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

continued on page 2

Dan Duchaine's
DIRTY DIETING™ NEWSLETTER
Militant Muscle Growth and Fast Fat Loss

Publisher: XIPE PRESS, Inc.
Editor-in-Chief: Dan Duchaine
Contributing Editor: Carol Jefferson
Art Direction and Production: Tilted 8 Design

Dan Duchaine's DIRTY DIETING NEWSLETTER is published 6 times per year by XIPE PRESS, Inc., 2533 N. Carson St., #2518, Carson City, NV 89706.

SUBSCRIPTIONS

To order your subscription by credit card, or to resolve subscription service problems, call Fitness Cargo at 888-48-CARGO. For payment by check or change of address, write to Subscription Department, XIPE PRESS, Inc., 2533 N. Carson St., #2518, Carson City, NV 89706.

DISCLAIMER PLEASE READ

The right to express ideas about matters which are provocative, controversial or offensive is protected under the free expression guarantees of the United States and California constitutions.

The content of **Dan Duchaine's DIRTY DIETING NEWSLETTER** is provided solely for informational purposes and is intended for your education and enrichment only.

The ideas expressed in this publication are not intended as, and should NOT be construed as, a substitute for professional advice, care or attention from a qualified nutritional or other health care practitioner. This publication is not designed to deliver professional advice or other health care service and the information provided herein should NOT be relied upon for personal diagnosis, treatment, or any other health related purpose.

ALWAYS consult your own nutritionist or other competent health care professional for the care appropriate for you or for answers to your questions or concerns about your condition, or a program of treatment.

Neither XIPE PRESS, Inc. nor the authors advocate, promote, or encourage the use of dangerous and/or illegal drugs. The information printed in this publication is NOT intended to induce or persuade anyone to use or possess dangerous and/or illegal drugs. Any reference made directly about the effects of drugs or any other substances are for informational purposes only and are an expression of the authors' opinions.

Neither Dan Duchaine, XIPE PRESS, Inc., FITNESS CARGO, the editors, the authors or their affiliates are responsible or liable, directly or indirectly, for ANY form of damages whatsoever resulting from the use, or misuse, of the information contained in or implied by the **DIRTY DIETING NEWSLETTER**, and any choice to use or follow the information presented herein is at the reader's own risk.

Dan Duchaine's DIRTY DIETING NEWSLETTER is intended for adults only since it contains sexually oriented material that may be inappropriate for minors.

Subscribers in the United States or Canada pay

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*

continued on page 4

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

continued on page 5

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)

continued on page 15

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).

continued on page 9

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.

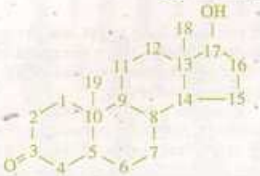
Quercetin has almost one-third the activity of Cytadren. What makes it better is that quercetin has less affinity (there has been no documented activity I could find) on any class of steroid binding globules, unlike Cytadren. Quercetin is quite safe to take all the time. However, taking quercetin in 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.

continued on page 15

from the desk of
Daniel Duchaine, PhD

STEROID BASICS PART 3

by Bill Roberts



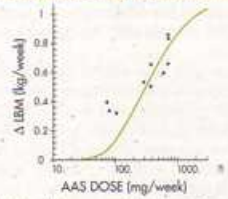
In Part I, we noted that in the normal male most androgen receptors (ARs) have androgen bound to them at any given time. This is because of the high binding affinity of testosterone and particularly DHT to ARs.

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

continued on page 13

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.

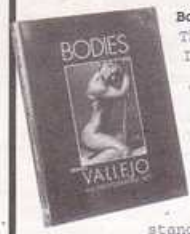
Can the highly anabolic state induced in the first few weeks of high-dose use continue forever? Can one gain 100 lbs of muscle per year? Of course not. Recall that there are negative mechanisms, as discussed, and furthermore we must consider effects on the natural hormonal axis.

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.

continued on page 15

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