheless, it is true that abnormally high cortisol is a problem and does lead to muscle wasting. **Therefore, if Cytadren is being used, its use should not simply be terminated abruptly.** Rather it should be tapered down over several weeks to avoid a cortisol rebound effect. And it is a serious mistake to continue high training volumes upon cessation of a cycle or during the taper.

The goal at that point is to maintain gains, not to gain yet more muscle. And muscle can be maintained with surprisingly little exercise. Even two or three sets per body part per week can do it. In the first week of the taper, I recommend a substantial reduction in training volume. For example, 30-40 sets/week would be appropriate, with no "high intensity" techniques being used.

By the time one is completely off of

continued on page 8

STEROID BASICS Part 6

(continued)

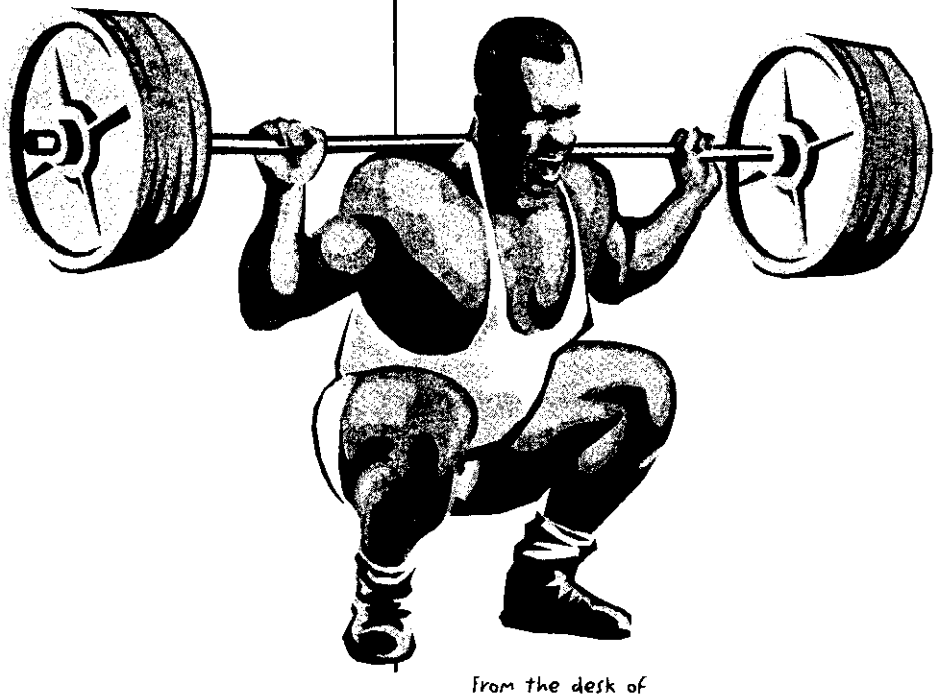
anabolics, the volume might be only 20-30 sets/week. Once it is felt that the body has renormalized, higher training volumes would again be employed.

The exact numbers are not important, and would vary according to the exercises done. It is the trend that is important. In my opinion, the reduction in catabolism after the cycle that can be obtained by using lower volume more than offsets the reduction in growth stimulus, especially since further growth is really not expected at this point anyway.

Q&A

Q. Will you please finally tell me how to take the estradiol from the Synovex pellets?

A Ether. But not with engine starting fluid, which has an added solvent that isn't easily evaporated. Good luck in finding a chemical company to sell it to you. It smells like "felony drug manufacturing", and knocks you out if you sniff it.



Q. In Pat's article in making injectable steroids, can I use this process for any other drug?

A Yes, Chemical Sellers of Santa Barbara sells powdered Yohimbine HCL, which is soluble in water. You could treat the Yohimbine HCL with lye to remove the hydrochloric acid, and the resultant precipitant would be pretty-much pure Yohimbine powder when dried. In this form, you could dissolve it into the 3 solvents (benzyl alcohol, polyethylene, and propylene glycol). Theoretically, you should be able to inject small amounts of this Yohimbine solution into stubborn fat areas, and the solvents would dissipate, depositing fine crystals of Yohimbine in the fat, allowing a very slow release into the immediate area. Keep in mind, too much might put you into cardiac arrest. So the usual "don't try this on yourself' applies. But I'll try it on myself, and let you know (if I make it).

Number 9 • July 1998

Dan Duchaine's
DIRTY DIETING NEWSLETTER
MILITANT MUSCLE GROWTH AND FAST FAT LOSS

This is Your Brain on Androgens

John Gulfstream

IN THIS ISSUE

Despite the wishes of bodybuilders, anabolic androgenic steroids do have direct influences on the brain that can cause an increase in aggression.

Among the many effects of androgens on the brain are increased aggression and heightened arousal used in situations of fighting and also for sex. On the other hand, the claims of some scientists indicating that androgens can make someone psychotic are far overstated. **What are the affects of androgens on the brain? Is there such a thing as 'roid rage?**

How can we use these affects to our advantage? Testosterone is thought to be responsible for both the sexual characteristics and behavioral patterns of males.

Interestingly, this affect may be brought about through estrogen, once aromatase converts testosterone to estrogen.

Testosterone has been shown to be important for sex-specific behavior in mice during development and in fully developed mice.

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from the desk of
Daniel Duchaine, PhD

Dan Duchaine's

Dirty Dieting Newsletter®

Militant Muscle Growth and Fast Fat Loss

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This is Your Brain on Androgens

(continued)

Male and female mice show little fighting behavior when raised together. However, if adult mice are isolated for 3 weeks, then put together, males show a great deal of fighting behavior. This fighting only occurs if the males have their testes when they're put back with the females. If the males are castrated before they are paired back with females, the fighting doesn't occur.

If castrated males are given testosterone, then the males will commence fighting. If the females are given testosterone at birth and again when they are adults, then they will fight like males. **Therefore, it appears exposure to testosterone soon after birth encourages fighting behavior at later times as long as testosterone is still present.**

Testosterone-induced aggressive behavior was also seen in a study using castrated male rats, the study found that rats without endogenous testosterone demonstrated normal, male aggressive behavior when supplemented with either testosterone propionate or methyltestosterone.

However, Winstrol was completely ineffective at eliciting aggressive behavior. The authors conclude that "the heterogeneity of anabolic-androgenic steroid effects on the nervous system and behavior indicate that the psychological effects reported by human anabolic-androgenic steroid users may depend upon the distinct chemical structures of the abused steroids."

This is a very important finding because it indicates what steroid users have been saying all along: **Different steroids have different effects on their mood and training. Read on to find out what those effects are and how to utilize them for maximum benefit.**

Enough about mice and rats, what about humans? A study using hypogonadal adolescent males found that testosterone injections significantly increased physical aggressive behaviors and aggressive impulses.

Based on this study alone, it may only mean that low testosterone levels causes a decrease in aggressive impulses, and that the converse (testosterone levels above the normal range cause an increase in aggression) may not necessarily be true.

Another study using humans found that exogenous steroids may have psychological and behavioral effects in some patients and athletes. But the effects were variable, transient upon discontinuation of supplementation, and appear to be related to 17-alpha-alkylated rather than 17-beta-esterified anabolic androgenic steroids.

Genetic factors, peer

influence, medical history, expectations and a host of other factors determine the nature and the extent of effects. This study showed a lot of variability between patients, but 17 alpha-alkylated androgens were found to have the potential to cause an increase in aggression.

To further investigate the potential link between mood and aggressive behavior, healthy men were given 600 mg/week testosterone enanthate to assess any changes. The study found no change in aggressiveness before and after treatment nor when compared to untreated controls. This is not what most people would have predicted for testosterone and other aromatizable androgens.

However, one limitation of this study was that it used such a small number of subjects. Larger samples may be necessary to elucidate neurological effects of steroids on human behavior.

To summarize the studies examining androgen influence of behavior:

- **Testosterone causes aggression in mice (even female mice).**
- **Testosterone propionate and methyltestosterone cause aggression in rats while Winstrol does not.**
- **In humans, 17 alpha-alkylated androgens are more likely to be associated with increases in aggression.**
- **Testosterone injections cause an increase in aggression and aggressive impulses in some studies, but not others.**

What is the take-home message on androgens and behavior? **Androgens are capable of increasing aggression, but it is highly dependent on which androgens are used and the conditions associated with their use.**

To understand how androgens manipulate behavior and the propensity for aggression, it is important to understand a little about the region of the brain involved in aggression and other emotions.

One of the ways that scientists study the brain is to damage a region of the brain (or examine humans, animals, or both with accidental brain damage) and then see how the human or animal behaves and functions without that brain region. The brain damage is often referred to as a lesion. Some medical doctors purposely cause brain lesions or damage a specific region of the brain attempting to alleviate a medical problem.

For example, in the 1950's doctors tried to treat epilepsy by removing portions of the brain. In the 1960's doctors tried to treat psychoses by removing or lesioning portions of the brain. Patients were

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This is Your Brain on Androgens

(continued)

often cured of their illness, but they lost the brain function that was carried out by the removed or damaged cells. These have been some of the most useful studies for learning how the brain functions. The absence of a function following a lesion gives strong evidence for the purpose of the missing brain region.

The amygdala is a brain region long believed to be involved in emotion, arousal (for fighting and for sex) and aggression. Very early experiments using cats found that when they put an electrode in a cat's brain and stimulated the amygdala, then the cats would hiss and act real pissed off. So, scientists had good reason to suspect that the amygdala was involved in aggression. When patients demonstrated disorders of aggression, doctors attempted to cure the problem by removing the amygdala, or damaging portions of it.

In one such study, 15 epileptic patients showing abnormally aggressive behavior were given small lesions of their amygdala. Half the patients showed reduced aggression following the surgery.

Another study found that lesioning the amygdala will result in a loss of emotional responsiveness in man and monkey. The threshold for emotional responses becomes higher following lesion of the amygdala. The emotional responses could still be achieved, but it took dramatic events to evoke the emotions.

In 1963, 60 aggressive, hyperactive patients received small lesions damaging 1/3 of the amygdala. 85% of the patients showed reduced emotional excitability and a normalization of social behavior. The patients became calmer, but not apathetic. They could become angry and excited when appropriate, but they lost the inappropriate behavior[7].

More extensive lesions of the amygdala were performed in other studies. A more recent study (1988) involved lesioning 2/3 of the amygdala on 481 patients (most of them were aggressive and destructive psychotic patients). They found that about 70% of patients showed a reduction in destructiveness and restlessness and that 50% remained calm and quiet even in the face of provocation.

This surgery was often referred to as "sedative surgery" because the patients were so calm they appeared to besedated. One patient (known by his initials HM) who had his entire amygdala removed rarely complains about anything, even if he is unwell. He appears to be content and placid all the time. Of course, this guy also lost an additional part of his brain that was used for memory - the hippocampus.

Despite this, it is still a good argument for the role of the amygdala in

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SYNEPHRINE

Lynn Christenson

As of March 1997, ephedrine products have been banned or restricted in 20 states. Further FDA legislation may limit the quantity of ephedrine in products and/or prohibit the sale of products containing both ephedrine and sources of caffeine. As a result, manufacturers are looking for alternative substitutes. Synephrine, the active ingredient in the herb Zhi Shi (citrus aurantium), may hold some promise.

Synephrine is an isoquinoline alkaloid found in citrus aurantium and other citrus fruits. The unripe fruits of citrus aurantium are called fructus aurantii, so sometimes these names are used interchangeably.

As a drug, synephrine is considered to be a catecholamine adrenergic agent. Like other adrenergic catecholamines such as norephinephrine, synephrine appears to be more active on alpha receptors (particularly alpha 1 receptors) than on beta receptors. Unfortunately little research is available on the herb, so determining its thermogenic potential is difficult.

With regard to the cardiovascular system, Zhi Shi has been shown to increase the contractile force of the heart in dogs[1] as well as increase the heart rate in guinea pigs. Whether or not this effect is due to synephrine or another active ingredient in the herb is unknown.

Zhi Shi (fructus aurantii) has also been shown to increase arterial and systemic blood pressure while reducing portal and arterial blood pressure in rats[2]. It is not known if changes in blood pressure can be completely attributed to synephrine. However, it is known that synephrine stimulates the alpha adrenoceptors in the sympathetic nervous system, leading to peripheral vasoconstriction. This action is similar to other thermogenic compounds like caffeine, kola nut, ma huang, and ephedrine.

Interestingly, although peripheral blood pressure is elevated resulting in decreased arterial blood flow, there is an increase in coronary, blood and renal blood flow[3]. Thus the herb may have both positive inotropic and chronotrophic effects.

As a result of its ability to raise blood pressure and its inotrophic and chrontrophic effects, Zhi Shi has been examined as a possible therapy for treating shock. Both Zhi Shi[4] and synephrine alone[5] were found effective at treating various types of shock in lab animals. Although studies have shown that synephrine increases production of cyclic AMP in mice[5], it is not clear whether or not the Zhi Shi herb increases human thermogenesis.

Further, if synephrine works more on alpha receptors rather than beta, then there would be an inhibition of cyclic AMP since alpha receptors (alpha 2 to be exact) inhibit cylic AMP production. Apparently results varied among different test subjects due to receptor specificity of muscles and organs.

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This is Your Brain on Androgens

(continued)

aggression because other patients losing their hippocampus show no changes in aggression. Additional damage to brain regions near the amygdala may bring about a more complete effect. In addition, numerous other reports have shown that lesioning of the amygdala can reduce levels of rage and aggression.

Not only is the amygdala involved in aggression but it is the region of the brain responsible for arousal and the desired heightened arousal which can be quite useful to powerlifters and football players. This heightened arousal is often felt just before a heavy lift and right at game-time for the competitive athlete.

Interestingly, the amygdala is loaded with aromatase, 5-alpha reductase and it does contain androgen and estrogen receptors. A study on aromatase activity in the mouse brain found that the highest levels of aromatase activity occur in the amygdala. Since testosterone levels have been linked to aggression and the amygdala (with high aromatase activity) has been linked with aggression, this raised the possibility that many of the effects of testosterone on aggression are carried out by estrogen.

A study using quail tested this very idea. The researchers found that testosterone-induced aggression can be prevented by an aromatase inhibitor indicating that estrogen levels actually mediate aggressive behavior.

In addition, they found that dihydrotestosterone (non-aromatizing androgen) does not induce aggression like testosterone. **These results indicate that estrogen is the primary mediator of testosterone-induced aggression in the brain.**

Along those same lines, a study using rats found that androstenediol is readily converted to testosterone, which is readily converted to estrogen, in rat brain. This finding is interesting in light of the mechanism by which many German athletes were taking androstenediol.

The German patent for androstenediol indicates that nasal administration of androstenediol causes a greater rise in serum testosterone than oral ingestion. The nasal ingestion avoids the first pass effect of liver detoxification which allows for much smaller doses of androstenediol to be used to achieve effective increases in serum testosterone. In addition, nasal entry of androstenediol allows for a much more rapid access of the steroid to the brain. **Therefore, it is likely that nasal ingestion of steroids could lead to a significant enhancement of aggression compared to other steroids and methods of delivery.**

What is the take-home message about aggression and the brain?

The amygdala is the region of the brain responsible for aggression and heightened arousal brought about largely due to estrogen binding to its receptor in the amygdala.

If this is so cut and dry, then why is there so much confusion over androgens and their influence on the brain?

Everything would be simple if androgens only activated androgen receptors and the effects of every androgen were consistent. However, some androgens activate other receptors in the brain and not every androgen is equal. Bodybuilders know that androgens are different, but scientists are just beginning to find out some of the reasons why.

A recent study found that Dianabol and androstenedione could bind to GABA receptors in the brain increasing chloride influx in a similar manner to alcohol and Valium.

Many of you may already be familiar with GABA (Gamma-amino-butyric acid). It is a neurotransmitter in the brain that is inhibitory. It tends to prevent neurons from firing their electrical impulses. Some time ago, GABA was pushed as a supplement to increase Growth Hormone release. While it failed at that task, it succeeded in putting bodybuilders to sleep.

The reason is that large scale activation of GABA receptors will inhibit a lot of brain activity causing a type of sedation. Alcohol and Valium exert their influence on this same recep-

tors. The sedative and euphoric effects of these drugs mediated through GABA receptors. **This finding, showing that androgens can activate GABA, receptors suggests that steroids could have a sedative or calming effect under the right circumstance.**

Few people report feeling calmer after ingesting Dianabol so this result is somewhat paradoxical. However, I suspect that any inhibitory effect of Dianabol binding to GABA receptors is outweighed by its potential excitatory effect on the amygdala.

Another study found that Winstrol and testosterone cypionate were capable of binding to a similar class of receptors in rat brain. **This means that the good feeling often reported when taking Winstrol or testosterone could be explained by the binding of these androgens to GABA receptors as if you were feeling the effects of alcohol or Valium, although to a much lesser degree.**

Interestingly, that same study found that methyltestosterone and deca-durabolin were incapable of binding to these receptors. This is a novel finding for the scientific community, but not for bodybuilders. Bodybuilders have been saying all along that different steroids effect them differently while the scientific community has stated that androgens are androgens and they all make you aggressive.

What is the take-home message for androgens and the receptors they activate?

Winstrol, Dianabol and testosterone are capable of activating receptors that can decrease arousal and aggressiveness - something you don't necessarily want to happen when moving heavy weight. However, there may be some situations where this is beneficial.

For example, testosterone or Dianabol activation of GABA receptors may provide a slight euphoric feeling that overrides some pain signals while relieving some inhibitions at the same time. In addition, we stated earlier that aromatizing androgens and 17 alpha-alkylated androgens tend to increase aggressiveness which means that inhibitory effects of GABA receptor activation may be very small compared to the arousal and excitatory effects of activating the amygdala.

So, what does it all mean when we put it together?

- **Any competitive athlete, whether powerlifter, football player, or what have you, can benefit from aggression and heightened arousal during competition.**

  In addition, bodybuilders can benefit from enhanced aggression and arousal in their workouts allowing them to work with more intensity on a daily basis.

- **Pharmacological enhancement of aggression and arousal should benefit any iron athlete.**

What pharmacological methods aid in the enhancement of arousal and aggression?

1) **Aromatizing androgens,**
2) **17 alpha-alkylated androgens**
3) **nasal ingestion of androstenedione or other androgens can all be used to enhance aggression and arousal. In addition, aggression and arousal can be enhanced by**
4) **avoiding Winstrol**
5) **and avoiding aromatase inhibitors.**

The bottom line is that androgen influence of brain and behavior is very complex and poorly understood. In addition, your genetic makeup and environmental influences play a huge role in how you respond to androgens psychologically and how you deal with arousal and aggression.

For example, if you're so timid that you can walk through a cafeteria line without receiving a single dish, then you might benefit from some testosterone or Anadrol. However, if you have already attempted to murder your lifting partner, you would probably benefit by sticking with Winstrol and Deca.

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SYNEPHRINE

(continued)

Further, most studies examined intestinal and uterine muscle rather than skeletal muscle.

Finally, synephrine has been shown to reduce immobility in mice and exert an antidepressant like activity[6]. However, results indicated that the reduction of immobility was not due to a stimulation of the overall motor activity or stimulant activity, such as seen with amphetamines. Further, these results found that alpha receptors are involved in the synephrine-induced decrease in immobility.

Increased activity in alpha receptors may have an added benefit to burning fat from stubborn areas since a high number of alpha receptors are located in the thighs, hips and buttocks of women and the chest and abdomen of men.

Just a word of caution, products containing synephrine are not recommended for use by anyone taking a MAO inhibitor. Nor should they be used by anyone with high blood pressure, heart or thyroid disease, diabetes, or prostate enlargement.

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UPDATE FROM THE UNDERGROUND (continued)

the US black-market, which is why after buying crap locally, they often consider taking a trip to Mexico to buy some cheap juice, and smuggle it home.

Many people get away with this time after time, but there are the unlucky few who get caught.

This can result in seizure of the vehicle used, the purchases, a body-cavity search, thousands in legal fees and a possible conviction for drug trafficking.

You would be wise to rebottle or expertly hide your purchass before crossing the border.

Safest option in my opinion is foreign mail order for those purchasing genuine personal use amounts. This really means singular, personal amounts. It's not for those of you who purchase enough for you and your entire touch football team to cover the cost of the gear.

The amount for an average cycle, say 20 amps, is unlikely to cause any legal hassles even if confiscated by customs and ordered in your real name (Unless your name is John Kill the President Doe).

The mail-order horror stories you might have heard happen to those involved in importation of large amounts, most of which is to be sold.

Obviously, they don't admit this when caught, which is why mail-order often gets a bad reputation. There are probably hundreds of packages of foreign juice being delivered to US homes every day. If the risks really were high, I'm sure those of us who write for the muscle rags would hear about it. **The main risk in overseas mail-order is losing your money.**

Here are the more common ways of "losing it". The money is stolen during transit. Assuming you are being required to send cash, you will want to ensure that when the envelope is held up to a light that the outline of the money is not visible. **It's common for people to wrap the money in sheets of paper or to put it inside a greeting card.** You may want to fold the notes so that they are not instantly apparent if the envelope is ripped. Always seal any vulnerable edge of the envelope with clear tape.

In earlier times, the money should be wrapped in carbon paper to prevent x-rays. Unfortunately, modern carbon paper no longer uses real carbon and won't work for this purpose. There is also a theory that the new metal strip $100 bill contains a tiny microchip which allows money inside packages to be instantly counted.

Whether this is true or not nobody knows for certain, but keeping the total dollar value low or using old $100 bills might be prudent. You don't want anyone taking the cash because they suspect you of money laundering (which is a cash purchase over $800). Some of UK postal workers can actually smell (through the envelope) US currency, so you might hide the odor with a drop of cologne.

The money might make it, but the steroid dealer you order from might simply take your bucks and send you nothing. Unless you send your money using a registered/recorded delivery service you will never know for sure whether the supplier received your payment. Unfortunately, many sources don't like the use of these services because it leaves an incriminating paper trail.

Your best bet is to find a supplier with a reputation for reliability which is often easier said than done.

The goods you order could be seized by US customs. What can you really say other than "tough shit"? Eventually it's gonna happen to everyone. Obviously, if you have access to several different mailing addresses this isn't a major deal. In years past, Customs always sent a seizure notice, listing the contents found, but this is no longer the case (to the delight of the unscrupulous steroid dealer).

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HAIR LOSS TREATMENT

(continued from page 3)

men. The only ones occurring in more than one percent of patients were decreased libido (1.8 percent of patients treated with Propecia versus 1.3 percent on placebo) and erectile dysfunction (1.3 percent Propecia versus 0.7 percent placebo). Decreased volume of ejaculate was reported by 0.8 percent of men treated with Propecia versus 0.4 percent of those taking placebo.

These side effects were reversible in men who discontinued therapy and even resolved in many of these patients who preferred to continue treatment.

A finasteride topical lotion would be a better choice but is not commercially available. Finasteride does not dissolve easily in alcohol or water, which are commonly used for medical lotions. Finasteride lotion must be specially prepared by a Pharmacologist or MD specializing in medical hair restoration.

THE POOP ON YOU

John Kabranza

Even with all of the potential legal risks, bodybuilders will do just about anything to get their hands on real anabolic steroids. Whether it be finding the best local black market dealer or finding the best foreign mail order house, they'll do it, no questions asked. Heck, they'll even pay higher for real stuff since it's so hard to come by these days. Put all of the "cons" and weigh them against the "pros", and anyone else outside of our subculture would think the risk isn't even worth taking. Bodybuilders quickly disagree.

And we all know too well who our "legal" enemy is: Drug Enforcement Administration (DEA). You can add on the potential threat from the US Postal Service and US Customs as well. For anyone who has been "privileged" (for lack of a better word) to see the inner workings of our government's attitude on the crackdown of anabolic steroid possession, then you know all too well that it has nothing to do with law anymore. Those SOB's are damn near reckless. And they don't give a shit. They get off on busting 250 lb. bodybuilders. It's an ego trip for them. Never mind all of the entrapment techniques, intimidation, and the fact that several agents specifically target and single out certain bodybuilders as a method of paycheck. That's a whole other topic.

Every bodybuilder who has broken the law by either buying steroids from a local at the gym or imported them from an off shore source has wondered... "What do they (DEA) know about me, are they onto me?". Or, "Is this new steroid dealer in Canada really a company disguised as a sting operation?". Been there, done that. For bodybuilders who deal steroids on the side, I'm sure they worry even worse. As you can imagine, sleepless nights fucking suck!

Well, now you have the opportunity to get rid of some of those worries and fears. Enclosed is a blank form request that allows YOU, the bodybuilder, the constitutional right to legally Neat, huh? That's right.

Whether your concerned about the DEA, FBI, Customs, ATF, etc., a nice little federal law - that far too many people do not know about - exists!

Basically, it's called the Freedom of Information and Privacy Acts, 5 USC 552, and what this means is that you can legally request the documents (which detail every little investigational thing they know about you) from a Federal Agency (such as the DEA)! And they have to release those documents to you, unless you are under current investigation. But that'll give you a pretty good clue if they send you a letter saying your request has been denied due to a pending investiga-

Okay, here's what you gotta do. Take the enclose request document and make copies of it.

You'll want to have extra's in case you ever need to do it again. Next, you'll want to get the address of the agency you want to get the files from. To get it, call the directory of your state (1-area code-555-1212) and get the address for the state headquarters. You may also want to send one to the branch office, which are usually located in each town or nearby city (note: some small towns may not have a branch office). After you get all of the proper addresses to send it to, fill out all of the information on the sheet. If you want to find out the files on whatever agency had on you at a previous address, be sure to fill in that address and mail them a copy as well. The last step is have the form notarized and signed by a notary, so go to your local town courthouse. Ask one of the people there and they'll point you to the right office. Be sure to bring couple forms of identification, as it is a major felony to pretend to be someone else. After you do all of that, make a copy of the notarized and signed form for your records. You should get copies of your files very shortly. Be careful and stay safe!

from the desk of
Daniel Duchaine, PhD

FINDING A SOURCE

There are plenty of sources if you know where to look. You can find adverts on pro-steroid web discussion boards such as Elitefitness.com and Aabolix.com, or newsgroups such a misc.fitness.weights, and from traditional media, i.e, magazines and newsletters.

There is a lot of secrecy surrounding good mail order sources. **The customers using them are not keen to share their good fortune with others, fearing their source might be shut down, or put under increased scrutiny by customs.**

At this moment in time I know of mail-order operations in the following countries: Canada, England, Finland, Greece, Honduras, India, Korea, Mexico, Panama, Portugal, Spain, Sweden, Thailand, Turkey and the Ukraine.You can rest assured that even in the countries where the sale and export of juice is legal, they are not paying taxes on all the payments received, and are keen to stay anonymous. The DEA monitors all the newsgroups and websites and reads all the muscle classifieds.

Payments in US cash are the most common form of payment and it's ideal for small purchases. The charges for Western Union or International Money Orders are prohibitively large percentage-wise for sending small amounts.

Personally, I find it odd that certain mail-order operations are so slow (e.g. three weeks plus) to fill an order. In present day, a package sent Airmail from Europe to the USA only takes four to seven days. So there is no real reason why an order should take in excess of 12 days from the day you send it. Unless, of course, the source is selling stock it doesn't really have (leading to huge backlogs) or is forwarding the orders to a third party.

A legit supplier will want to get your order out as fast as humanly possible as the chance you will reorder the minute you receive the goods is high. He also won't want to be caught with your money and order information.

In choosing a supplier, a source who speaks good English will lead to less confusion over the specifics of your order and will enable you to specify any unusual shipping requirements you might have.

If the selection is too good (numerous products or low prices) to be true, then it probably is. I'd be especially weary of sources offering rare products such as Parabolan or other French products. If the source stocks multidose human vials as opposed to veterinary multidoses then you can safely assume they are buying from clandestine labs and that his amp-based products have a far higher chance of being counterfeit.

You also want to check that your source has the products in stock when you send in your order. **Warning them in advance might be wise if you intend to purchase a large quantity of any one product.** Ask the supplier how long a typical order will take, and if it is more than two weeks, ask him why. If he can't come up with a reasonable reason find someone better. Prompt answering of communication is important. If the source is taking days to answer something as simple as an E-mail you can rest assured he'll be slow in other areas. If the source hasn't sent your stuff, you'll want to know rather than have them lie, wasting your time waiting for that delivery or sweating because you think the Feds have seized the stuff.

If the stuff does get seized, it would be good to know what, if any kind of compensation, the source will offer (e.g. a discount on a reorder). If a source genuinely stocks a product he should be able to supply you with detailed info on how it's packaged. If he can't, then he obviously doesn't really have it in stock. Are the products names spelled correctly on his price list? If he can't spell the product names, do you really trust him to decide if the stuff is real or not?

MAILING ADDRESSES

Over the years the muscle rags have written many negative things about using post office boxes to receive products. I have yet to see any clear trend showing that items addressed to these boxes are more likely to be checked by customs.

So you need another mailing address? **Once you've had a shipment of juice seized to your home address it is unwise to use it again.** Aside from using their work or college time address, most people won't have access to any other addresses. One possible answer might be to take your legit identification and open as many boxes as humanly possible. Then report that your wallet with your ID cards has been stolen. Really throw away your current ID, and apply for a new ID. Use the PO boxes one-by-one and if anything gets seized switch to the next box.

If the authorities ever realize that the same ID was used to open the boxes, you're covered because it's been reported stolen. Or you can simply have the stuff delivered to an

other person's home and remove it from their mailbox before they collect their mail, although this will involve a certain amount of surveillance and lawbreaking.

Many people use a modified version of their own name, e.g., Fred Flinstone, and use Frederick Flinstein instead, or they might simply swap surnames around (e.g., James Harold would become Harold James). This is felony fraud, though, in the states.

ENDORSEMENTS

Those of you with access to the Internet will know about Hulkster and his source endorsement scheme. Unfortunately, many endorsed services ended up offering a really poor level of service. Personally, I don't believe he could have prevented these problems.

The main problem was that the public placed too much emphasis on his endorsement rather than on what they could have found out through reading posted messages.

His endorsement of a source local to him caused the most problems. They even accused him of running the service and pocketing customers money. Here's a quote from Hulkster's retirement post

"Everyone states that they are getting ripped off by Sources!!!

I have developed a Source Endorsement Program. A new Source is asked to submit samples for testing. While that is getting done, a few friends will make some small orders to see if they get filled. After the samples come back good, and the test orders are filled, I ask the source to try his best to maintain a fast and reliable service and he gets ENDORSED!!!

Of course: someone can send in some samples, fill the first couple orders and then run, leaving me holding the bag and getting FLAMED!!!"

Musclebuilder also runs a top ten of mail order sources. Personally I would avoid sources which court publicity in this way. It's pretty obvious that those wishing to prevent mail-order would use these types of publications to find out valuable information.