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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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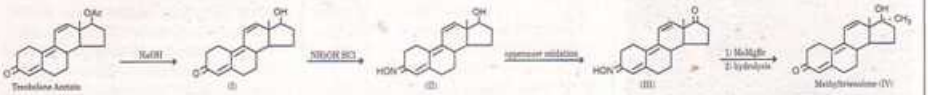
STEROID BASICS PART 2

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