



CHEMICAL MUSCLE  
ENHANCEMENT II

BY AUTHOR L. REA

[HTTP://WWW.ANABOLICBEAST.COM](http://www.anabolicbeast.com)



# BUILDING THE PERFECT BEAST

Featuring "Frank N. Steroid"

Building The  
*Perfect*  
**BEAST**

SECOND EDITION

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## **AUTHOR'S NOTE**

The information in this book reflects the author's experiences and is not intended to replace medical advice. It is not the intent of the author to diagnose or prescribe. The intent is only to offer information to help you cooperate with your doctor or a qualified physician in your mutual quest for desirable weight levels (muscle gains and/or fat loss) and overall health. Only your doctor can determine whether this regimen is appropriate for you. Before embarking on this or any other weight-loss/gain program, you should consult your physician. In addition to regular check-ups and supervision, any questions or symptoms should be addressed to your physician. In the event you use this information without your doctor's approval, you are prescribing for yourself, and the publisher, editor and author assume no responsibility.

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**"Building The Perfect Beast featuring 'Frank N. Steroid'" is part II in the Chemical Muscle Enhancement book series. The following websites/pages have more detailed information:**

**<http://www.anabolicbeast.com/cme/>  
<http://www.anabolicbeast.com/btpb/>**

Printed in the United States of America

SECOND EDITION

# EDITOR'S NOTE

I'd like to first congratulate you on the purchase of this book. This is the second edition run of *Building The Perfect Beast* and the first edition received so much positive feedback that Author L. Rea (ALR) decided it would best to rewrite most of the book and add in an additional 75+ pages of NEW information!

## Too Much Information?

The only "complaint" we have ever received is that this book has *"too much information"* and would be best if we split it up into multiple books. We thought about that and yes, it would be easier to digest all this information in smaller bites. But unfortunately, if we were to turn this book into 3-4 separate "mini" books, the cost would also increase 3-4 times (*most of the cost is in the setup and cover, not the actual total pages printed*) ... which means YOU would end up paying for it at the end in higher book prices.

I doubt that is what you would want to do since after all, the only thing we all care about is the actual information. So, we decided to "cram" everything into one big book!

Yes, the information is very dense and it's best to **read this book at least twice** to fully understand the concepts. I've read and edited this book MANY times and with ever single reread, I learn something new or am reminded of a concept that I had forgotten.

## The BLAH Factor ...

Now, before we begin with all the introduction and stuff, let's first discuss **WHY** you are really reading this ... how much this book is worth to you ... and most importantly, how VALUABLE this information is to you.

I've written hundreds of articles, dozens of books and information courses on a variety of subjects spanning over a decade now. I've taught classes, conducted seminars in front of hundreds of people and have even dabbled in the "infomercial" business as well.

And there is something I've learned about the "money" and "marketable" aspect of any product or service and that is - most people just don't know the different between **QUANTITY versus QUALITY of information.**

## Nothing Could Be Further From The Truth!

Most of us unconsciously (or even sometime consciously) do "judge a book by its cover". If the book has lots of pages and is long ... and is "pretty" and has a nice design and a good title and lots of pictures and so forth, then most of us automatically (*and many times incorrectly*) assume that the information must be valuable.

Nothing could be further from the truth. I know many people who can sit down and write and write and in a few weeks they've written VOLUMES of pages on a specific topic. And guess what, most of the information is just JUNK. Lots of "blah, blah, blah".

As stated already, this book is VERY dense. It can be a difficult read at times and you'll need to really focus. There aren't endless pages of made up theories or pictures of counterfeit steroid bottles (*which is useless because by the time the book comes out, the entire counterfeit world has changed and the*

*information is completely outdated and useless*) or tons of promotions for supplements or other bullshit sales tactics.

Yes, you will run into grammar mistakes, misspellings, and other printing errors. Yes, there will be information that will need to be updated due to new scientific research and studies ... Yes, there will eventually be new drugs out and protocols that will be "better" than the ones listed in this book. And yes, we will have ALL the new updates at our private site [www.AnabolicBeast.com/btpb/](http://www.AnabolicBeast.com/btpb/) ... but please **do NOT concentrate on a few naturally occurring imperfections.**

Never lose site of the actual information and the concept behind it. The foundation concepts of muscle building will not change much over time, just the approach. What we have listed is ONE of the best or *better* approaches at the time of writing.

In Conclusion ...

Anyway, now I'm starting to "blah, blah, blah". But I hope you get my point by now that length, quantity, pictures, typos, occasional grammar mistakes, "super cool" designs and so forth do NOT matter. **What matters is the *END RESULT*.**

And yes, I'm sure occasionally you'll read something we write and think to yourself, "*What a waste ... I already knew all that*". But it's NOT a waste because **mastery through repetition is the path to success and greatness!**

Sure, it's all "simple" once you figure it out ... or SOMEONE ELSE (like ALR) shows you the way and helps you. But until then, it's all complicated.

I am extremely confident that the information found in this book will bring you a step closer to success. I've seen it happen over and over again to hundreds of people who have read the information found on these pages before you.

Enjoy it ... Read it multiple times ... take notes and then "just do it". And please have some fun while doing it and above all, BE SAFE!

Warmest Regards,



Sam Phillips

## Pictures For Proof ...

Some (or I should say, many) people doubt what Author L. Rea says and does with his clients. Most of the "doubting" emails I receive are things such as "no way the body can grow that much"... or "change that much" ... or "ALR's methods aren't going to make that much of a difference compared to what other 'gurus' recommend for cycles".

### My Own Doubts ...

Of course, being in this business for 15+ years myself ... and dealing with pretty much ALL the major industry people and gurus (Dr. Scott Connely, Joe Weider, Dan Duchaine, etc.) I had my own doubts. In fact, I started this specific business as a learning experiment by hiring ALR as my own personal coach just to see if he was "for real".

After speaking to him for many hours and days, I quickly came to realize just how intelligent and knowledgeable he was about this whole "chemistry" of the body. Please note, that I got into medical school myself ... so I'm no beginner when it comes to this stuff.

Anyway, after a while, there were no more doubts in my mind that ALR "knew **his shit!**" better than anyone I had ever spoken to. Now, at the time of this writing, ALR is well over 275 lbs and around 7% body fat and this is only at a height of 5'7"! He's going to Las Vegas for the 2003 Mr. Olympia, so he thought he'd add on a couple more pounds of muscle to make a good impression on the bodybuilding world (let's face it, we'd ALL do the same thing if we could).

As amazing as his size, weight and muscle mass was/is to me, I couldn't help but think to myself "ah, this guy must just have some awesome genetics!"... right? Wouldn't you think the same?

### No More Doubts!

Well, check out the pictures on the next page. You'll see the amazing transformation of a "skinny" ALR at a bodyweight of only 127 lbs ... more than **DOUBLE his weight in less than 5 years** and weigh 260 lbs. After seeing this, I was sold and there were ZERO doubts as to what the human body can do with the right guidance.

Yes, these are real pictures. Yes, I've seen more of them for proof (hey, I was more amazed than anyone cause I'm always been a skinny bastard all my life!). And YES, he still maintains all of this muscle AND more for almost 20 years now!

He is basically someone who follows his own advice ... started off just like a regular "Joe" walking into any gym in the world and has kept it all while staying perfectly healthy. This is very inspirational to me and I'm sharing this with you hoping it will do the same.

Warmest Regards,



Sam Phillips

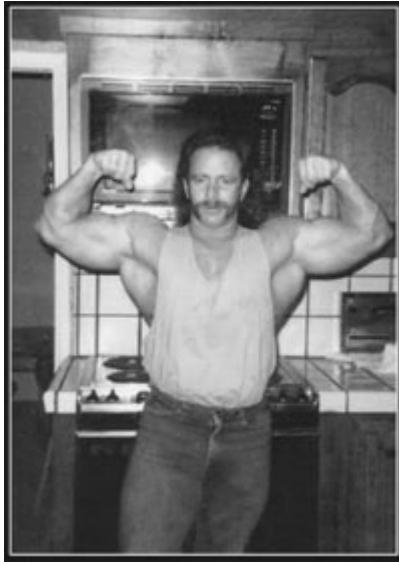
## Pictures Of Author L Rea - 20 Year Time Span

1982 - 127.5 lbs



This picture was taken in late 1982. I was cage fighting at the time for some extra income, as kick boxing paid badly and school was no cheap. I weighed in at 127.5 lbs as a rule for both cage fighting and kick boxing. Even at 5'7", that seems really small now ... even for a fighter. Nice arms, eh? I think my forearms measure bigger than my upper arms!

1987-258 lbs



This second picture was taken in mid 1987 ... about 4 ½ years later. I was prepping for a 3 show run bodybuilding contest and weighed about 258 lbs. I wish I had one with my shirt off, but you can see the lats flaring out. I can't believe the beard was actually on my face. Yikes! As you can see, a LOT has changed during the past 4 years or so.

2003 - 263 lbs



This is the most recent picture taken June 2003 and I weighed in at 263 lbs. A few things have changed ... no more beard (thank God!), no more post-fighting bruises ... but unfortunately, I do have a bit less hair. And no, I wasn't preparing for anything, my wife just decided to take a quick snap shot of me just for fun. My weight is normally around 260 lbs through out the year.

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## WEASEL STATEMENT

This book is not intended to diagnose, treat, cure, or prevent any medical condition or disease. Nor is it a guide or endorsement for drug use. The author and publisher do not condone any illegal drug use or abuse for any purpose. All readers are advised that substances described may be prohibited by law or only used with a physician's prescription.

**Warning:** Sexual explicit material may be offensive to some reader's as may be some of the author's commentary. Do not read this book if any subject matter could offend or upset you. I apologize to no one who reads beyond this point. I apologize to no one for my choice to exercise my freedom of expression. And after all...it is my crayon!

This book is written in an "**accumulative knowledge**" format. It was also written in a manner that assumes for the most part, that the reader has read "Chemical Muscle Enhancement" which is focused upon the drugs and classical cycles that have been utilized by athletes in the past. Re-explaining this information here would be a waste of the reader's time and perfectly happy trees. "Building The Perfect Beast (Frank N. Steroid)" is a continuance of the discussion of advanced chemical protocols begun in "Chemical Muscle Enhancement."

*To learn we must experience a subject from all angles possible.*

Like its for-runners, this book is not a children's "pop-up picture book" full of useless pictures of drugs. Instead, it is a discussion of advanced muscle chemistry techniques utilized by some of the top "big boys" in the bodybuilding world. Though intended for entertainment and discussion purposes only, I simply could not bring myself to fill it with filler pages of useless pictures.

Frank N. Steroid and Doc are purely **fictional** beings cloned from my imagination. In fact, they were created as a means of re-telling a tale from many individuals whom have chosen the chemically enhanced path in competitive sports. Any resemblance to a certain pro-bodybuilder or his "trainer" currently tearing up the competition world is merely coincidence. The following is a discussion of how some current competitive beasts were built. The reader must remember that this book is a story based discussion and not a guide or endorsement for this type of choice.

*"Genetics are nice, but limitations can be over come to an extent most individuals have never even imagined. Even an average athlete possesses the potential for an amazing physique far beyond their dreams."*

*Coach*

And the story begins...

## THE PURCHASE OF A WRECK

My life thus far has been the creation of chemical muscle enhancement protocols that produced the world's most perfect chemically enhanced beasts. It is simply a fact that all foods, supplements, and yes drugs, are nothing more or less than chemicals that have specific effects upon the body we refer to as Action/Reaction Factors. Some chemistry can destroy the body while aiding in the building of the facade. Sadly this means that some athlete's only *appear* as healthy as a superhero figure while inside a self-imposed life threatening time bomb may be ticking away.

Though the media and governments may either embrace or deem villainous any given chemical, my life has been a personal mandate for knowledge. By the way, most call me Coach. No, I am not a medical doctor nor should this story be taken or viewed as medical advice or endorsement in any way, shape, or form. I simply tell the tale for what it is.

I first observed him at a local iron house while training with an obnoxious client. The client was one of those guys who had more money than sense and an ego to match.

There he was another young man old before his time. A little over 6 feet tall, Genetically average, and making the same mistakes, training wise, as so many of his sponsored brothers had before him. Obviously he was the product of an uncaring owner...now cast aside. Sadly, bad muscle chemistry and dietary choices mandated by an unconcerned owner had left him a wreck before he had a chance to even get started. I'm sure you have seen him at one time or another. He is that guy who became a local hero that had something special about him. Picked up on a marketing contract by a major sponsor, 18 months later he re-appeared the wreck he is from misuse. His name was **Frank N. Steroid.**

My client became a bit aggressive due to my obvious interest in Frank. More so after I inquired about his name and owner. I only pissed my client off further when I told him Frank could have been the perfect beast. My rich obnoxious client voiced a challenge: If I could turn the "has been" into a beast that wins, he would foot the bill. If not, I was to become my client's "personal consultant" free for life and foot his bills. Like I said, the client has more money and muscle than sense.

I asked Frank one question: "what's your dream?" he replied simply "to dominate." Frank N. Steroid would be the scariest beast of our time, but first we must build the perfect beast...

After purchasing the lad it was time for a trip home to my lab. My lab is in that fictional land where muscle chemistry is legal and all athletes are required to take classes in Chemical Muscle Enhancement 101. Restaurants also feed bodybuilders for free in this

wonderful place. Though much of this would appear a dream, much of my time was spent in Mexico.

## **Tests and Blood Work**

First concern was Frank's health. Luxury type tests may include "quantitative urinary amino acid screening test" (QUAAST) to pin point specific amino acid deficiencies, but they are not mandatory. I have used both Smith Kline Bio-Science Labs, 7600 Tyrone Ave., Van Nuys ca 91405, or Doctor's Data 30 W. 101 Roosevelt Rd. West Chicago IL. 60185.

Below are 15 tests that I consider mandatory, and a few additional tests that may need to be added. These 15 tests needed to be run pre-cycle, mid-cycle, and post-recovery. In the future, problem areas obviously needed to be retested and evaluated at the appropriate point in each cycle. This was the only way to find out if the Program was working at peak efficiency, if doses are appropriate for that individual etc.

### **HORMONE**

1. Cortisol, Total
2. DHEA Sulfate
3. IGF-1
4. IGFBP-3
5. T3, Free
6. T4, Free
7. TSH
8. Testosterone, Total, Free and Weakly Bound
9. Hemoglobin A1C
10. Fasting Insulin

### **CARDIOVASCULAR**

11. CBC
12. Comprehensive Metabolic Panel
13. Lipid Panel

### **OTHER**

14. GGT Important Liver Value not included in Comp Metabolic Panel
15. PSA

(Thank you Dr. Weiser for your experienced aid in this area)

When we added to this test series to body symmetry measurements, body composition, chemical enhancement and medical history, we had a basis for intelligent protocol strategies.

\*It should be noted that in some cases other hormone levels should be evaluated such as prolactin, estrogen/17 $\beta$ -estradiol and estrone.

*"Success requires a plan. Without a plan, any level of success achieved is merely a lucky accident. Maximum results require maximum planning. All things are possible if Action/Reaction Factors are correctly anticipated and accounted for in our maximum plan. (Awesome Dude)"*

*Coach*

## STARTING FROM SCRATCH

### Day#1 Frank N. Steroid

**Body weight: 219 LBS., 11% fat, lean mass weight: 194.91 LBS**

**Measurements: Chest 49.2", Arms 18.2", Legs 27.1", Waist 34.5".**

Frank's blood work showed some liver enzyme concerns. This was not a surprise since Anadrol-50/oxymetholone (100mg 3x/d) was part of his last three Anabolic/Androgenic Steroid (AAS) cycles, as was the nightly practice of eating Tylenol.

As readers already have read in CME, Anadrol-50 is a c17-alpha alkylated oral steroid quite toxic to the liver under certain conditions. Most monitored athletes who had utilized this drug reported unfavorable liver enzyme test results after 6 weeks of continuous administration. For this reason the protocols designed for my beast never included utilization of this drug for periods of more than 4 weeks (or less) with a close eye on blood tests results. IN Frank's case, 24 weeks of prior use in one year was not only dangerous but counter productive for long term permanent gains.

### Liver Clean-up

The first step for Frank was to clean up his liver while resetting his entire body's physiology for optimum growth. Milk thistle (silymarin) is an over the counter (OTC) product clinically proven to aid liver rehab. It does so by stabilizing cell membranes and stimulating protein synthesis while increasing the process of regeneration in damaged liver tissue.

Milk Thistle actually aids in blocking the entrance of harmful toxins while aiding in the removal of toxins from liver tissue. It also has the potential to increase the rate of

RNA synthesis which accelerates the cell regeneration process. Other aids would be Evening Primrose Oil and Alpha Lipoic Acid (which will come later)

## Shrunken Objects and HPTA Function

Of course no hard-core chemistry was utilized for the first 8 weeks due to a period of clean-up. Of paramount concern was Frank's raisin size testes (from a health prospective).

For this a non-pharmaceutical protocol was possible. A product sold by **Hazardous Materials** ([www.hmgear.com](http://www.hmgear.com)) called **Testosterone OH (4-Hydroxytestosterone)** respectively worked well. The HM product is a "sterile oral" androgen that has high anabolic anti-estrogen effects that are surpassed only by the HPTA (Hypothalamus-Pituitary-Testes-Axis) regenerative effect. Many have used the product to aid HPTA regeneration while maintaining an anabolic environment between protocols with respectable success.

\* At the time of this writing, *Hazardous Materials* is producing the new line called **Hardcore Muscle Gear**, but it is the same company. (I guess Hazardous Materials was a bit too scary for some to deal with as a name for a supplement company)

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*Editor's Note: The above product not only aids in HPTA regeneration and hence, get the "balls working" again, but they are very anabolic! Yes, you actually put muscle on and reduce body fat with this product ... similar to taking regular steroids. Will you get the same results as "real" steroids? ... probably not. At least not in the recommended dosages (hint, hint)!*

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As some readers are aware, this kicks the HPTA function and therefore natural androgen production into over-drive like Clomid/HCG protocols. In most cases, if HPTA suppression is not too significant, this more than normalize natural testosterone and sperm production endogenously (made inside the body). In fact, as a result endogenous testosterone production elevates slightly above that realized by Clomid/HCG employment.

Most important is the reality that the product is legal in most countries since it is essentially an altered prosteroid. If Frank failed to respond adequately to this OTC protocol, a pharmaceutical HPTA regeneration method would have followed. (But this was not likely necessary)



Before experiencing **so-called** receptor-site burn-out, Frank was about 22 LBS heavier (off season) and had competed at 210-213 LBS 4% bodyfat during his short career. During this clean out phase, Frank trained 2 days on-1 day off and each body part was trained in the following sequence and protocol:

## **THE TRAINING PROTOCOL**

<b><u>DAY#1 CHEST</u></b>	<b>SETS</b>	<b>REPS</b>
30' Incline bench/bar	3	6-8
Pec Dec	3	8-10
Flat Bench /D.B.	3	6-8

<b><u>DAY #2 BACK/TRAPS</u></b>	<b>SETS</b>	<b>REPS</b>
70 Degree bent bar row	3	6-8
Weighted Pull-ups (Wide)	3	6-8
Dead Lift	3	6-8
Machine Shrug	3	8-10

### **DAY #3 Rest**

<b><u>DAY #4 Shoulders</u></b>	<b>SETS</b>	<b>REPS</b>
Behind Neck Press	3	6-8
Wide Upright Row	3	6-8
D.B. Side Lat D.B.	3	10-12
Bent Rear Delt	3	10-12

<b><u>DAY #5 ARMS</u></b> Close	<b>SETS</b>	<b>REPS</b>
Grip Bench	3	6-8
Push-down (Rope)	3	8-10
Bar Curls	3	6-8
EZ Bar Preacher-curl	3	8-10

### **DAY #6 Rest**

<b><u>DAY #7 LEGS</u></b>	<b>SETS</b>	<b>REPS</b>
Wide Squats	4	10-8-6-4
Sissy Squats	3	12
Leg Ext.	3	12-10-8
Straight Leg (D.L)	3	12-10-8
Seated Leg Curls	3	8-10

### **DAY #8 Repeat DAY #1 and continue protocol**

\* Listed "SETS" did not include warm-up sets and each listed set was taken to positive failure. This meant it was necessary to adjust weight loads to match the approximate "REPS" range as listed.

## Diet

Frank's diet provided 17 calories per pound of bodyweight daily (219 lbs x 17cal.=3723cal.), 2g of protein per pound of bodyweight (1752 calories/438g/47.0% of daily calories) and 1.5g of complex carbs per pound of bodyweight daily (1314 calories/328.5g/35.2% of daily calories). This left about 631 calories daily from fats or about 17% of Frank's 3723 daily calories. Fats came mostly from unsaturated and EFA sources. So Frank consumed 8 meals providing about 465 calories each, daily. Pretty simple, huh?

This lower carb/fat and high protein diet may seem severe to some readers. However, remember we had started from scratch and the fact that it was paramount to utilize the body's natural Action/Reaction capacities properly to obtain more permanent results. ***Not merely chemically maintained periods.***

This training and diet protocol allowed us to gather necessary genetic information including muscle group fiber types and ratios, individual muscle group protein turn-over-rate (PTOR), and work-load capacity ratios before inducing the first step... **Controlled Catabolism.** It was also necessary to allow time for liver enzymes to reach normal or better levels. Of course blood pressure monitoring was wise.

## MAPPING A MONSTER

### DAY #56

Frank's SMA-25 and lipid profiles were excellent and his endogenous testosterone levels were actually above normal. His thyroid T-3 production was averaging about 23 mcg daily, which was an important bit of information relating to dosages and long term results. An average T-3 production of 26 mcg daily is about average for most healthy individuals. And about 78 mcg daily of T-4 is normal.

## After The Clean Up

(He sucked) Body weight: 210 LBS, 10% body fat, lean mass weight: 189 LBS. Measurements: Chest 49", Arms 17.1", Legs 26.75", Waist 33.25"

Frank lost only 9 LBS of bodyweight total, 1% body fat, and 5.91 LBS of lean mass. (He actually gained fat weight) This was not significant as his prior misuse of AAS and necessary crash was to blame. However, what was significant was the fact that Frank lost just over 6% in arm mass 0.4% in chest/lat mass, 1.2% in leg mass while the only appreciable fat reduction was from the waist. Most would suggest this

was due to muscle fiber ratios (which of course was not the prime reason). Although muscle fiber types II a, b, & c are strength and major growth potential fibers, during a non-anabolic period it was the type I fibers that utilized calories much more conservatively.

No, the reason Frank lost 5 times greater arm mass as compared to leg mass, and 15 times greater arm mass as compared to chest/Lat mass was (PTOR) Protein Turn-Over Rate. As explained in CME, the body has a balance between anabolism (growth) and catabolism (tissue wasting) which is the body's state of homeostasis or **no change**. Anabolism is controlled by anabolic hormones and intermediates such as testosterone, 17-hydroxy-androst-1-ene-3-one, 4-androstene 3, 17, diol, 4-androstene-3,17,dione, T-3 & T4 Thyroid hormones, IGF-1&2, GH, Insulin, Interleukins, Prostaglandins etc. Catabolism is controlled by glucocorticoid steroids such as cortisol, and of course, glucagon.

## PTOR?

The body has a base protein turn-over rate (PTOR) of bodyweight multiplied by 1.818. So Frank's PTOR was 379.96 g of protein daily. So doing the math would look like this: 209 LBS x 1.818 g = 379.96 g.

However, due to different muscle groups having different PTOR's, each muscle will grow and shrink at different rates. This is due to available circulatory nutrient and hormone supply as well as the number and pattern of receptor-sites for each of the anabolic and catabolic biochemistries in each area. As example, a large number of muscle cell receptor-sites for cortisol and low number of receptor-sites for androgens, insulin, GH, IGF'S, and other anabolic goodies would result in a higher catabolic rate. Obviously this would worsen during low calorie periods and grow slowly during high calorie periods.

Another cause of different PTOR's imbalance is training induced. High trauma/low trauma training effects growth rates as well as levels of catabolism and recovery as does training volume and secondary trauma induced by compound exercises.

An example is the flat bench press. When done properly, this compound (multi-joint /muscle) exercise mostly effects or traumatizes the chest. However, the anterior (front) delts and triceps act as secondary muscle involvement during execution. Muscles do not grow during training. They grow during recovery. Training initially induces catabolism. Recovery, which can be influenced in many ways, is when anabolism occurs. For an increase in mass, a greater ratio of anabolism must result. This is why training protocols must be specific to each individual.

If for example, Frank trained chest with heavy or high traumatic sets and then trained triceps the following day, this would interrupt the anabolic phase of tricep recovery/growth due to secondary muscle involvement during compound chest exercises. If Frank's triceps were lagging this would result in very poor tricep growth. Unless, of course, his genetic PTOR were chemically altered throughout the body or site specifically.

## **FIBER TYPE RATIOS**

Muscle tissue contains two basic fiber types. Type I is typically referred to as endurance or aerobic fiber. Whereas type II is typically referred to as strength or anaerobic fiber. Average individuals have approximately an equal number or ratio of each fiber type though many have a predominance of one or the other. This is why some athletes seem genetically predisposed to be great long distance runners or weight lifters. (But never both) If this is not enough for a bodybuilder to deal with, realize that different muscle groups can vary greatly in fiber ratio in any one individual.

Type I muscle fibers do possess a growth potential, but nowhere near the growth potential of the type II muscle fibers. Type II fibers also have sub-groups called type II a, type II b, and type II c. Though the scientific community is still debating exact numbers, "generally speaking" type II a fibers actuate significantly during set weight-loads where positive failure is achieved in the 11-15 rep range, type II b in the 6-10 rep range, and type II c in the 1-5 rep range.

An athlete's muscle tissue contains all of the muscle fiber types in different ratios. This is why no two individuals should train in the "exact" same manner. It is also important to accept that there is a carry over or somewhat synergist action between fiber types and sub groups. For example, a drop-set where failure is achieved at rep 2, then rep 2 of the first drop, rep 2 of the second drop, rep 2 of the third drop, and rep 2 of a fourth drop should activate all 3 type II sub types. (Gee, ya think so?)

High / low drop sets (80% -85% single rep max to failure/ 50% single rep max to failure) where initial failure is achieved at rep 8 and the drop sets reach failure at rep 12, would result in an activation of both type I & II fibers. Cool huh? Ya, boring so far, but Frank needs 22" arms...at least.

## TESTING FIBER RATIO

*To do a simple test for fiber type ratios (without a muscle biopsy):*

\*Find the single rep max (SRM) for each body part.

\*Next reduce the weight to 85% of SRM and do a set to positive failure with reasonable form.

An example would be curls. Someone with a SRM of 100 LBS would reduce the load to 85 LBS. Say this person could curl 100 LBS for one rep but 85 LBS for 20 reps. It would be fair to say the athlete is predominantly type-I slow twist endurance fibers in the bicep area.

However if the individual were able to curl 85 LBS for only 9 reps, it would be obvious that there is an existing higher ratio of type II fast twist anaerobic fibers. This is basic, not exact. The lower the reps possible at 85 % the higher the type II fibers and the greater potential growth in most cases.

For the greater part, isolation exercises are best to evaluate fiber ratios since compound exercises utilize multiple muscle groups and therefore multiple fiber ratios. This is also a reason why many bodybuilders discover their triceps or anterior delts fail before their pecs on chest exercises that employ pressing. It explains lagging Pec development often for the same reason.

The following list is of isolation exercises best utilized. (Utilizing a 1.5-2.0 second positive, 1.5-2.0 second negative rep speed)

### Target Muscle

### Isolation Exercise

<b>Pecs</b>	(Mid) Pec-Dec (Upper) 30' Cable-Cross
<b>Anterior Delts</b>	Low pulley front Delt raise or Barbell raise
<b>Lateral Delts</b>	Seated DB Laterals or Cable-Cross Side Laterals
<b>Posterior Delts</b>	Reverse Pec Dec or 20' reversed incline rear Delt raise
<b>Biceps</b>	Hammer Strength Curl or 90' Preacher Curl
<b>Triceps</b>	Hammer Strength Tricep Machine or Push-down
<b>Lats</b>	Hammer Strength or Straight Arm Cable Pull-over
<b>Traps</b>	Full Range Shrugs (D.B. or Machine)
<b>Erectors</b>	Good Mornings or Weighted Hyper Extension
<b>Hams</b>	Bicep Femorous (lower) Leg Curl
<b>Quads</b>	(Lower) Leg Ext. / (Mid & Upper) Parallel Squats
<b>Calves</b>	Seated Calf Raise or Strict Donkey Calve Raise

## HORMONES AND FIBER TYPES

As we age the ratio of fiber types changes toward an increase in type I fibers. This is usually not a problem for life long weight trainers. The reason is simple: Use it or lose it. The good news is that fiber type ratios can be altered as can muscle cell number and fiber number/ size. Training stimuli and hormones greatly influence this as well due to specific intent training techniques. However, hormones, intermediates, and ATP have the greater potential for influence.

Why not combine specific training stimuli with specific chemistry protocols?

**\*Oh Ya, a few site quotes to validate what we have seen to be a fact before we continue.**

Testosterone increases the percentage of type II muscle fibers at the expense of type I fibers as well as inducing hypertrophy (Increased cell volume and/or increased muscle contractile protein synthesis...anabolism) (**Vaughan et al. 1974; Kelly et al. 1985; Holmang et al. 1990**).

Growth Hormone, testosterone and creatine can induce hyperplasia (the splitting of existing muscle fibers to form new ones or the joining of satellite cells to create additional fibers and/or cell count) and GH increases type I fiber counts (**Ayling et al. 1989**) or both type I and type II in the presence of testosterone elevation as well.

*\*Training protocols of a specific nature obviously effect specific fiber types as do specific chemical protocols.*

Frank had now been tested, poked, prodded and was ready to go... almost. (Even "the boy's" were checked for normal size ... by someone else)

## **CONTROLLED CATABOLISM FOR MAX ANABOLISM**

During any period in which calorie expenditure is greater than calorie absorption, a catabolic (tissue wasting) state exists. (DUH!) Bet you did not know that such a state also induces an anabolic environment? (HUH?)

That's right boys and girls! Catabolism creates the best environment for freak status growth. This is due to the body's survival response during which all kinds of enzymes, intermediates, hormones, and receptor-sites are up-regulated to store and utilize every possible nutrient absorbed as, or within, metabolically active tissue (protein based like, uh, muscle!).

Have you ever noticed what happens when a competing bodybuilder diets for weeks and months to get body fat levels down to 3-4% or so, then face-slams anything that doesn't eat him/her first about 2 seconds after final judging? What happens? For about 14 days body weight increases at an incredible speed with little fat gain.

The body reacts positively to most stimuli for 14-21 days before initiating counter measures.

**Remember:** The body seeks homeostasis? It is also paramount to realize that we grow (or not) as a result of what we "absorb", not due to what we eat. During this controlled catabolic period (Frank whined at this point) we create the ability to absorb, transport, and utilize amazing amounts of calories.

### **There are Always Choices**

There were two best choice protocols possible for setting the perfect environment for Frank. One was simply a matter of reducing calories (fats/carbs) to about 50% (or less) normal and over-train for 14 days before beginning Phase I. The other involved a little chemistry and 1 1 days. (Gee, which do you think Frank N. Steroid chose? Ya, so let's get on with it.)

### **CONTROLLED CATABOLISM (Training/Daily Calorie Decreases utilized)**

**DAY 1: CHEST-** 6 triple drop sets/shoulders 3 triple drop-sets (Any pressing exercise), super-set side laterals/ rear delt raises 3 sets/ cut 500 calories from diet.

**DAY 2: BACK-** 3 triple drop-sets any rowing exercise/3 triple drop-sets any pull-down exercise/2 down the rack sets of shrugs/ cut 250 more calories from diet.

**DAY 3: LEGS**-Squats 3 sets of 20 reps/leg press 3 triple drop-sets/2 down the stack sets of leg extensions/leg curls 3 triple drop sets/drop 250 calories diet

**DAY 4: ARMS**-Super set E.Z curls with skull crushers 4 sets/ Non stop super-set rope push-downs with preacher curls 4 sets/1 set triple drop wide bar push-downs/ 1 set down the rack DB curls. (Calories remain constant from this point through day #1 1)

**DAY 5:** 20-30 minute aerobic periods (Stair climber or bike) 90-120 minutes "Freaky monkey type love" sex. (With partner)

**DAY 6:** Repeated day #1 training , 5mg DNP per kg of body weight daily .

**DAY 7:** Repeated day #2 training, 5mg DNP per kg/d, 3 table spoons PG oil

**DAY 8:** Repeated day #3 training, 5mg DNP per kg/d, 3 table spoons PG oil

**DAY 9:** Repeated day #4 training, 5mg DNP per kg/d, 3 table spoons PG oil

**DAY 10:** Repeated day #5 training, 5mg DNP per kg/d, 3 table spoons PG oil

**REPEATED SEX OLYMPICS** (Again with partner)

**DAY 11:** Slept

\*During the 11 day controlled catabolic period it was paramount that Frank drank at least one gallon of water daily. It takes a great deal of water to remove catabolic waste. A good daily multi-vitamin/mineral was a must. 1 5-50 grams of glutamine daily reduced protein loss (divided into 3-5 even dosages). Peptide glutamine is said to be best (Though valid research is lacking), and 50-100 g/d was better. Alpha Lipoic Acid (ALA) is a great anti-oxidant wisely added to Frank's periods of DNP employment.

**PG OIL MIX:** 2 parts flaxseed oil/1 part extra virgin olive oil/1 part evening primrose oil. Divided dosages into 1 tablespoon 3 times daily. 25 ml of glycerine in 16 oz of water helped, too. PG oil was used as a supply of omega 3, omega 6, and GLA fatty acids. Perfect for prostaglandin production. (Go read prostaglandins "Chemical Muscle Enhancement")

Frank had done near non-stop AAS cycles for the past 56 months with few, if any, off periods. At this point he was working out but small for obvious reasons on both counts. However, he was ready to become a beast now. A dangerous chemical called DNP helped burn off fat as well as clear out androgen (and other) receptor-sites.



By now, the body had up-regulated anabolic receptor-sites, storage enzymes and hormones, as well as intermediates that focus upon lean protein based tissue anabolism.

## **THE OFF SEASON GOALS INTENDED FOR FRANK SHOULD SEEM OBVIOUS...**

1. Keep liver enzymes within acceptable ranges.
2. Avoid excessive fat build up (12%-14% max body fat)
3. Induce as much lean mass growth as possible
4. Control excessive water retention and heart/ kidney trauma.
5. Maintain estrogen and prolactin levels within growth ranges (estrogen increases IGF- production and glycogen storage) but low enough to avoid gyno, female pattern fat deposits, and excessive water retention (continue to the next section)

## ESTROGEN CONTROL

**Estrogen "Control"** was paramount for health and result potential reasons. High/prolonged circulating estrogen levels could have negatively affected the heart and other organs due to excess fat deposits and water retention. As you know, most severe water retention during AAS phases is due to estrogen and the resulting hormonal production increase of aldosterone.

Elevation of aldosterone affects the body's water table by altering electrolyte balances favoring sodium retention. Since sodium regulates extracellular (outside the cell) water and potassium regulates intracellular (inside the cell) water, an imbalance favoring sodium results in extra water in the vascular system and under the skin. This is the reason that some athletes look like "Bloat Boy" and have very high blood pressure.

It is important to remember that electrolytes such as sodium and potassium regulate the electric charges for flow of water molecules across cell membranes and are an intricate part of the sodium-pump mechanism that allows goodies like nutrients and creatine to actually enter the muscle cells.

### Blood Pressure and Water Retention

Normal blood pressure readings for average individuals is around 120 over 80. In fact, this is considered pretty healthy. However, bodybuilders and power lifters are not average. (Certainly none of my monsters) They tend to be much larger and heavier, carrying significantly more lean mass tissue. For this reason, they have larger more powerful hearts to supply high volumes of blood to a greater amount of tissue.

The first number of a blood pressure reading indicates blood pressure after the heart contracts, which raises vascular pressure, and blood is pumped through the body. Obviously the second number indicates or measures blood pressure before the heart contracts.

Since bodybuilders and power lifters have more powerful larger hearts, a first number of 150, 160, or even 180 is relatively normal. (And as a lone factor was not usually a reason to be concerned for Frank) However, if the second number was over 100, it was time to **be very concerned!** If the second number exceeded 100 during an AAS phase, it was usually due to excessive water retention and that meant that counter measures were immediately taken.

The most commonly used water retention counter measure was a low dose diuretic such as 20 mg Lasix daily. I have found natural diuretics such as dandelion root often resolved the problem. Regardless of diuretic form, liquids were always replaced before bedtime. Sometimes counter measures required backing off on

aromatizing androgenic dosages, but usually this could be resolved with estrogen control.

Most AAS Aromatize to estrogen to some degree. The higher androgenic steroids more so than the higher anabolics. In the first book "Chemical Muscle Enhancement" under drug descriptions, it is easy to find "aromatization". This gives an evaluation of each drug's reported aromatization characteristic.

## **We employed three types of estrogen control drugs with Frank:**

1. **ANTAGONISTS**: These are drugs that act as competitive inhibitors by simply competing with the estrogen molecule for its receptor-site.

The antagonist is like a little brother that beats you to the bathroom. If he gets in first, he only sits there doing nothing but keep you out so you can't do your thing. If estrogen can't get in, nothing happens.

Contrary to what is often claimed, antagonists are not "great" at controlling water retention, in my opinion. Since they simply block receptor-sites they do not decrease the levels of *circulatory* estrogen. This means potential increased levels of aldosterone. They do have "some" positive effects upon water retention, but they were best utilized as a drug for prevention of gynecomastia (Gyno/ bitch tits) and female pattern fat deposits. Nolvadex and Cyclofenil are examples of antagonist.

An advantage of utilizing the properties of specific estrogen antagonist (Faslodex, Clomid or Cyclofenil) for Frank was IGF-1 production. When estrogen is deactivated by the liver, IGF-1 secretion results. It is also interesting that under the right conditions, estrogen can increase GH levels by positively influencing the pituitary gland.

So in short, antagonists inhibit estrogen at receptor-sites and some aid in GH/IGF-1 secretion.

2. **AROMATASE INHIBITORS**: Aromatase inhibitors are sometimes called antagonist also (which is a pretty loose term). Aromatase inhibitors control estrogen by limiting or preventing the activity of the aromatase enzyme.

Normally, the aromatase enzyme induces the conversion of some types of androgens into estrogens. By utilizing an aromatase inhibitor this conversion is either limited or prevented. In most cases the difference between limiting and preventing conversion is dose dependant. Pretty simple, huh?

By inhibiting the aromatization of androgens to estrogen there is not a build-up of estrogen in the system. This was a plus during contest prep for Frank since elevated

estrogen levels inhibited fat loss and increased water retention. It also meant less of an estrogen induced negative feed-back loop inhibiting HPTA function for Frank.

Post cycle lean mass tissue loss was a significant concern.

Most AAS suppress HPTA function and therefore inhibit endogenous androgen production. Elevated estrogen levels further inhibit HPTA regeneration post-cycle. By utilizing estrogen inhibitors that decrease aromatase enzyme activity during AAS phases, there was a much lower build-up of estrogens post cycle to inhibit HPTA regeneration.

So the lesson here was that "inhibition" was finally a good thing. Arimidex, Teslac, 4-OH Testosterone, Formastane, Aromasin and Proviron are examples of aromatase inhibitors. In short, aromatase inhibitors prevent the introduction (or limit) of more estrogen into the system than is normally produced by biosynthesis. Some also possess low antagonist qualities as well.

3. **BIOSYNTHESIS INHIBITORS:** These inhibitors control estrogen at its very base, by preventing its endogenous biosynthesis. Normally the body begins the synthesis of most hormones with the conversion of cholesterol into pregnenolone. As usual, this is due to the activity of an enzyme. In this case it is the P-450 enzyme complex.

The hormone estrogen is several biochemical steps away from cholesterol...as you know already. If the P-450 enzyme complex is inhibited then the chain or sequence of biochemical steps are inhibited. Therefore so is the formation of estrogen and every other endogenous P-450 enzyme dependant hormone.

Cytadren is a 2-step estrogen inhibitor. First Cytadren inhibits the P-450 enzyme complex, and second it inhibits the aromatase enzyme. There are other biosynthesis inhibitors such as Trilostane and Metyrapone. Normally all 3 drugs are utilized in medicine as cortisol biosynthesis inhibitors.

That is entirely another issue. Since biosynthesis inhibitors control estrogen beginning at the very base of hormone biosynthesis, obviously they are effective at controlling water retention. Unfortunately, some negatively effect endogenous androgen production as well.

## LIVER HEALTH (GROW FASTER/LIVE LONGER)

Changes in liver enzyme values can occur during AAS use. This is especially true when oral c17-alkylated steroids or high dosage Tylenol are utilized. When a c17-alkylated steroid is introduced into the system, the liver must work harder to deactivate its molecule before excretion. Levels of Bilirubin, LDH (lactate dehydrogenase), and alkaline phosphatase are all good indicators of liver health and stress. I wrote a great deal more about this in "Chemical Muscle Enhancement". The upper normal level for LDH is about 250 U/L.

However, during Frank's AAS phases employing oral c17-alkylated drugs, this number **may** have risen to 400 U/L and above. "Usually" most doctors do not note this as a major concern unless other indicators suggest excessive liver stress. If alkaline phosphatase was above its upper limit of 150 IU/L as well, liver stress became a factor. Often levels were reported to elevate for the first 2-3 weeks of a cycle only to return to acceptable ranges a week later. For the most part brief variations in liver enzymes were not harmful as this was potentially an adaptive reaction. Another consideration was excessive alcohol use during AAS phases.

\*You should see most people's blood work after a hard night of drinking and partying. You would think Anadrol-50 was mild in comparison. This is not to say that the abuse of any drug is a good idea.

## BETTER HEALTH/BETTER BODY

I often find myself amazed at the great lengths some athletes will go to build the ultimate living physical edifice with little regard for actual health. When reviewing blood work-ups, it often becomes necessary to explain the most fundamental reality; Destroy the inside and the outside will follow quickly. Sounds kind of silly to say, but some individuals have invested as much as \$50,000.00 a year into various polypharmacological chemistries but not even \$10.00 into health.

The body is an adaptive organism with **Action/Reaction Factors** both good and bad. In the case of building the perfect beasts each factor had to be considered and responded "to" or "with" the appropriate response for long term ultimate progress to be achieved. In most cases, the beast's bodies reacted positively to chemical muscle enhancement protocols of a brief intense nature. **"Get in, grow harder, get out"** was the ideal intent. However, if any phase or protocol ran too long the body would have begun counter measures that could have eventually resulted in failure and the destruction of the organism.

## **AAS and The Heart**

Studies based upon valid research show prolonged high dose AAS use can have negative effects upon the heart. Eventually non-stop high dosage AAS cycles will lead to a decrease in high-density lipoprotein cholesterol (HDL). HDL protects against cardio vascular disease by shuttling cholesterol out of the blood and back to the liver. The liver degrades cholesterol into bile and only then can it be excreted from the body. This is the only method the body has to get rid of excess cholesterol. It is not like fat which can be burned or oxidized. A possible explanation for the significant increases in low-density lip-protein (LDL) cholesterol during long AAS protocols is simply a matter of biosynthesis.

Normally the body utilizes cholesterol to synthesize sex hormones endogenously (produced inside the body). Testosterone, estrogen and others all begin as cholesterol. When AAS are introduced exogenously, this process is inhibited. This in turn allows cholesterol to build-up to dangerous levels until discontinuance of the AAS. An interesting fact is that anything that stimulates HPTA (hypothalamus-pituitary-testes-axis) function/activity also decreases bad cholesterol levels. It does so by increasing hormone (like testosterone) production endogenously. Oral AAS have the greatest negative effects upon HDL. Nandrolones have little or no negative effect. In fact there are some studies that support the belief that nandrolones have a positive effect upon HDL levels, though I have found this not to be true in all athletes.

## **AAS and Red Blood Cells**

Another concern realized from AAS use is due to AAS induced increases in production of red blood cells. AAS stimulate the kidneys synthesis of erythropoietin (EPO) which in turn stimulates red blood cell production. Anadrol-50 was actually medically prescribed for this purpose. An increase in red blood cell counts increases oxygen transport, vascularity, muscle fullness/hardness and to a lesser extent body weight. From Frank's point of view this was cool, to a point! Having too many red blood cells for prolonged periods increases blood volume to a point of slowing circulation. This increases the chances of blood clots and therefore also increases the chance for strokes and heart attacks.

## **AAS and Aldosterone**

AAS induce activation of the renin-angiotension system in the kidneys. This in turn promotes the release of aldosterone from the adrenals which has a connection to some types of high blood pressure or hypertension. Aldosterone is a hormone that helps preserve blood volume via increased sodium retention and therefore water retention. High water retention increases blood pressure and heart health risk if the condition is over prolonged.

So obviously AAS **abuse** is a bad idea. However, brief AAS protocols were seldom reported to cause these negative effects simply due to their brief nature. Like most things in life, it is not so much a matter of what we did, as how we did it. ACTION/REACTION.

## **Diet Plays An Important Role In Heart Health**

Diet plays an important role in heart and total physical health. A diet higher in monounsaturated fats and Omega-3/Omega-6/GLA essential fatty acids aids in preventing heart rhythm disturbances and raise protective HDL levels. They also promote formation of good prostaglandins for growth. Any diet should not allow more than 10% saturated fats by macronutrient ratio.

For elevated cholesterol there is a prescription drug called Mevacor that contains Lovastatin which inhibits the liver from synthesizing cholesterol. It does so by inhibiting a specific liver enzyme.

**Red yeast rice** contains Lovastatin naturally and is available over the counter. 2.5-3 grams daily has been shown to have similar effects as the drug Mevacor. Lovastatin can cause muscle damage in higher dosages. So again more is not necessarily better. Coenzyme Q-10 appears to prevent this effect.

**Niacin** (Flush-free) also aids in reducing negative cholesterol problems. 1-3 grams daily has shown significant results in most individuals. Niacin should not be taken before training unless the goal is carb depletion. B-complex vitamins can inhibit the use of fat as an energy source and increase glycogen use during training. (B-vitamins should be ingested **after** training)

**Guggal sterones** also seem to aid in reducing negative cholesterol problems but I am still researching this one. So far it looks very good.

## **Thyroid Drugs**

Another issue we needed to look at was prolonged high dosage administration of thyroid drugs. Much more will be discussed later on the metabolic thyroid hormone factors, but for now I have a specific point.

Basically thyroid hormones reduce body fat stores by increasing the resting metabolic rate. If more thyroid hormone is introduced exogenously, the body burns more calories. To do so the dosage must be in excess of endogenous production. The resulting effect is called hyper metabolic. When exogenous thyroid hormones are introduced the result is a negative feed-back loop to the pituitary gland that shuts off the release of thyroid stimulating hormone (TSH). This in turn shuts down thyroid production/release of endogenous thyroid hormones. The higher and longer the exogenous dosage, the worse or more powerful the negative feed-back loop. Several

elite level athletes utilized thyroid hormone dosages daily that are 4-8 times over natural production in the belief that this would cause an equivalent increase in protein synthesis, protein-turn-over-rate (PTOR), and fat burning aspects. This was true to a point.

When metabolic rate is increased, there is a resulting increase in calorie expenditure. These calories come from amino acids, glucose/glycogen, and fatty acids. Not only fat stores (Fatty acids).

**Initially** the body will burn more lean mass than fat. Can you say muscle loss? When anabolic and anti-catabolic (Protein synthesis and protein sparing) chemistry was stacked with thyroid hormones the focus turned to burning fat stores. The main problem was that most athletes introduced more *thyroid* hormone than the counter chemistry could react to. This resulted in decreased muscle strength of up to 40%, soft tissue damage, and suppressed endogenous thyroid hormone production post-cycle.

*"More is not always better unless we are discussing breasts".*

*- Coach*

Post-cycle thyroid function was restored fairly quickly with 250 mg of guggal sterone (or 25 mg pure extract) per 50 LBS of bodyweight daily in 4 divided dosages. 3-5 grams of the amino acid tyrosine daily and 1-2 grams of phosphates helps also. This also worked well during the first week of GH use. More on that later.

So my point is that some athletes decided their muscle building chemistry dosages based upon thyroid hormone dosages. It should have been the other way around.

If the goal was to add mass or/and lose fat stores it was paramount to long term potential (and health) that no dosage threshold be exceeded before a dosage failed to provide acceptable results.

When excessive dosages of thyroid hormones were erroneously utilized this necessitated the need for excessive counter chemistry. As a direct result GH and other protein synthesis/protein sparing drug dosages would be quickly ramped up beyond what was necessary. The result was a significant reduction in long term potential for those whom chose this path.

The other issue should be the negative feed-back loop realized from exogenous thyroid hormone use (or abuse). If normal TSH production was not restored post-cycle, reported bodybuilding progress came to a stand still.

You will understand this better as we continue. I have never met anyone who has abused thyroid drugs to a point of "permanent" TSH suppression, but have noted



many individuals who had over administered to a point of catabolism exceeding anabolism. A few of these individuals thought so-called androgen-receptor-site burn-out was the cause of their lack of progress.

They then proceeded to increase AAS dosages to the point where negative side effects far outweighed the positive. It took some time and work, but they too began to progress again.

*"More is not always better. Enough is just right. Think of it like this: If more were always better, why do you hate alimony payments, Frank?"*

- Coach

## PSA

I will remind you of this several times, but PSA level test were important. The normal reference range is 0.0-4.0 ng/ml. The lower the better. By the way, different countries and labs will use slightly different reference ranges on any testing protocol. A PSA level test indicates the risks of prostate cancer among other bad things. If the level is high, DHT (Dehydrotestosterone) and estrogen together can trigger growth. Recent studies have often cited the 5-LO enzyme as the mediator for BPH and prostate cancer.

An interesting study relating to testosterone and its effects divided 61 men ranging from 18-35 years of age into different dosage test groups utilizing testosterone enanthate. The group divisions were: 25, 50, 125, 300, or 600 mg. One of the tests monitored was PSA. Interestingly enough after 20 weeks there was no significant difference in PSA levels between the lower and highest testosterone dosage. **Bhasin, s., et al. (2001) Testosterone dose response relationships in healthy young men. Am J Physiol. 281 (6):E11 72-81**

"There will be lots of sites and research quotes at the end of this text. Sometimes they are just too interesting to wait.

## EFFECTIVE DOSAGE FACTORS

If any chemical muscle enhancement substance introduced was to have an effect, it had to first establish a plasma level above normal for that individual. As example, normal endogenous testosterone production for a male is "about" 50 mg weekly. This is a 50 mg weekly plasma circulating level. Not 50 mg per day, but 50 mg per week, which amounts to about 7 mg per day.

For simplification, let's say that the 50 mg weekly production represented the normal plasma threshold for Frank, and that plasma level refers to the circulating plasma level per week. In order for an injection of (or oral dosage) exogenous testosterone to have an effect, it had to be at a dosage which entered the circulatory system at a rate and dosage that exceeded the normal plasma level and therefore the normal plasma threshold. This threshold had to be maintained or elevated to remain effective. This means that if the example AAS utilized suppressed HPTA function (natural endogenous testosterone production is suppressed) then this too had to be compensated for with exogenous dosages (longer low dosage cycles only).

## Active-Life and Half-Life?

Each AAS and chemical enhancement compound has a ***theoretical*** active-life of its own. An active-life is the period of time that a chemical remains active. Also each has their own half-life. A half-life is the period necessary for half of the dosage to migrate from the injection site into the vascular system and clear. So a 200 mg injection of testosterone enanthate would theoretically release 100 mg of testosterone by its half-life of 4 days, but remain ***effectively*** active for its active-life for 8 days. Naturally there is a continued clearing of each half-life after this time frame, but not to any real significance in our discussion.

So if testosterone enanthate was injected on day #1 and then released half of its 200 mg dose (100 mg) by day #4, then this would have exceeded the normal plasma level/threshold and establish a new one. Thus caused an effect superior to normal plasma levels. This is what is meant by the term supraphysiological by the way.

When I wrote "CME", a few critics complained about the fact that I listed drug active-life ranges (in some examples) rather than a firm single period for each. After years of monitoring blood tests it became obvious a drug could have different active and half -lives in different people, under different conditions. I created a series of charts which included multiple dosage thresholds during those years and utilized the information to establish **actual** ranges. Obviously a "firm" number was not possible.

## **Dosage Thresholds**

There also were lowest and highest thresholds for most drugs with multiple thresholds in between. The lowest threshold was the dosage required to cause an effect, where as the highest was the maximum dosage: The point where any higher dosages failed to cause better or more results (the latter is not listed in Chemical Muscle Enhancement). There were notable multiple thresholds in between as well. Simply stated, some drugs caused positive results at 100 mg but did not improve results until 400 mg dosages were utilized.

To express another example of thresholds, 10 mg of oral Dianabol daily was about equal in "activity" to normal male endogenous androgen activity. Yet it took 20 mg, usually, to exceed this threshold. The highest threshold, usually, was equal to 5 mg per 25 LBS of body weight daily for Dianabol (methandrostenolone).

Chemical and training history also greatly influenced dosage factors. A novice who had a few years of training experience only required 1 mg per LB of bodyweight of nandrolone decanoate (Deca) weekly to make excellent progress on a 28 day cycle.

My experience has been that athletes were foolish to exceed any threshold level of a drug until it failed to deliver acceptable results. This was one of the greatest long-term mistakes I saw and it significantly reduced long-term growth potential. If all of this was not enough to deal with when structuring a cycle for Frank, consider this: Different AAS have different affinities for receptor-sites and each has different activity.

Some are more potent androgenics or anabolics than others and would therefore require lower dosages for equal response or goal. Some have longer or shorter circulatory half-lives. Some even have different anabolic/anti-catabolic ratios to consider. Therefore each drug is individualistic in action and reaction and these factors had to be accounted for in order to make the best possible progress.

## **Please Read...and Think**

I write to encourage thought. Controversy is great if it forces individuals to be individuals who think! Well, are you frustrated yet? For some readers, much of that information is simplistic, but in truth most individuals just play gun-slingers shooting combinations in any order and for any period of time and hope for the best. Then they wonder why they lost most of their gains post-cycle or failed to respond significantly. In our perfect imaginary bodybuilding country, I started by making all of this information clear to Frank.

In the following sections, several of Frank's and other beast's protocols are explained. Each of these took into consideration effective dosage factors and some are out lined in multiple levels, **as they were utilized by Frank N. Steroid and other beasts**. More is not always better and synergy is a key to success.

Remember, once a dosage established a threshold, concurrent dosages of other AAS elevated the plasma level, and properly timed consecutive dosages of other AAS would have maintained it. Does that make sense? If not, it will soon. A fact I must make clear about half- and active-life periods is that some drugs with longer active-lives, required a longer period to reach their effective potential. An example is Deca. Deca has an active-life of about 14-16 days. However even after migrating from the injection site a day passed before the drug became initially effective.

## PROTOCOLS

The goal of any growth inducing protocol was to gain as much lean mass as possible. To do this with a plan meant to gain, not merely maintain or regain losses from prior cycles.

### The Body Has Action/Reaction Periods and Factors

The body has Action/Reaction periods and factors. Basically the body begins to adapt significantly to most attempts at altering homeostasis after 2-3 weeks. This sucked since the best results from a cycle usually came during days #10-30. But we also profited from this information and used it to Frank's advantage by utilizing brief phases and cycles of 21-30 days, (this is the point where cycles provided serious results) then got out before side effects out weighed benefits.

*"To be successful it is necessary to create maximum growth thresholds and stop before the body is able to achieve its own counter measure thresholds."*

- Coach

### Action/Reaction

Let me explain. My experience has been that a cycle of 8 weeks providing a continuous plasma level weekly of 400-800 mg testosterone begins to fail to provide results about week #6 in most cases. This is because the level of endogenous catabolic hormones becomes elevated as a **reaction** to the exogenous testosterone administration and cellular signaling proteins begin to become overwhelmed, which is due to the **action** of increase androgens. So anabolism and catabolism are again about equal. Remember to create a growth environment one or both sides of the anabolic/catabolic ratio must be altered in favor of either more tissue building or less tissue wasting.

At the 6 weeks point an athlete's body produces and maintains somewhat higher cortisol levels than normal. An increase in the administered dosage would again overwhelm the cortisol and increase signaling protein activity, but eventually it will catch up...again. Worse yet, when the athlete goes off cycle, the elevated levels of catabolic hormones overwhelm the declining anabolic chemistry (and activity) and most of the cycle gains are lost.

*"Yes, Frank. I realize that you lost a great deal of lean tissue mass after AAS discontinuance in the past. But what if an athlete, yes you Frank, utilized a brief protocol that allowed maximum growth and got out before endogenous catabolic and anabolic chemistry altered significantly? Yes, you are right. The athlete would retain much more of the protocol gains post cycle, and "Look honey, I don't have raisins" could be declared to your significant other."*

*"Now, what if we created following or layered protocols that either reduced the catabolic side of the ratio or used other metabolic possibilities to again increase anabolism? Yes, again you are right. The beast would be in a near continuous growth phase instead of fighting to regain."*

*"Now, what if we inhibited the negative feed-back loops responsible for post-cycle loss of lean mass tissue? I will explain this too, my lad. All in good time. The days of the genetically gifted only are over. The hard-core average genetic athlete is and will have their days of domination. Mediocrity sucks, Frank."*

*- Coach*

## **DRUG'S "MOST EFFECTIVE PERIODS"**

Now that we have discussed theoretical half- and active- lives of drugs and plasma levels/plasma thresholds, let's look at **"most effective periods"**. I realize this may seem like a great deal of information to deal with. But to construct any protocol that would provide maximum results all action/reaction factors had to be considered. Frank was not a steroid gun-slinger, but he did become a beast.

We know the half- and active- lives of injectable AAS are determined by the chain length of their ester. We have discussed the method by which the enzyme esterase acts upon an injection site dosage to induce migration of the freed AAS into the vascular system. We have also seen how different stacks and athletes have realized alterations in active- and half- lives. Now we need to understand drug **"Most Effective Periods"** or ranges.

Any drug introduced into the body by any method will have a range or period of time that it is "most effective." This is the part of a drug's active- life that provides the greatest results. It would appear a drug would have the same effect during its complete active-life. Sadly, it is not that simple.

After an injection of a drug is given, it takes a period of time for the enzyme esterase to act upon the drugs ester and subsequent release into the vascular system. The longer the AAS (or any drug) ester chain, the longer its active-life. This also means the longer it will take before migration.

Once the drug has migrated into the vascular system it may, depending on its structure, take a period or time to become effective. Believe it or not, this is not as difficult to predict as it would seem. I will give a few charts and graphs to help in a minute, but generally speaking a drugs most effective period is about equal to its half-life.

No, not the first half of its active-life, but usually the center portion of its active-life. As example, if a drug has an active-life of 16 days, it will have a "most effective period" of about 8 days. (Huh?)

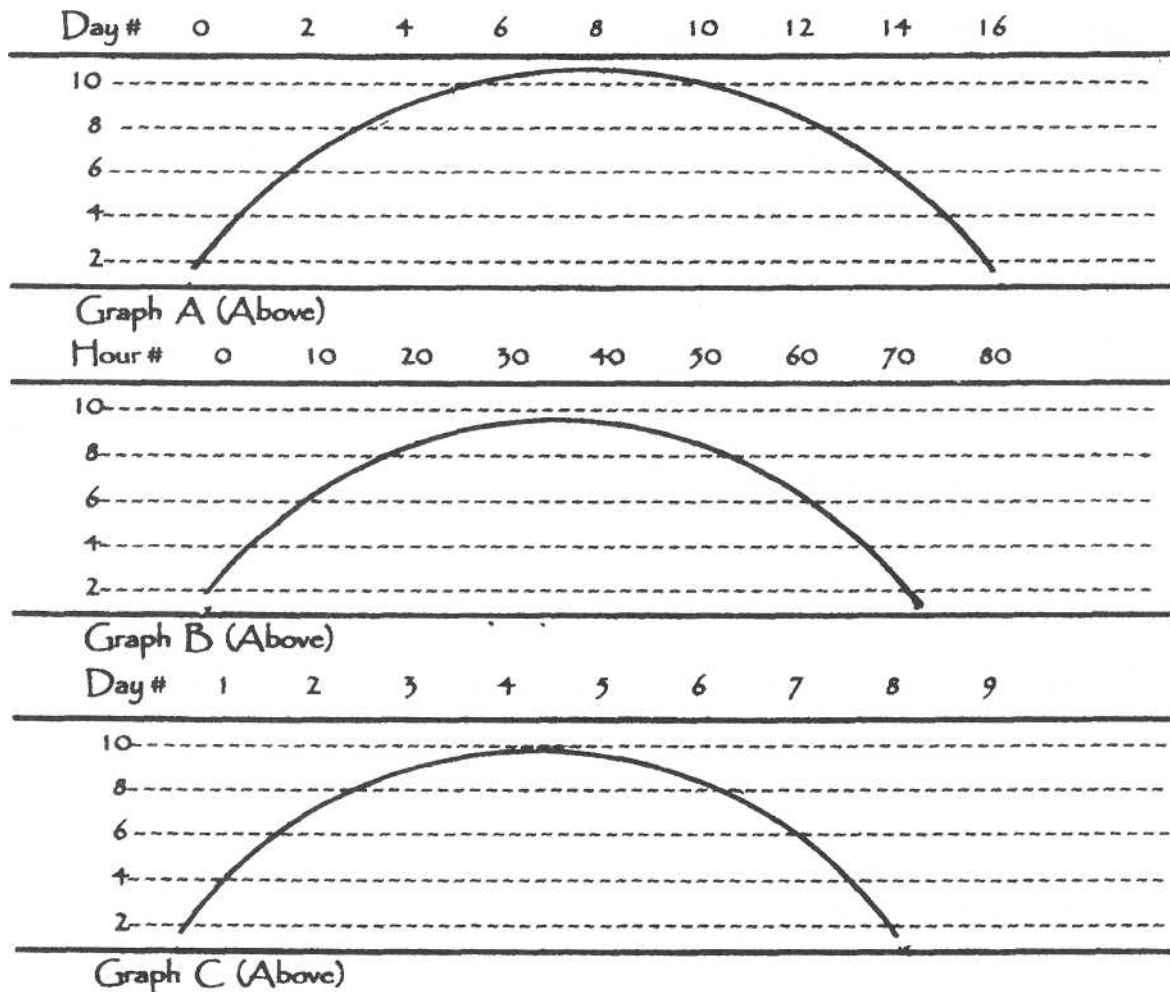
## MAX ANDROGEN PHASES

Now that we have a few fundamentals let's begin putting to rest all of the "*what and huh?*" moments I have created. But first, let's look at the below graphs ...

**Look at graph A.** (next page) This is a drug "most effective period" for an ester with an active- life of 14-16 days. The drug begins activity on day #1 but does not begin its true effect potential until about day #4. This drug would remain in its "most effective period" from about day #4 until or through about day #12, which is 8 days. Deca Durabolin, Testosterone Cypionate and Primobolan Depot are examples of the graph. (The drug remains "active" until day #16)

**Now look at graph B.** (next page) This is a drug with an active- life of about 3 days or 72 hours. The most effective period would be from about hour 18 to hour 54. Remember; the drug is still active before and after the most effective period. That is why it is called an active-life. Durabolin and Testosterone Propionate are examples of this graph. Bet you already knew **graph C** would be for an ester such as testosterone enanthate. Pretty simple, huh?

Max Androgen Phases Graph A-B-C ("Most effective periods")



## PTOR and Homeostasis

The body normally maintains weight and size of all cellular structures through homeostasis (assuming that our discussion focus is our protein synthesis rate/PTOR). The body maintains homeostasis or balance by both building and destroying an equal amount of protein based tissue daily. In fact, most research states that the body both gains and loses protein bases tissue at a rate of bodyweight multiplied by 1.818 expressed in grams daily. So a 200 LB bodybuilder gains and loses 363.3 grams of protein bases tissue daily;  $200 \text{ LBS} \times 1.818 = 363.3 \text{ grams}$ . So a 200 LB bodybuilder has a PTOR of 363.3 grams.

Homeostasis is controlled by hormones and hormone-like substances. Some are anabolic and create a state of protein synthesis and growth, while others are catabolic and create a state of protein destruction or waste. When chemical/hormone levels are



balanced, or equal in Action/Reaction, there is a state of balance and no change we call homeostasis. If you do not get this yet, do not worry. It is explained and referred to multiple times throughout this book. This is due to the fact that without understanding of this fact, maximum progress is dwarfed. Understanding all details of Frank's story is paramount.

To increase protein based tissue mass (Like uh, muscle) we must alter the ratio of "protein synthesis/protein wasting in favour of net total protein mass increase. This means triggering either anabolism (protein synthesis) in excess of catabolism (protein wasting) or decreasing protein wasting. **Any** substance that decreases the catabolic side of this ratio is considered anti-catabolic or protein sparing.

If we could increase the anabolic side of this ratio 100% without altering the catabolic side, our 200 LB bodybuilder would realize a daily net increase in protein based tissue of 363.3 grams. If we decreased the catabolic side of the ratio 100% the result would be the same. Many chemical muscle enhancement substances possess both anabolic and anti-catabolic qualities in various ratios.

The goal of Frank's Max Androgen Phases was to stimulate protein synthesis on multiple levels through multiple metabolic pathways. By stimulating the muscle cell androgen receptor-sites, we triggered cellular protein synthesis signals. By inhibiting cortisol receptor-sites, we decreased catabolism.

Also, by inducing a very high androgenic environment we allowed the musculature to significantly increase weight (strength) and work-load capacity. This was quite synergistic: We were able to train muscle more intensely. By increasing protein synthesis and decreasing protein wasting we were able to quickly repair the damage induced. With adequate macronutrient intake, we allowed for super over compensation or adaptation. The result was more muscle mass to carry greater work-loads. This was an adaptive process due to **Action/Reaction Factors**.

Unfortunately, the body realized we had altered homeostasis and the PTOR all to quickly. The body began to react to our anabolic/androgenic steroid (AAS) induced alteration after two to three weeks and **began** its own catabolic counter measures as a means of re-establishing homeostasis. To do this the body stepped-up production of cortisol a bit and estrogen as well. Since estrogen triggers a negative feed-back loop that induces HPTA (Hypothalamus/Pituitary/Testes/Axis) inhibition, the result was little or no endogenous testosterone synthesis.

Cortisol is a catabolic hormone that triggers cortisol receptor-sites. This results in protein wasting. If an AAS protocol ran too long, circulatory cortisol levels became elevated to a point where they equaled circulatory androgen activity even from exogenous sources. The result was homeostasis again. When the AAS protocol was discontinued and circulatory androgen levels decreased, the elevated cortisol levels overwhelmed the anabolic/catabolic ratio in favor of protein wasting. The result was

the loss of most, if not all, lean mass tissue gains induced by the AAS protocol. **Which sucked!** We had to exit before this could happen.

\*I have (and will again) explained the effects of elevated estrogen levels post-cycle or after AAS discontinuance.

To further aggravate this post-cycle catabolic situation from long AAS protocols, the HPTA was suppressed and natural or endogenous androgen production was on girl-status. This allowed still existing elevated cortisol and estrogen levels to remain unchecked. (Which should cause testes challenged readers to say "yikes" in a high pitched voice) But this was all so unnecessary and no male needs "raisin syndrome".

But on the plus side, shaving would be much easier as would singing those old girl-band song of the past. Again, we had to exit the AAS protocol before this can occur.

First things first: The Max Androgen Phases constructed for Frank were intended as a means of altering the anabolic/catabolic ratio in favor of net protein mass increase on a very significant level. (I do like that word "significant") To do this Frank needed a plan that took into account Action/Reaction Factors so that he could keep much more AAS induced muscle mass gains post-cycle to build further upon as we progressed.

*"Okay, we know the body adapts by reaction to AAS beginning after 2-3 weeks. We know that some AAS aromatize to estrogen which needs to be checked and eliminated before we allow you to exit your AAS protocol, Frank. But we also know estrogen levels can actually enhance AAS results by several pathways including increased GH/IGF-1 production and increased muscle glycogen synthesis. We also know any androgenic induced muscle mass gains not solidified into high quality lean muscle tissue by a high anabolic environment will be lost quickly post-cycle. There is only one more main factor to consider. "Support networks". Think, Frank. Think!*

*Coach*

**\*Just remember the term "support networks" for now. It will be explained later...a lot.**

## **The Right Time Frames**

We saw the greatest results from any chemically induced alteration in homeostasis and the PTOR rate when we had a plan. First, we got in, grew hard, and got out before Frank's body could mount adequate counter measures. This meant a

time frame of 21-30 days. So no Max Androgen Phase (or any other protocol) could have had a high activity level beyond 30 days.

We had to create a quick and elevated androgenic environment to quickly increase mass and strength. No time was wasted that would have allowed the body to catch up with its anti-muscle counter measures. We called this the "**androgenic dominance period**". We also had to allow a high anabolic moderate - low androgenic elevated environment to solidify Frank's mass gains into quality muscle. We called this the "**anabolic dominance period**".

Most athletes have realized the greatest results and post-cycle lean mass retention when these two periods were about equal with an equal androgenic - to - anabolic transition period in between. Additionally, we had to create a long "most effective period" without significant inducing HPTA inhibition. Easy !

## **Estrogen Control, NOT Elimination**

Estrogen control was paramount for health and long-term result potential. But estrogen can increase IGF-1 production too, which was good. Estrogen also increases androgen receptor-site sensitivity. So we wanted estrogen to run rampant for the first two weeks of Frank's Max Androgen Phases **without** allowing it to cause gyno and female pattern fat deposits. Simple: We used an estrogen antagonist to block receptor-sites but allowed plasma estrogen levels to remain high.

Using Clomid as an example, it has been my experience that a novice AAS user required (if any) only 50 mg/d (50 mg per day). And an intermediate AAS user required 20-30 mg/d. An advanced AAS user commonly required 30-50 mg/d. A very advanced AAS user sometimes required 40-60 mg/d, and in most cases, some additional help from an aromatase inhibitor.

The key was to watch for signs of gyno and female pattern fat deposits, while keeping a close eye on blood pressure. This was always of the utmost concern during the building of the perfect beast. High blood pressure can introduce a variety of long term and life threatening negative side effects.

**\*Nolvadex decreases GH/IGF-1 synthesis and is therefore a poor choice as an estrogen antagonist.**

*Things we have learned from experience...*

Estrogen levels were kept near normal or below before we exited the AAS protocols. So we added an estrogen esterase inhibitor at about day #15 of a Max Androgen Phase to clear the system of excess estrogen before we exited. I have not noted many novice AAS/Max Androgen Phase users whom needed this precaution. But this was in relevance to dosages administered.

Some intermediate AAS users opted for Arimidex 0.5-1.0 mg/d, or Proviron 50-100 mg/d. Most advanced AAS users successfully utilized Arimidex 1.0-2.0 mg/d or Aromasin 50mg/d. This was, of course, unnecessary when a Cortisol/Estrogen Suppression Phase was layered in at the half-way point or beginning day #15 of a Max Androgen Phase.

*"Patience, Grasshopper!"*

*Coach*

I have and will repeat this fact again and again: Any effective plasma threshold exceeded before it failed to provide results was a growth period lost. This was true of any chemical muscle enhancer. This is in fact why so many athletes reached only 50-70 % of their potential. Receptor-site insensitivity (not AAS receptor sites) and the body learning new tricks to force homeostasis was the number one reason why we had often seen 220 LB 6 foot off-season bodybuilders using crazy dosages with poor results.

Of course there are ways to beat this too that I created for Frank, but we will discuss that later. First let's look at what the basic threshold for results were when long term potential and permanent gains were to be realized.

Since this section is about Frank's AAS protocols, let's focus upon them for now. Growth thresholds were established by plasma level in this discussion. Although there were several thresholds for each level of experience and drug, there were predictable **ranges** of dosages expressed in daily plasma levels we used as a basis.

First let me say again that it has been my experience that no athlete should have ever utilize AAS or other muscle chemistry until they have trained hard-core for at least 2-3 years as a natural.

If this was Frank, and he was a natural or somewhere in between, I would have utilized the following rough guide-lines. Natural training is a growth threshold also. I have trained some 250 LB, 8% body fat naturals, too.

*"Never waste a growth threshold. You need to do the physical work required first and foremost, you weenie!"*

*Coach*

**MAX ANDROGEN PHASE DOSAGES (EXAMPLES)**  
**(Actual Dosage of Androgen Minus Ester Weight)**

<b>Novice</b>	<b>Intermediate</b>	<b>Advanced</b>	<b>Very Advanced</b>
1.0-2.0 mg/lb weekly	2.0-3.5 mg/lb weekly	3.5-6.75 mg/lb weekly	6.75-insanity mg/lb weekly
<b>31.25-62.5 mg/d plasma level</b>	<b>62.5-125 mg/d plasma level</b>	<b>125-250 mg/d plasma level</b>	<b>250 mg/d plasma level</b>
218 mg-437.5 mg total weekly	437.5 mg-875 mg total weekly	875 mg-1750 mg total weekly	Above 1750 mg total weekly

**As I said, these are the rough guide-lines I utilized and they assumed bodyweights of the following with below 12% bodyfat:**

- Novices:** 185-218 LBS
- Intermediates:** 218 240 LBS
- Advanced:** 240-265 LBS
- Very Advanced:** 265 and up.

Of course bone structure and height played a role in potential weight/mass possibilities as did genetics. However, I have met few "average" individuals who could not have realized at least 265 LBS-plus with an off-season body fat level below 12%. Those who have failed usually did so by not planning for long-term potential adequately.

***"By learning Phase Cycling and Action/Reaction Factors this, like most obstacles, can be over come."***

Now that I have established some of the criteria utilized and incorporated into structuring protocols employed by Frank N. Steroid, it is time to discuss how it was applied.

The examples that follow are **not** intended as a guide or endorsement. They are simply what they are: The synergistic protocols that were used to build the perfect beasts, and another piece of Frank's story.

## MAX ANDROGEN PHASE Example #1 - Chart

DAY	A.	B.	C.
1.	Theramex 250 mg	Theramex 500 mg	Theramex 250 mg
2.	Theramex 250 mg	Theramex 500 mg	Theramex 250 mg
3.	Theramex 250 mg	Theramex 500 mg	Theramex 250 mg
4.	Theramex 250 mg	Theramex 500 mg	Theramex 250 mg
5.	Theramex 250 mg	Theramex 500 mg	Theramex 250 mg
6.	Theramex 250 mg		Theramex 250 mg
7.	Theramex 250 mg		Theramex 250 mg
8.	Theramex 250 mg		Theramex 250 mg
9.	Theramex 250 mg		Theramex 250 mg
10.	Theramex 250 mg		Theramex 250 mg
11.			Deca 200 mg
12.			Deca 200 mg
13.			Deca 200 mg
14.			Deca 200 mg
15.		Deca 400 mg	Deca 200 mg
16.		Deca 400 mg	Deca 200 mg
17.		Deca 400 mg	Deca 200 mg
18.		Deca 400 mg	Deca 200 mg
19.		Deca 400 mg	Deca 200 mg
20.	Durabolin 25 mg		Deca 200 mg
21.			
22.	Durabolin 50 mg		
23.			
24.	Durabolin 75 mg		
25.			
26.	Durabolin 100 mg		
27.			
28.	Durabolin 125 mg		

Deca = Deca Durabolan (nandrolone decanoate)

## MAX ANDROGEN PHASE

### Example #1 - Descriptions

#### Example #1 A

Theramex is a long acting testosterone. Like all testosterone it is highly androgenic and highly anabolic. It seems to be a common error to list the drug as a more powerful testosterone. Since this drug is esterized, suspension is still the more active. Theramex has an active-life of about 20 days and a half-life of about 10 days. Theramex in this example remained active in down-ramping dosages until day #30.

Durabolin is a short or fast acting nandrolone that is highly anabolic and moderate - low androgenic. It is also very protein sparing like all nandrolones. Durabolin has an active-life of about 3 days and a half-life of about 1.5 days. Durabolin will remain **effectively active** in this example until day #30-31.

**Example #1 A** was a single ramp Max Androgen Phase that was the first of the AAS protocols structured for my beast. This means that the plasma level established by Theramex was maintained and then replaced by Durabolin as Theramex daily plasma levels ramped down. This allowed for an excellent transition from a high androgen to a high anabolic environment.

If you look at the rough graph you will see this cycle example had a potential 20-day "**most effective period**" from about day #10-30. This is not to say that the first 10 days lacked activity of course. Naturally to waste any portion of a phase without gaining maximum results before the body could mount counter measures to alterations in homeostasis was silly.

At a later date Frank utilized Anadrol-50 in down- ramping dosages the first 9-10 days of this protocol, to maximize potential due to a quick up-ramp in plasma androgen levels. For this example he used Anadrol-50 at the dosages of: Day #1-2 300 mg, #3-4 250 mg, #5-6 200 mg #7-8 150 mg, #9-10 100 mg of Anadrol-50. This advanced technique is sometimes referred to as front-loading and allowed for about a 28-30 day most effective period.

The androgenic dominance period of this example was about 75% with only a 25% anabolic dominance period. Later we will discuss methods of dosage utilization employed by Frank for long-acting testosterone that allowed for a shorter androgenic dominance period.

## **Example #1 B**

Since Theramex has an active-life of about 20 days and half-life of 10 days, we know a single 250 mg injection would theoretically allow 12.5 mg to migrate into the vascular system daily. In example #1 A the 10-day administration period would therefore contribute 12.5 mg daily for each of the 10 injections. This means at the end of 10 days, the plasma level has been established at a threshold of 125 mg, theoretically. But it took 10 days plus to get there. The created advantage was a longer most effective period, and a slower androgenic down-ramp for the androgenic/anabolic transition.

In **Example #1 B**, we had doubled the daily Theramex dosage and cut the injection period in half by utilizing 500 mg each day for 5 consecutive days. This means each injection donated 25 mg daily to total plasma levels, again theoretically. The established plasma threshold was about 125 mg daily after day #5.

This was a single ramp Max Androgen Phase also. Since the androgenic activity down-ramped so quickly, a high anabolic/moderate androgenic AAS such as Deca Durabolin was a better transition choice in later protocols. This is because Deca is a little more androgenic in action than Durabolin. Deca has an active-life of about 14-16 days and half-life of 7-8 days. Deca Durabolin brand of nandrolone decanoate was usually dependable for a 15-16 day active-life. By beginning Deca on day #15 we were able to extend the 125 mg daily plasma level and most effective period to 25 days, or from about Day #5 to about day #30.

For other beasts, the initial 5 day period of this example has been quickly added into the 25 day most effective period by adding Anadrol-50: day #1-2 300 mg, #3-5 250 mg. Another option used was a fast/short acting testosterone such as Testosterone Propionate: Day #1-150 mg. This created a potential 28-30 day most effective period.

The androgenic and anabolic dominance periods of this example were about equal or 50% and 50%. But the androgenic to anabolic dominance transition could have been a little better. This would have mattered only when the example was utilized without other phase layers we will discuss as we continue.



## Example #1 C

This was a double-ramp Max Androgen Phase. A double-ramp protocol uses one drug to establish a first plasma level or threshold, and a second drug to continue the dosage up-ramp effect to a second plasma level or threshold.

This was for very advanced athletes (which Frank was not, yet) since it exceeded the 1500 mg weekly total plasma level. As you will read several times, an effective plasma threshold exceeded before results ceased was a growth level wasted.

Looking at the rough graph you will see Theramex established a potential plasma level of 125 mg/d after day #10. Deca Durabolin continued by creating a second plasma level up-ramp until a theoretical plasma level of about 250 mg/d was reached after day #20.

Since Theramex has an active-life of 20 days, the first day's (day #1) Theramex injection "ran out" about day #21 and the high androgenic period began to ramp-down until day #30, theoretically. Of course the androgen period down-ramp was mediated by the high anabolic period, so the most effective period was about 10 days. But it would be crazy to assume the Theramex established plasma level of 125 mg/d beginning about day #10-11 was not highly effective.

The androgenic period up-ramp between days #1-10 was again later augmented to increase the most effective period by utilizing a short/fast acting androgenic. Several possibilities existed as you will see as we continue. However, I had a favorite for this example. Parabolan/trenbolone is seriously androgenic stuff. Day#1-228 MG, day #4-152 MG.

Another common beast utilized option for example #1C was the addition of a high anabolic /low androgenic such as Primobolan to create a second step in transition from high androgenic to high anabolic periods. This would have been best utilized if Frank was one of those athletes who either lost post-cycle lean mass more easily than others, or if he had suffered HPTA suppression on a serious level even when employing such brief protocols.

Instead of administering 200 mg/d of Deca only: Beginning day #1 6 Deca 150 mg/Primo D 50 mg, #17 Deca 100 mg/Primo 100 mg, #18 Deca 100 mg/Primo 100 mg, #19-20 Deca 0 mg, /Primo D 150 mg. No doubt some would say this was useless. I say they have not dealt with the problem. This was about a 50%/50% ratio of androgenic/anabolic dominance period example.

The same 2 step transition was applied to **example #1 B** by a few other elite beasts: Day #1 7 Deca 300 mg/Primo D. 100 mg, #1 8 Deca 200 mg/Primo D. 200 mg, Day #1 9-20 Deca 150 mg/Primo D. 250 mg.

**Example #1A** has been adjusted to accommodate an intermediate athlete's needs by either cutting dosages in half or by utilizing the injections scheduled on days#1,3,5,7,9,20,22,24.

**Example #1B** was adjusted for the same athletes simply by cutting listed dosages in half. This provided a theoretical daily plasma level/threshold of about 62.5 mg/D.

"... which ain't no joke for an intermediate."

*Coach*

Each of these examples allowed Frank and other beasts to get in, grow hard and get out before the body could mount a full growth hating counter attack. With that said, let's go to the next page and check out Example #2, chart and description

## MAX ANDROGEN PHASE

### Example #2 - Chart

DAY	A.	B.	C.
1.	Sustanon-250 250mg	Test Mix 250mg <b>(B)</b>	M/P Mix 250mg <b>(D)</b>
2.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
3.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
4.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
5.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
6.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
7.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
8.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
9.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
10.	Sustanon-250 250mg	Test Mix 250mg	M/P Mix 250mg
11.	Dura/Deca 250mg <b>(A)</b>	D/E/D Mix 250mg <b>(c)</b>	D/P Mix 250mg <b>(E1)</b>
12.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
13.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
14.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
15.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
16.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg <b>(E2)</b>
17.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
18.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
19.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
20.	Dura/Deca 250mg	D/E/D Mix 250mg	D/P Mix 250mg
21.			D/P Mix 250mg <b>(E3)</b>
22.			D/P Mix 250mg
23.			D/P Mix 250mg
24.			D/P Mix 250mg
25.			D/P Mix 250mg
26.			Dur.250mg (Optional)
27.			Dur.250mg (Optional)
28.			Dur.250mg (Optional)

**(A)** Durabolin 50 mg + Deca Durabolin 200 mg

**(B)** Testosterone Propionate 50mg + Testosterone Enanthate 100mg + Testosterone Cypionate 100mg

**(C)** Durabolin 50mg + Equipoise 50mg + Deca Durabolin 150mg

**(D)** Masteron 50mg + Test Propionate 200mg

**(E1)** Durabolin 100mg + Test Propionate 150mg

**(E2)** Durabolin 150mg + Test Propionate 100mg

**(E3)** Durabolin 200mg + Test Propionate 50mg

## MAX ANDROGEN PHASE Example #2 - Descriptions

### Example #2 A

The use of multiple esters can create timing problems. In Frank's case this was simply solved, at times, by inducing a continuous daily plasma level of a chosen threshold, then continued with the same level during the high androgenic to high anabolic transition.

The example as shown induced a daily plasma level of about 250mg daily until day #24. At that point the plasma level gradually ramped-down (tapered off) until about day #36. However, it should be noted that by day #30 the existing plasma level were not excessive and therefore allowed Frank to stay within our intended 30 day high-activity time frame.

This was about a 33% Androgenic Dominance Period, 33% Androgenic/Anabolic Equivocal Period, And 33% Anabolic Dominance Period example. Some advanced athletes have cut the listed dosages in half and still made excellent progress. This was easily done by either utilizing the every-other-day listed dosages, or by cutting the dosage schedule in half. It should seem obvious that intermediate level athletes of my creation further decreased the dosage schedule to stay within acceptable ranges.

The example as listed was a single-ramp protocol that came on pretty fast due to Sustanon-250 containing 2 fast acting esters (Testosterone Propionate and Phenylpropionate). The use of Durabolin during the Anabolic Dominance Period was utilized to replace the declining plasma level of Testosterone Propionate contained in Sustanon-250. In truth this was not **necessary**, but it shows how a protocol was made far more efficient than was normally applied. This addition did increase the anabolic edge to some degree.

When Frank chose to induce an even quicker androgenic ramp he added an oral AAS up-front: Dianabol 40 mg/d on day #1-3, 30 mg/d on day #4-6, and 20mg/d on day #7-9. Since Dianabol is highly anti-catabolic in nature this addition helped to hold off the body's **initial** cortisol reaction a bit longer when administered for this brief period.

### Example #2 B

By mixing testosterone Propionate, Testosterone Enanthate, and Testosterone Cypionate, the androgenic period up-ramp came on pretty fast. This closely simulated Sustanon 250 in action and effects. The use of Durabolin, Equipoise, and Deca Durabolin mimicked the active and half-lives of the Testosterones as listed in order.

1. Durabolin/Testosterone Propionate -72 hour active-life, 36 hour half-life.
2. Equipoise/Testosterone Enanthate -8 day active-life, 4 day half-life.
4. Deca Durabolin /testosterone Cypionate 14-16 day active-life, 7-8 day half-life.

\* Interesting: I have found 2 active and half-life listings for Equipoise (Boldenone Undecylenate) 16 and 8 days as well as 8 and 4 days respectively.

*Monitoring blood tests however supports the latter more closely.*

This example also established about a 250 MG daily plasma level and had nearly the same androgenic/anabolic dominance ratio. Since any example shown that provided a 250 MG daily plasma level was an upper effective threshold, this too was a very advanced protocol. All advanced athletes such as Frank made exceptional progress by utilizing the injection dates and dosages listed on odd days only (which allowed for a 125 MG/D plasma level).

As in **example #2 A**, novice and intermediate Testosterone users followed the odd day only (EOD) protocol and cut dosages in half (this provided a 62.5 MG/D plasma level). This example provided excellent results as discussed. But the effects were compounded when administered as a Max Mix and site injection protocol (Which we will discuss later). This was a single ramp example.

Again, sometimes getting creative with esters was just fun. And it was a chance to clean out the gear-box from unused post-cycle chemistry. Of course, this example has been simplified by utilizing one Testosterone ester with one high anabolic ester and adding an androgenic oral in the front... when needed.

### **Example #2 C**

**Example #2 C** utilized all short /fast acting esters. Each of which had an active - life of about 72 hours and a half-life of 36 hours.

**For athletes that were prone to estrogenic side effects I liked Masteron up-front in some Max Androgen Phases for 2 reasons:**

1. It is more androgenic than Testosterone.
2. It possessed strong anti-estrogen qualities. Add to this its anti-catabolic effect and quick up-ramping effect.

**The use of Testosterone Propionate in a Max Androgen Phase had advantages:**

1. Time frames were easily controlled. It ramped up IGF-1 production in the liver.
2. It was a more singular potent Testosterone than any other except Suspension.

The use of Durabolin was perfect for well-timed high androgenic to high anabolic period transition. However, since it was only moderate-low androgenically, it was best utilized by those who had above average post-cycle lean mass loss problems.

If the reader has been doing the Math on active and half-lives, you already know this was another very advanced dosage threshold of 250 MG/D and how it was adjusted for advanced, Intermediate, and Novice threshold levels. Learn the Math! It took the guess work out of any protocol.

The reader should also realize this was a single ramp example and that the most effective period was from about day #3 to about day #27. Would orals have been effective up front? No! But you knew that already. Right?

\* Now, can you do the Math for most effective periods for example #2 A, and B? By using a graph anyone can.

We have the first two phases down ... let's move on to next page and take a look at phase three ...

## MAX ANDROGEN PHASE Example #3 - Chart

DAY	A.	B.	C.
1.	Sustanon250 1250mg	Theramex 500mg	T.S. 100mg <b>(B)</b>
2.	Laurabolin 500 mg	Theramex 500mg	T.S. 100mg
3.	Test. Prop. 200mg <b>(A)</b>	Theramex 500mg	T.S. 100mg
4.		Theramex 500mg	T.S. 100mg
5.	Sustanon250 1250mg	Theramex 500mg	T.S. 100mg
6.	Laurabolin 500 mg	Anadur 500mg	T.S. 100mg
7.	Test. Prop. 200mg	Anadur 500mg	T.S. 100mg
8.		Anadur 500mg	T.S. 75mg/W 25mg <b>(C)</b>
9.	Sustanon250 1250mg	Anadur 500mg	T.S. 75mg/W 25mg
10.	Laurabolin 500 mg	Anadur 500mg	T.S. 75mg/W 25mg
11.	Test. Prop. 200mg		T.S. 75mg/W 25mg
12.			T.S. 75mg/W 25mg
13.			T.S. 75mg/W 25mg
14.			T.S. 75mg/W 25mg
15.			T.S. 50mg/W 50mg <b>(D)</b>
16.			T.S. 50mg/W 50mg
17.			T.S. 50mg/W 50mg
18.			T.S. 50mg/W 50mg
19.			T.S. 50mg/W 50mg
20.			T.S. 50mg/W 50mg
21.			T.S. 50mg/W 50mg
22.			T.S. 25mg/W 75mg <b>(E)</b>
23.			T.S. 25mg/W 75mg
24.			T.S. 25mg/W 75mg
25.			T.S. 25mg/W 75mg
26.			T.S. 25mg/W 75mg
27.			T.S. 25mg/W 75mg
28.			T.S. 25mg/W 75mg

**(A)** Testosterone Propionate

**(B)** Testosterone Suspension

**(C)** Testosterone Suspension 75mg + Winstrol Depot (Injectable) 25mg

**(D)** Testosterone Suspension 50mg + Winstrol Depot (Injectable) 50mg

**(E)** Testosterone Suspension 25mg + Winstrol Depot (Injectable) 75mg

## MAX ANDROGEN PHASE

### Example #2 - Descriptions

#### Example #3 A

**Example #3 A:** When looking at this example the reader should realize a few facts. First, that the example was utilized by a very advanced athlete and that a plasma level threshold of 250 mg/d was established very quickly. Second, that the dosage schedule allowed a better high androgenic /high anabolic transition than **example #3 B** (which also has been altered to accomplish the slower transition period for some applications in the past) and that the dosages as listed required a crazy injection site distribution to administer 35 ML of Laurabolin in one day.

What is interesting is that I had utilized this protocol at half the dosages while on a two week Mexican vacation with great success. Sometimes crazy things simply worked. However, some athletes (like Frank) who attempted this suffered serious oil trauma (which are flu like symptoms) due to the high oil volume introduced in such a brief period. I utilized the protocol by creating a Max Mix for days #2, 6, and 11 that allowed for a total of 20 ML on each day. Then site injected 2 ML into each bicep, tricep, lateral delt, calve, and outer pec. I did so after training chest/shoulders/arms on these days. Hey, I was on vacation! Again, I did this at half the listed dosages.

This example was a single ramp since we established the 250mg/d plasma level threshold up front. The androgenic/anabolic transition period ratio was about 40-60% respectively.

While in Mexico, I found some Fludestrin injectable 100 MG/ML, which is the injectable form of Teslac (Testolactone) and utilized 100 MG per day with 30 MG of Nolvadex before bed. This had some prolonged estrogen inhibiting effects long after discontinuance since I only used 0.5 mg/d of Arimidex for the rest of the protocols active-life. Faslodex (fulvestrant) would have been another long-acting injectable anti-estrogen option, but none was found.

So do the Math. Sustanon-250 has an active-life of about 20 days though some minor activity exists for a couple more days. Laurabolin has an active-life of over 26 days with a half-life of about 13 days. This example provided adequate androgenic stimulation until about day # 26-29 and excellent anabolic support for transition and mass gain solidifying effects until about day #32. So using two long acting esters together was quite effective when timing issues and chemical active durations were considered. Omnadren has been employed to replace Sustanon-250. But the stuff was often dirty.



I spent most of this vacation on the beach enjoying the sites. The rest was training, eating, and sleeping. This allowed me to travel to my next stop in the USA without chancing legal problems. Smuggling was and is a very bad idea. And why bother since it was not necessary?

The next example utilized Theramex and Anadur, which are both French AAS. I ran into a lot of both during my Mexican vacation if asked for by name. This of course provided another example of how to avoid legal issues when someone was living in, or traveling to, a less tolerant country. I have a friend who lives in Canada. He travels to France on business 3-4 times a year. He has a prescription for Growth Hormone and a doctor that treats him for "excessive estrogen accumulation". He is also smart enough not to destroy his career by getting involved in smuggling. He wears very large suits to his business meetings, by the way. Concerning the products I had encountered, the French spelling was Anador and the German and Swiss stuff was spelled Anadur.

### **EXAMPLE #3 B.**

This was the protocol my friend used for his trips to France. As you know, Theramex has an active-life of about 20 days and half-life of about 10 days. This means that the androgenic period of dominance was brief since Anadur has an active-life of 28 days theoretically, and a half-life of about 14 days. I liked Anadur for its high anabolic and protein sparing qualities. Unfortunately, it only came in a 25mg or 50mg/ml strength. This again was best utilized as a Max Mix for multiple site injections.

The example was a quicker double ramp protocol. Meaning the first substance established a plasma threshold and the second continued it. In this example, Theramex had established a 125mg/d plasma level after day #5 and Anadur continued the up-ramp until a second plasma level and threshold of 250mg/d after day #10. Obviously my friend was a very advanced athlete. (Who really should have been competing at high levels)

The example has been adjusted to a 125mg/d maximum plasma level by cutting daily dosages listed in half. (Duh). This was well below the 1500-1750 mg/week maximum AAS total plasma level I felt was the top for any beast including (and especially for) Frank N. Steroid.

This example provided excellent results and good high quality lean mass due to the prolonged anabolic dominance period. Sustanon-250 has been employed to replace Theramex to induce a quicker androgenic ramp. When that was the goal I have utilized the protocol as is for a very advanced athlete and added Testosterone Propionate to establish a rapid androgenic ramp and change the protocol to a single ramp: Day #1-200mg, #3-100mg, #5-50mg, or half that for the advanced 125 MG/D alteration.

### **Example #3 C.**

This was a somewhat dirty protocol... but it worked so darn well! Both Testosterone Suspension and Winstrol Depot have an active-life of just about a day. So the plasma level threshold that resulted from this protocol was about 100mg/d by day #2.

Testosterone Suspension is the most androgenic Testosterone, in my opinion. The results from a 100mg/d plasma level realized with this example was about the same as a 125mg/d plasma level threshold from Testosterone Propionate. This is obviously due to the adjoined ester weight for the propionate.

Testosterone Suspension hurt going in and left the site area sore for a day or two. Winstrol Depot is actually a dirty drug due to its c17-alkylated structure. I have very rarely exceed 100 MG/D and then only for a period of no more than 21 days. Milk Thistle was a must with this drug for any beast who liked having a liver.

"A necessary note. There are those who wholeheartedly believed stanozolol (Winstrol) in its injection form was not hepatic toxic at any dosage. I have reviewed Chemical Panels for many years and can say conclusively that the drug does cause liver stress at elevated and prolonged dosages without fail.

Personally I was amazed at the results this protocol provided. Especially when it was utilized site-specifically. Some of the most striated bodies in competition have been created by injecting each day's dosage directly into the muscle after it was trained (site specifically).

I have a theory as to why the results were so profound. It was simply due to the fast acting qualities of a water based product and the lack of an ester requiring the activity of esterase enzyme. The lion's share of the esterase enzyme exists in the *circulatory system*, not in muscle tissue itself. Therefore the free active form of an esterized drug has less effect upon site of injection but non-esterized drugs have the greatest localized effect.

Another effect was the synergy of IGF-1 production increase from c17- alkylated AAS during the deactivation process in the liver. This is also true site specifically in the case of non-esterized AAS due to muscle cell receptor interaction.

A second synergy exists between the Winstrol molecule and progesterone. Winstrol has a molecule structure very similar to progesterone. Progesterone is an estrogen of course, but it possesses androgenic qualities as well. However, even though Winstrol fits into progesterone receptor-sites, it does not activate estrogenic activity. Kind of like Nolvadex: it acts as an estrogenic antagonist to a certain degree, yet induces secondary androgenic effects through progesterone receptors. Unfortunately this can affect sex drive negatively.

When using Testosterone Suspension athletes needed all of the estrogenic-control possible. This example as listed was a great protocol for Frank when evaluating result potential: The androgenic dominance period and anabolic dominance periods are about a 50% equal ratio. The most effective period is about 28 days, and the system cleared within a couple of days after discontinuance. Too bad it was dirty.

When I wanted to increase the plasma threshold of **Example 3C**, I did not increase the dosages of Winstrol. I instead added Equipoise. Since the protocol had dramatic hardening effect over all (with good estrogen control), I preferred to add the Erythropoiesis stimulation effects of Equipoise. This increased red blood cell count and added to vascularity and muscle fullness. However, it was a mandatory factor to monitor CBC's so as to protect against platelet issues.

When I chose not to increase Testosterone Suspension dosages to accommodate the plasma increase from Equipoise, I choose Testosterone Enanthate. How was that done? Go back and look at example #2 B. I followed the schedule for Testosterone Enanthate as exemplified and doubled the Equipoise dosages. The result was a 200mg/d plasma threshold level. I know several very advanced athletes who favored this layered combination for Max Androgen Phases.

We have discussed the single and double ramp plasma level examples developed for Frank N. Steroid and other beasts. For novice, intermediate, advanced or very advanced athletes, double ramp protocols were the best "transition" Max Androgen Phases. Instead of simply jumping to the next plasma threshold, double ramp protocols allowed an athlete to increase long-term growth potential by utilizing both their prior threshold and the next step. Think about it.

## Other Max Androgen Phase Examples?

Of course there many examples of Max Androgen Phases possible including layered, multi-staged, single and double ramped...and others.

### 3 Stage Max Androgen Phase

DAY	DRUGS	DAY	DRUGS
1.	Test Cypionate 200 mg + Test Propionate 100mg	22.	Boldenone Undecylenate 100mg
2.		23.	Boldenone Undecylenate 100mg
3.	Test Cypionate 200 mg + Test Propionate 100mg	24.	Boldenone Undecylenate 50mg + Nandrolone Decanoate 150mg
4.		25.	
5.	Test Cypionate 200 mg + Test Propionate 50mg	26.	Boldenone Undecylenate 50mg + Nandrolone Decanoate 150mg
6.		27.	
7.	Test Cypionate 200 mg + Test Propionate 50mg	28.	Nandrolone Decanoate 200mg
8.		29.	
9.	Test Cypionate 200 mg + Test Propionate 25mg	30.	Nandrolone Decanoate 200mg
10.		31.	
11.	Test Cypionate 200 mg + Test Propionate 25mg	32.	Nandrolone Decanoate 200mg
12.		33.	
13.	Boldenone Undecylenate 100mg	34.	Nandrolone Decanoate 200mg
14.	Boldenone Undecylenate 100mg	35.	
15.	Boldenone Undecylenate 100mg	36.	Nandrolone Decanoate 100mg + Trenbolone Acetate 50mg
16.	Boldenone Undecylenate 100mg	37.	
17.	Boldenone Undecylenate 100mg	38.	Trenbolone Acetate 100mg
18.	Boldenone Undecylenate 100mg	39.	
19.	Boldenone Undecylenate 100mg	40.	Trenbolone Acetate 100mg
20.	Boldenone Undecylenate 100mg	41.	
21.	Boldenone Undecylenate 100mg	42.	Trenbolone Acetate 100mg

- Liquidex 1-1.5mg daily
- Day 43-56 T-3 25mcg 2xd, Clenbuterol 80-120mcg 1xd, PCF-2a 1 mg 2-4xd, Clomid 50mg/d

## Max Androgen Phase Example with Recovery Layer Option 1A

DAY	DRUGS
1.	Testosterone Cypionate 200mg/D-Bol 50mg
2.	Testosterone Cypionate 200mg/D-Bol 50mg
3.	Testosterone Cypionate 200mg/D-Bol 50mg
4.	Testosterone Cypionate 200mg/D-Bol 50mg
5.	Testosterone Cypionate 200mg/D-Bol 40mg
6.	Testosterone Cypionate 200mg/D-Bol 40mg
7.	Testosterone Cypionate 200mg/D-Bol 40mg
8.	Testosterone Cypionate 200mg/D-Bol 40mg
9.	Testosterone Cypionate 200mg/D-Bol 30mg
10.	Testosterone Cypionate 200mg/D-Bol 30mg
11.	Nandrolone Decanoate 200mg/D-Bol 30mg
12.	Nandrolone Decanoate 200mg/D-Bol 30mg
13.	Nandrolone Decanoate 200mg/D-Bol 20mg
14.	Nandrolone Decanoate 200mg/D-Bol 20mg
15.	Nandrolone Decanoate 200mg
16.	Nandrolone Decanoate 200mg
17.	Nandrolone Decanoate 200mg
18.	Nandrolone Decanoate 200mg
19.	Nandrolone Decanoate 200mg
20.	Nandrolone Decanoate 200mg
21.	Oxandrolone 20mg
22.	Oxandrolone 20mg/Clomid 50mg 2xd/Glucophage 425mg 2xd
23.	Oxandrolone 20mg/ Clomid 50mg 2xd/Glucophage 425mg 2xd
24.	Oxandrolone 30mg/ Clomid 50mg 2xd/Glucophage 425mg 2xd
25.	Oxandrolone 30mg/ Clomid 50mg 2xd/Glucophage 425mg 2xd
26.	Oxandrolone 30mg/ Clomid 50mg 2xd/Glucophage 425mg 2xd
27.	Oxandrolone 40mg/Clomid 50mg/Glucophage 425mg 2xd
28.	Oxandrolone 40mg/ Clomid 50mg/Glucophage 425mg 2xd
29.	Oxandrolone 40mg/ Clomid 50mg/Glucophage 425mg 2xd
30.	Clomid 50mg /Arimidex 0.5mg/Glucophage 425mg 2xd
31.	Clomid 50mg /Arimidex 0.5mg/Glucophage 425mg 2xd
32.	Clomid 50mg /Arimidex 0.5mg/Glucophage 425mg 2xd
33.	Clomid 50mg /Arimidex 0.5mg/Glucophage 425mg 2xd
34.	Clomid 50mg /Arimidex 0.5mg/Glucophage 425mg 2xd
35.	Clomid 50mg /Arimidex 0.5mg/Glucophage 425mg 2xd

- Day 5-35 Nolvadex 10mg 2-3xd
- Day 29-42 HCG1000iu "
- Continued Glucophage through day 42
- Glucophage was administered AM and PM 1 2 hours apart (425mg = half tab)

## Max Androgen Phase Example with Recovery Layer Option 1B

DAY	DRUGS
1.	Aratest 250mg/Oxymetholone 200mg
2.	Oxymetholone 200mg
3.	Aratest 250mg/Oxymetholone 200mg
4.	Oxymetholone 200mg
5.	Aratest 250mg/Oxymetholone 150mg
6.	Oxymetholone 150mg
7.	Aratest 250mg/Oxymetholone 150mg
8.	Oxymetholone 150mg
9.	Aratest 250mg/Oxymetholone 100mg
10.	Oxymetholone 100mg
11.	Aratest 250mg/Oxymetholone 100mg
12.	Oxymetholone 100mg
13.	Aratest 250mg/Nandrolone Decanoate 125mg
14.	
15.	Aratest 250mg/Nandrolone Decanoate 125mg
16.	
17.	Aratest 125mg/Nandrolone Decanoate 250mg
18.	
19.	Aratest 125mg/Nandrolone Decanoate 250mg
20.	
21.	Aratest 125mg/Nandrolone Decanoate 250mg
22.	
23.	Trenbolone Acetate 50mg
24.	
25.	Trenbolone Acetate 100mg
26.	
27.	Trenbolone Acetate 100mg
28.	

- Arimidex/Liquidex 1-1.5mg daily (or 50mg Aromasin daily)
- Day 29-35 Clomid 100mg daily and day 36-4650mg daily
- HCG 500iu daily day 26-46

## MAX ANDROGEN PHASES Max Mix/Site-Injection (Examples)

Lets go back to Max Androgen Phase **example #2 C** and discuss additional possibilities that were employed. I have noted over the years that AAS injected directly into a muscle belly increased localized growth. I believe this was due to two factors. First, that "on site" availability of any fast/short acting muscle chemistry induced greater localized receptor-site stimulation. This may have been due in part to the fact that lagging body parts often have a lower circulatory supply of blood and the chemistry/nutrients it carries.

This in turn often relates to inadequate development of vascular and nerve factors in these areas as well. ("Support Networks". We are getting there) Second, was the theory of site-injection protocols. The theory was that by introducing a space occupier, muscle fiber and belly fascia was forced to stretch and increase in area. This induced localized growth in turn due to... greater area allowed the hypertrophy of muscle cells and fibers to increase.

Think of it this way: If you put a balloon in a coffee can, you would only be able to blow it up so far. It would simply occupy available space. However, if you created a larger coffee can, you could more easily increase the volume of the balloon.

Without a doubt, Testosterone Propionate was one of the best site-injection androgens I know of. It was fast acting and seriously ramped up both IGF-1 production via the liver. However, the most effective AAS for the purpose of localized androgen receptor stimulation were non-esterized. This was due to the fact that a greater percentage of the enzyme esterase exists in the blood than in muscle tissue itself. Since esterized AAS are dependent upon this enzyme to free the active substance from the ester, and that not much esterase is present at an intramuscular injection site, less localized androgen receptor-site stimulation occurred.

\*Examples of non-esterized AAS: Winstrol Depot, methandrostenolone (Dianabol) injection, and Testosterone Suspension. There are black market versions of Masteron and Boldenone exempt of additional esters that appeared to work quite well too.

So let's go back to the basic structure of **example #2 C** and I will explain the protocol I created for Frank's initial Max Mix for a site-injection protocol. We established a daily plasma level of 100mg/d and used the same androgenic and anabolic dominance periods.

Site-injection protocols increased muscle size significantly. (Like arms 1-3" in about 3 weeks). After the initial size protocol was completed, a maintenance period of 6-8 months was necessary utilizing 2-3 ML of **SEO** (site enhancement oil... like one of the [www.biodesignlabs.com](http://www.biodesignlabs.com), [www.hmgear.com](http://www.hmgear.com), or Synthol products) per localized

muscle per week to realize more permanent and significant results. We will discuss this more in detail in a moment. Let's first look at how Frank's Max Mix was created for this purpose.

### **Example #2 C 100 MG/day plasma level (Excluded Masteron)**

To utilize Example #2 C as a site-injection protocol it was necessary to create 4 Max Mix vials. Frank's protocol called for 3ml per injection site. So 6ml total daily (3ml each bicep, tricep, lateral delt, pec etc.)

#### **Max Mix Vial A**

Test. Prop. (100mg/ml) 14 ML  
SEO 28 ML  
Benzyl Alcohol ½ ML

#### **Max Mix Vial B**

Test. Prop. (100mg/ml)10.5 ML  
Dura(50mg/ml) 7ML SEO 24.5  
ML Benzyl Alcohol ½ ML

#### **Max Mix Vial C**

Test. Prop. (100mg/ml)7 ML  
Dura(50mg/ml) 14ML SEO 21  
ML Benzyl Alcohol ½ ML

#### **Max Mix Vial D**

Test. Prop.(100mg/ml)3.5ML  
Dura(50mg/ml) 21 ML SEO  
17.5 ML Benzyl Alcohol ½ ML

Vial A Days #1-7 = per ml 33.3mg Test. Prop.

Vial B Days #8-14 = per ml Test. Prop. 25mg Durabolin 8.3mg

Vial C Days #1 5-21 = per ml Test. Prop. 16.6mg/ Durabolin 1 6.6mg

Vial D Days #22-28 = per ml Test. Prop. 8.3mg/Durabolin 25mg

\*Benzyl alcohol burned a little but was a must for sterilization with all but the hm-gear space occupiers.



## Max Mix/Site-Injection Training (Example)

Training took into account each injection site. Frank's arms had good shape but sucked in size, his biceps needed the most help. His triceps were only lacking overall size, and lateral delts needed to be rounder.

Body Part Trained	Site Injected
Day#1 Legs (Complete)	Tricep belly 3ml each
Day#2 Chest	Tricep horse shoe 3ml each
Day#3 Back	Bicep Peak 3ml each
Day#4 Off	Lateral delt 3ml each
Day#5 Arms	Bicep Peak 3ml each
Day#6 Shoulders/Traps	Lateral Delt 3ml each
Day#7 Off	Bicep Peak 3ml each

It was important to do site-injections after the work-out so as to avoid disturbing the newly expanded fiber area. This also helped reduce bruising. Frank was often sore but much larger.

The IGF-1 produced from Testosterone Propionate and the initial tissue stretching, aided in vascular tissue growth within the newly acquired area quite nicely. But, of course, not as well as a GH/Insulin /T-3 thyroid **Absolute Anabolic Phase** layer (which aided in nerve growth as well). It was layered over this example at a later date but not during this initial protocol.

Maintenance injections were utilized for 6 more months with SEO (Site Enhancement Oil). It was Paramount to assure sterile conditions no matter how clean or pure a product was supposed to be. The benzyl alcohol did help to maintain sterility in each vial. However, before loading each syringe, Frank dropped the Max Mix vial being used at the time into a pan of water Oust below boiling and not on the stove) for 10 minutes and allowed to cool before use.

## Aspiration?

Also, it was mandatory to pull back on the syringe plunger once the needle was inserted into the muscle. If it filled with blood, it was in a vein and would have been a very bad place for administration. This was true of any intramuscular injection.

***"Often I have seen those who hurry along an unplanned path to destruction".***

*Coach*

<b>Max Mix Vial A</b>	<b>Max Mix Vial B</b>	<b>Max Mix Vial C</b>
4 Sust. 250 (1000mg)	3 Sust-250 (750mg)	5 ml Test. Prop (500mg)
1 Parabolan (76mg)	2 Parabolan (152mg)	3 Parabolan (228mg)
2 ml Equipoise (100mg)	4 ml Equipoise (200mg)	6 ml Equipoise (300mg)
34 ml Protest	32ml Protest	28ML Protest
½ ml Benzyl Alcohol	½ ml Benzyl Alcohol	½ ml Benzyl Alcohol
<b>42 ml total approx.</b>	<b>42 ml total approx.</b>	<b>42 ml total approx.</b>

- **Vial A** Day #1 -7 6 ml per day
- **Vial B** Day #8-14 6 ml per day
- **Vial C** Day #15-21 6 ml per day

What Happened Next...

## **Anabolic Lean Mass Retention Period**

### **Day#**

- |                        |                         |                         |
|------------------------|-------------------------|-------------------------|
| 22. Clenbuterol 80 mcg | 29. Ephedrine 75 mg     | 36. Ephedrine 150 mg    |
| 23. Clenbuterol 80 mcg | 30. Clenbuterol 100 mcg | 37. Ephedrine 150 mg    |
| 24. Ephedrine 75 mg    | 31. Clenbuterol 100 mcg | 38. Clenbuterol 120 mcg |
| 25. Ephedrine 75 mg    | 32. Ephedrine 150 mg    | 39. Clenbuterol 120 mcg |
| 26. Clenbuterol 80 mcg | 33. Ephedrine 150 mg    | 40. Ephedrine 150 mg    |
| 27. Clenbuterol 80 mcg | 34. Clenbuterol 100 mcg | 41. Ephedrine 150 mg    |
| 28. Ephedrine 75 mg    | 35. Clenbuterol 100 mcg | 42. Clenbuterol 120 mcg |

## **Closing Thoughts**

This was an interesting protocol. Day 1-7 was a high androgenic period, Day 8-14 began the androgen/anabolic transition, day 15-21 completed it.

This was intended as a site-injection protocol (sometimes referred to as Symmetry Rounds) utilizing 6 ml daily split between (3 ML each) left and right sides. The following 21-day period (days 22-42) utilized the anticatabolic/thermalgenic qualities of Ephedrine and Clenbuterol.

The anabolic lean mass retention period? Think about it for a few minutes.

On day #42 the system was mostly clear and ready to go again. The "most effective period" of each 7 day period continued a good 7-10 days following each final injection. This of course means the activity and growth period continued until about day #27-31 from the AAS period, in a down ramping manner, as Clenbuterol (In divided dosages of course) and Ephedrine (In 3 divided dosages) aided in lean mass retention. Anti-estrogen measures continued through day #30-35. Frank utilized the

IGF-1 production increase from elevated estrogen levels for the first 15 days by following a Clomid protocol administering 50mg daily.

## **Points To Ponder During Protocol Construction ... But Few Do**

### **Estrogen Antagonist**

Clomid is an estrogen receptor antagonist, which means it only blocks estrogen receptor-sites, not the circulatory effects of estrogen passing through the liver ***or the positive effect it has upon GH release.***

### **Aromatase Inhibitors**

The last 13 days Frank followed a simple 1-1.5mg/d protocol using Arimidex or 50mg/d of Aromasin (25mg/d was not effective when higher dosages of aromatizing AAS were employed. I am sure some will disagree, but blood tests for 17 $\beta$ -estradiol do not lie). These of course are estrogen aromatase inhibitor. Inhibitors of this nature prevent or minimize aromatization of AAS, and natural endogenous estrogen production.

This was quite important because coming out of any high androgen cycle with elevated estrogen levels trying to dominate endogenous androgen production almost always results in even further decreased HPTA function. This means post-cycle lean mass retention would have been less and fat accumulation would have been a problem.

### **Trenbolone and Boldenone**

A notable benefit of Trenbolone and Equipoise (Boldenone) administration was the distinct lack of aromatization attributed to the prior drug and moderate to the latter. And both provided high quality lean mass results.

Parabolan really was (real Parabolan) an amazing drug. It provided rapid strength gains while aiding in excellent lean mass gains. It also tended to aid in fat loss. But it can be rough on the kidneys so lots of water consumption was important. 3 weeks at these dosages left little reason for concern however.

Parabolan had a great deal of activity, but in a pinch I have substituted Masteron. I once ran this cycle 3 times in a row and gained over 30 LBS. That would not seem amazing except that I dropped about 8 LBS of fat during that period which means lean mass gains were serious. (I also had these crazy spider veins on my legs, arms, and chest) Frank's results were similar.

\*Several different products contain trenbolone acetate. No adjustment in dosage was necessary when substituting trenbolone acetate for Parabolan due to frequency of listed administration. (Parabolan is only Black Market manufactured now)

### **Trenbolone and Tissue Duress**

Trenbolone acetate is usually a black market of kit manufactured AAS. As such the amount of benzyl alcohol in the products tends to cause coughing and other respiratory trauma for some. (Not good. That is the body removing the benzyl alcohol by way of the lungs) Benzyl alcohol can be toxic to tissues in higher dosages. This is why the older Finabol and other black market preparations sometimes induced liver and kidney duress. Of course the amount of benzyl alcohol in those products was much higher than current products.

### **Anti-Progestin?**

We often hear of "Deca-Dick" or trenbolone induced erectile dysfunction. Oddly enough, both trenbolone and nandrolone possess progestin-type qualities. This means that both can bind to progesterone receptors and induce gyno, erectile dysfunction and sex-specific fatty deposits.

Though tamoxifen citrate (Nolvadex) does have some limited anti-progestin value in that there is a small degree of progesterone receptor blockage that occurs from its use. With exception of RU-486 (the abortion drug) there has not been an effective anti-progestin drug available. Until now!

### **Don't Kill The Messenger ... Kill the Receiver**

**Faslodex** (fulvestrant) is an estrogen receptor antagonist similar in action to Nolvadex and Clomid. But the way it does so is really interesting.

Faslodex effectively induces a *decrease (down-regulation) in estrogen and progesterone receptor concentration*. The drug causes the estrogen and progesterone receptors to change shape and become less functional. Without functioning estrogen receptors, estrogen cannot signal the tissues to do estrogenic/progestin things like grow gyno, deposit female pattern fat deposits or hinder sexual function. (Nor is there an increase in the female hormone prolactin. Huh? More later when we talk about anti-prolactins)

The drug is available as strength of 50 mg/ml (available in one 250 mg or two 125 mg pre-filled syringes) and administered intramuscularly at a dosage of 125mg 2 times monthly or 250mg once monthly. (Yup, pretty long acting stuff)

In clinical studies Faslodex has been shown to be more effective at a monthly dosage of 250mg than 1 mg daily of Arimidex. Interesting is that there is only a positive alteration in LH/FSH release during use therefore supporting HPTA function in males.

## **Another Antagonist?**

**Fareston** (toremifene citrate) is an estrogen receptor mixed agonist/antagonist. In fact it is classified as a true Selective Estrogen Receptor Modulator. This means that the drug is selective in specific types of estrogen receptors it blocks and those it activates. For AAS users this is a real plus due to the reality that many estrogen receptor antagonists hinder glucose storage and GH release...and Fareston does not.

Sadly, its use also hinders LH/FSH production thus having no value in HPTA regeneration. In studies upon males there was a marked reduction in HPTA activity resulting in a decrease in androgen and sperm production. But on a plus side it has a positive effect upon cholesterol levels favoring HDL (good cholesterol) production and significant LDL (bad cholesterol) reduction.

An effective daily dosage for Fareston is 60mg/d and it best administered as such even with its half-life of over 4 days. This is like Clomid in that the daily accumulation acts as a buffer for physiological adaptation to some extent.

\*I note this only due to a bottle I came across that had the right name but the wrong chemical on the label.

FARESTON (toremifene citrate) Tablets for oral administration each contain 88.5 mg of toremifene citrate, which is equivalent to 60 mg toremifene. FARESTON is a nonsteroidal antiestrogen. The chemical name of toremifene is: 2-(p-[(Z)-4-chloro-1,2-diphenyl-1-butanyl]-phenoxy)-N,N-dimethylethylamine citrate (1:1).

## **Back To Breast Implants ... Err ... I Mean, Site Injections ...**

The idea of site-injection protocols was similar to the intent of breast implants. Only in this case, the muscular tissue size was real. Let me explain.

Muscle is like water in a fish tank. Muscle is the water, and the fascia that encases and contains it is the tank. If the fascia is stretched and enlarged, muscular tissue will, in time, fill the larger area. When inert (Hopefully) substances such as Synthol were injected directly into a muscle belly in great enough volume (*In an accumulative manner*) the fascia containing muscle fibers and the fascia containing the individual muscle section itself had to stretch in order to maintain a sort of balance or homeostasis.

When a product such as those offered by Hardcore Muscle Gear (hmgear.com), that contained free prosteroids and prohormones (which were free non-esterized androgens) was utilized, the hypertrophy of muscle cells accelerated thus filling the new area with vascular tissue more quickly. And when an AAS mixture was utilized with the SEO, growth was increased again.

Almost any cycle structure one could think of has been utilized as a site-injection protocol. However, some were significantly more effective than others. Personally, I preferred faster acting esters or non-esters because cycles were easier to control for length of activity and the maintenance injection periods did not interfere with probable release periods of long acting AAS. More important the AAS activity was readily available site-specifically for symmetry alterations.

## **WHAT IS AN ESTER AND WHY WOULD ANYONE CARE?**

First let me explain, esters and long-vs-fast acting AAS. Way back when testosterone was first isolated and utilized as an injected exogenous steroid, raw testosterone (Like suspension) was the only choice. Raw testosterone is not very soluble. It dissolves better in water or blood than in fats, but not much better.

As you know, fats take a longer period to be metabolized and some fats take longer than others. So chemists decided to make testosterone fat soluble, which involves the additions of an ester to the AAS molecule.

The ester causes the AAS to remain within fat contained in muscular tissue until an enzyme called esterase helps it to get into the blood stream by cleaving the ester chain and freeing the attached AAS. This created a time-release effect. Cool, huh? As said prior, different esters have different release rates. This is how half and active-lives of many injection type drugs including AAS are created.

This is how half and active-lives of AAS are created, okay?

The Synthol and Hardcore Muscle Gear products Frank used were sterile (Again: so they claimed and I have tested both products. Yes, "the batches" I tested were clean) fatty acids. Remember that part. Fatty acids in these products had to be cleaved by the ester enzymes. So they too remained in muscular tissue for a period.

Once cleaved, they entered the blood stream and were likely metabolized as food. So site-injection Max Mixes were mostly created utilizing faster acting esters and non-ester products corresponding more closely with the period it took esterase to act upon the Protest or other space occupier.

It was not necessary, only preferable for control of periods utilizing AAS so as to avoid as many counter measures to growth as possible. Action/reaction, remember?

Maintenance periods had to be about 6 months for more permanent size increases and could overlap or be part of repeat cycles and phases we constructed.

\*It is important to remember that site enhancement techniques provide actual increased growth best for those who have increased their muscle mass to a point where fascia is constricting growth prior to employment. Joe Blow newbie to training realized the least benefits.

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*Editor's Note: Please allow me to further clarify the above. Site injections work ... you inject, that muscle will look and feel bigger and better. Pumps will even be better. So far, so good ...*

*But you will realize the MOST and BEST effects when you are more of an advanced bodybuilder who has pretty much done everything they can think of to make that specific muscle grow and nothing works any more. At that point, the "fascia " is so darn HARD, that it's just not going to give any more room to growth and hence, your muscles just have to sit there and stay the same size.*

*Now THIS is when site enhancement techniques work best. When you inject this oil continuously, the fascia has no choice but to expand and stretch out. Do this long enough with enough oil and it will stretch out and STAY stretched and BAM, now you have more free space that needs to be filled up with muscle.*

*So, now your muscle can BREATHE again and grow some more. It may not be much. Half an inch to as much as 2 inches for some lucky individuals. But hey, I'll take even a centimeter these days.*

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Almost any muscle imagined has been localized for site-injection protocols, but some were more difficult than others. Smaller muscles with shorter length and distinct bellies were easiest. Biceps, triceps, anterior and posterior or lateral delts, and calves for example. However Frank's pecs, lats and even traps were successfully localized.

Personally I believe the best permanent and natural results for any one area localized were best accomplished during a 6-month period and then discontinuance for 3-6 months. The reason was simple. It took time for vascular tissue to effectively fill the new area. When the site-injection space occupier was discontinued the oils were allowed to migrate out and the natural hardness returned to the muscle.

\*Anyone who watched the top 5-bodybuilding competitors in recent years had to have noticed things like very hard, tight, and dry competitors who had these balloon like delts and biceps with almost no definition (As well as other body parts). The rest of the musculature looked like rock while the over extended site-injection localized

muscles just looked like bags of oil. This was due to athletes using large volume site-injection protocols too close to competition.

***"Bigger is not better if it looks stupid and fake. Using arms for an example, it is possible to add 2-3" in size within a few weeks, my lad, but it looks very soft and lumpy."***

While maintenance periods were utilized and other phases were completed, vascular tissue filled the new areas. The total size gain during the 6-month period was at times more than 2-3", but once discontinued and the space occupier migrated out, it was about 1-2" of new muscle area (from the initial protocol) that remained in most cases. Even in the best chemistry environments the tissue can only grow so fast. But when was the last time a veteran bodybuilder put 1 -2" on his arms in under a year?

When not enough size was accomplished, it was simple to do it all again 3-6 months later. Since Frank did not compete again for about 2 years, the time allowed for 3 sets of sites to be localized and a 6-month clean-out period before competition. His arms were well over 21" first time back in the lights and his shoulders noted to be atleast 2" wider with much fuller pecs and lats...and deep cuts in all.

During the initial fascia stretching period of 3-4 weeks, 2-5 ml of **Max Mix** was utilized per site. Biceps for example was 2-5 ml injected 2-4 times weekly using 2-5 ml in each outer head. Then a maintenance period of 5-6 months consisting of one 2-5 ml weekly injection in each bicep was followed.

Since Frank's arms were his size/symmetry weak point, his first Max Mix needed to allow enough mixture volume to accommodate the number of sites while maintaining a pre-determined plasma threshold level. When the area being localized was larger, such as lats, the volume or amount of Max Mix needed to be greater but with the same total AAS mg as intended for the total daily plasma threshold. (More on that later) For now, I will put it this way. When each bicep was to receive 3 ml and the total AAS dosage intended was 100 mg, then the larger volume (say 10 ml divided into multiple administrations) for each lat was adjusted to also contain a total of 100 mg of AAS.



## **THE CRITERIA UTILIZED FOR CREATING A MAX MIX:**

The best stacks consisted of a good testosterone, a high androgenic, and an AAS that remains anabolic in a catabolic environment.

"A little Methandriol Dipropionate helped make receptors more sensitive.)

### **TESTOSTERONES**

Decanoate-Propionate-Undecanoate  
-Phenylpropionate-Enanthate  
Suspension

### **HIGH ANDROGENS**

Parabolan-Finajet (Trenbolones)  
DHT-Finabolan-Masteron

### **ANABOLICS**

Nandrolones (Esterless-Phenylpropionate-Decanoate-Cypionate in a pinch) Primobolan Depot-Masteron-Equipoise-Winstrol Depot (Masteron is a high androgen but has great anti-catabolic qualities and does not aromatize)

I have always liked the localized anti-catabolic effect of esterless nandrolone for site-specific enhancement.

**Note: The best results came from esterless, acetate and propionate AAS in that order.**

## **MAX MIX EXAMPLE #2:**

I have already outlined a common second favorite site-injection Max Mix in Max Androgen Phase example #3C, so let's look at a different and highly effective Max Mix Frank realized amazing progress from.

My intent was to create a high androgenic environment quickly, just as in prior Max Androgen Phase examples, then exit with a high anabolic environment. This required some creative use of esters but really was not all that difficult. Assuming 42ml used weekly (3ml x 2 injections left/right=6ml daily) Each weekly Max Mix vial contained the following: (3 weeks / 3 vials)

<b>Product</b>	<b>Provided per ml</b>
Trenbolone acetate 225 mg 3 ml	~5.29 mg
Nandrolone Decanoate 400 mg 4 ml (100 mg/ml) or Primobolan Depot	~9.52 mg
Test. Enanthate 400 mg 2 ml (200 mg/ml)	~9.52 mg
Test. Propionate 400 mg 4 ml (100 mg/ml)	~9.52 mg
Site Enhancement Oil (SEO) 29 ml	
Benzyl Alcohol 0.5 ml	

- Vial #3 did not need to contain Testosterone Enanthate
- Testosterone Propionate needed to be increased to 800 mg/8ml for vial #3 intended for days 15-21.
- Each Bicep and Tricep received 3-5 ml injections per week and lateral delts will each received 1 -3 ml injection per week. 3 vials each with 42.5 ml were mixed, allowing 6 ml per day, 21 days.

It was interesting how this stack worked out. The testosterone propionate caused a rapid initial plasma threshold while the rest ramped up over the first 2 weeks. This allowed Nandrolone Decanoate (Deca) to become dominant after the end of week #3. Testosterone propionate dropped off about day #22, and then Enanthate and Parabolan about day #28, and Nandrolone Decanoate slowly ramped back down until day #35. Then a weekly 3 ML injection of Prostate (Or other) only continued for about 6 months. Pretty simple huh?

Estrogen control was simple, too. 100mg/d of Clomid until day #28, and down regulate to 50mg/d there after unless a Cortisol/Estrogen Suppression Phase followed. In that case, no down-regulation of Clomid was noted to be necessary....but we will get to that.

At day #22, 1 mg of Arimidex or 250mg of Teslac daily was utilized as an estrogen/aromatase inhibitor when the protocol was run as a sole phase without additional layers such as a **Cortisol/Estrogen Suppression Phase**.

The 6-month Hardcore Muscle Gear SEO maintenance period also aided in supporting above normal androgen levels due to some products containing prohormones/prosteroids. (I kind of liked that) This meant better post-cycle mass retention and a superior androgen level during **Absolute Anabolic Phases, Cortisol estrogen Suppression Phases**, or simply during total off periods.

I have already described the locations and techniques Frank employed for bicep and tricep site-injections, so I won't make you read that again. (They have been described at length in "Chemical Muscle Enhancement")

### **Advanced Site-Injection Protocols**

Before we move on I would like to share a brief out-line of an Advanced Site-Injection Protocol we had employed when there was an issue of symmetry that required significant and quick alteration. This combination left the athletes quite sore days after application to a point of reduced poundages being necessary during training. However growth still occurred at a rapid pace.

The inflammation that occurs from this type of structure induces a cascade of on-site hormonal responses paramount to both repair and growth. As example we know IGF-1 synthesis and release dramatically increases locally when a stretching-type stress is applied to muscle tissue. This begins a cascade response that is synergistic to our goals by triggering an increase in PGF-2a and MGF (Mechano Growth Factor: Please see "Science Geek Stuff for further explanation). The result is super-sensitivity to androgens and other anabolic substances locally.

### **Warning: Science Geek Stuff on MGF**

Technology Summary: The cDNA of three human insulin-like growth factor I (IGF-I) splice variants have been cloned in human muscle by researchers at the Royal Free and University College Medical School, University College London. The mRNA of one of these IGF-I splice variants was found to be detectable only in exercised and/or damaged (e.g. stretched or electrically stimulated) muscle.

Its expression was found to be related to the level of muscular activity and it was subsequently named 'Mechano Growth Factor' (MGF). The biological activity of MGF has been tested both in vivo by intramuscular injection into mice of the cDNA contained in a suitable muscle expression vector, and in vitro by injection into cultured myocytes.

Intramuscular injection of MGF resulted in a **20% increase in muscle wet weight and a 25% increase in mean muscle fiber size after only two weeks**, with

subcutaneous administration resulting in no significant increase in wet weight of underlying muscle, thus strongly suggesting a rapid and profound **localized action**.

Development Status: MCF has been found to be expressed in mouse, rabbit and human muscle, with sequence studies showing MGF to have a number of domains, some of which have similar sequences to the liver systemic type of IGF-I, but one which activates muscle stem cells and another that recognizes a specific binding protein.

The binding protein has been found to be present in abundance in both skeletal and cardiac muscle, where it stabilizes MGF and localizes its action, thus reducing the risk of potential side-effects on non-target cells/tissue. MGF has not only been found to be expressed in muscle but also in other types of damaged tissue where it **up-regulates protein synthesis and induces the stem cells required for tissue regeneration**. A number of different therapeutic applications of MGF are currently being developed together with academic and commercial collaborators.

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**Editor's Note:** *Umm ... err ... did you get all that? Hehe ... okay, kinda confusing for us regular folks. I guess you 're thinking "cool... so what's the point of this MGF stuff".*

*Basically, for those who want to know MORE details about why site injections work, ALR has given you further research information and that's why there is a mention of MGF and the "science geek" stuff. Unfortunately, you can't go out and buy it at your local pharmacy ... but one day you might be able to get your hands on some. Similar to how IGF use to be a "mythical" drug and now a lot of people can get some.*

*Point of the above few paragraphs is that site injections work and one of its mechanisms or mode of action is through MGF. Got it? ...*

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Before we proceed to the next section, please turn the page and I'll give you one last Advanced Site Injection protocol ...

## Advanced Site-Injection Protocol Example 5

DAY	DRUGS
1.	(B) Testosterone Propionate 100mg/Trenbolone Acetate 75mq/Stanozolol 25mg
2.	(D) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
3.	(T) Testosterone Propionate 100mg/Trenbolone Acetate 75mg/Stanozolol 25mg
4.	(B) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
5.	(D) Testosterone Propionate 100mg/Trenbolone Acetate 75mg/Stanozolol 25mg
6.	(T) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
7.	(B) Testosterone Propionate 100mg/Trenbolone Acetate 75mq/Stanozolol 25mg
8.	(D) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
9.	(T) Testosterone Propionate 100mg/Trenbolone Acetate 75mg/Stanozolol .25mg
10.	(B) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
11.	(D) Testosterone Propionate 100mg/Trenbolone Acetate 75mg/Stanozolol 25mg
12.	(T) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
13.	(B) Testosterone Propionate 100mg/Trenbolone Acetate 75mq/Stanozolol 25mg
14.	(D) Testosterone Propionate 100mg/Methandrostenolone 50mg/Stanozolol 25mg
15.	(T) Testosterone Propionate 75mg/Trenbolone Acetate 75mg/Stanozolol 50mg
16.	(B) Testosterone Propionate 75mg/Methandrostenolone 50mq/Stanozolol 50mg
17.	(D) Testosterone Propionate 75mg/Trenbolone Acetate 75mq/Stanozolol 50mg
18.	(T) Testosterone Propionate 75mg/Methandrostenolone 50mg/Stanozolol 50mg
19.	(B) Testosterone Propionate 75mg/Trenbolone Acetate 75mg/Stanozolol 50mg
20.	(D) Testosterone Propionate 75mg/Methandrostenolone 50mg/Stanozolol 50mg
21.	(T) Testosterone Propionate 75mg/Trenbolone Acetate 75mg/Stanozolol 50mg
22.	(B) Testosterone Propionate 75mg/Methandrostenolone 50mg/Stanozolol 50mg
23.	(D) Testosterone Propionate 75mg/Trenbolone Acetate 75mg/Stanozolol 50mg
24.	(T) Testosterone Propionate 75mg/Methandrostenolone 50mg/Stanozolol 50mg
25.	(B) Testosterone Propionate 75mg/Trenbolone Acetate 75mg/Stanozolol 50mg
26.	(D) Testosterone Propionate 75mg/Methandrostenolone 50mg/Stanozolol 50mg
27.	(T) Testosterone Propionate 75mg/Trenbolone Acetate 75mg/Stanozolol 50mg
28.	(B) Testosterone Propionate 75mg/Methandrostenolone 50mq/Stanozolol 50mg

- Dosages were divided equally between two syringes then SEO was added to the 3cc mark.
- B=Bicep D= Delt T=Tricep
- Arimidex or Liquidex 1.5mg/d or Aromasin 50mg/d
- Before Frank began any AAS phase he had a PSA level test. (Prostatic Specific Antigen level test)  
PSA is a test to measure the "possibility" of prostate cancer. A high PSA level could allow activation of dormant malignancy in the presence of androgens and excessive estrogens.

## Hazardous Materials or Hardcore Muscle Gear?

When I wrote "Chemical Muscle Enhancement", I had out-lined a "topical/oral androgen posing oil" called Prefect from a company called **Hazardous Materials**. As I explained, it was a sterile MCT oil based product line that contained a series of high activity free prosteroids and prohormones.

It had become a favorite space occupier for both so-called naturals and chemically assisted athletes alike. It was kind of like "Super Synthol" in action. In fact, I know of several individuals who claimed it was as effective as testosterone propionate and Durabolin/EQ when utilized as a space Occupier / androgenic /anabolic protocol.

I do not agree that it was "that good", but results were very impressive as were resulting androgen plasma levels. It was my favorite and Frank's progress realized from Protest were significantly better than that accomplished from Synthol.

The problem was that it has become very difficult to find. "Sold Out" and "discontinued" were the answers I received with my mail and internet orders for more. I finally got a hold of the owner who explained that due to fear of FDA intervention and the products "drug-like appearance" its future was uncertain.

The out come of the conversation, was that their new product line became "orally administered" sterile oil based products containing high activity esterized prosteroids and prohormones, and a topical posing oil containing a sterile MCT combination. The line is now called **Hardcore Muscle Gear**, instead of Hazardous Materials mainly so it doesn't scare people off or give the wrong idea of what the company does.

Any way, the point is that it's still the same company, but they have even better products now. I'll list a few of their super-cool products on the next page. Just keep in mind that by the time you read this book, things may have changed with their product line. Maybe out of fear that the DEA or FDA or some other badge carrying organization considers their products too "hardcore" or borderline "drug-like", they may alter some things.

My advice would be to always visit their website for the most resent information by going to [www.HMgear.com](http://www.HMgear.com) ... Anyway, on to their legal androgen list ...

## A Few Hardcore Muscle Gear *Legal* Androgens

1. **Nandrolone-OH (AKA: "No Side Effects Deca")**: This essentially is nandrolone decanoate without the high potential for "Deca Dick" or gyno from estrogenic activity added to progestin-like qualities normally attributed to Deca. It is a sterile solution of 4-Hydroxynandrolone decanoate provided in a 100mg/ml 20ml bottle. Moderate androgenic and high anabolic structure.
2. **Testosterone-OH (Non-Aromatizing Testosterone)**: This product has nearly the same anabolic/androgenic profile as testosterone but it is not susceptible to the aromatase enzyme thus preventing aromatization to estrogens. A real plus is the lack of bloating and gyno from administration as compared to testosterone itself. Interesting fact is that it also inhibits estrogen formation and has "reported" HPTA regenerative qualities. I have used the product with excellent results and have to admit that there was an increase in HPTA activity when employed alone. Testosterone OH is a sterile solution of 4-Hydroxytestosterone decanoate provided in a 100mg/ml 20ml bottle. High anabolic and high androgenic structure.
3. **4-AD Decanoate**: A little known fact is that 4-Androstene 3, 17, diol is not a precursor of testosterone but actually a metabolite of it. It is more androgenic than testosterone and about 90% as anabolic. It does not aromatize directly to estrogens and induces low DHT activity from reduction to the metabolite. Oddly enough most users report a superior post-training recovery value to this product when compared to testosterone itself. 4-AD Decanoate is a sterile solution of 4-Hydroxytestosterone decanoate provided in a 200mg/ml 20ml bottle.
4. **1-Testosterone Decanoate (Burn Free T-Test)**: Many that have experienced 1-testosterone administration have also realized that the stuff hurts for days at the administration site. This is due to the structure of the hormone itself, actually. Though I disagree with the "Burn Free" labeling I do have to say that the effect has been reduced to an acceptable level. 1-Test is about 7-time more active than testosterone though its effects are somewhat of a cross between trenbolone and Primobolan. It does not convert to estrogens and it possesses a certain subcutaneous water reducing effect that is quite welcomed by most. This product does have a very high androgenic effect as well as significant anabolic value. Great for lean body mass gains and cutting protocols. 1-Testosterone Decanoate is a sterile solution of 1-Testosterone decanoate provided in a 50mg/ml 20ml bottle.
5. **Hardcore S.E.O.**: This is a sterile MCT oil product provided as a cosmetic enhancement. 100ml bottle.

**Important, please read:** As most are aware by now, the decanoate ester added to any hormone allows for an 8 day half-life when administered intramuscularly. Though sterile, I certainly would **not** suggest that anyone apply these products as anything but **as directed by the label and legal guidelines**. However there has been those whom have used them as Max Androgen Phases.

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*Editor's Note: Before we start with the protocols, I wanted to let you know that these*

*products by HM Gear are really awesome. One of the reasons that we mention them is because I haven't been able to take any steroids for a few years now. Actually, I never really did for ONE main reason - HAIRLOSS!*

*Pretty much allfreakin steroids make me lose hair right away, especially at effective dosages. Some people don't have the "balding" gene, so you can take whatever you want, at whatever dose and never lose your hair. I unfortunately, I'm not one of these lucky individuals.*

*One drug that I could take (in lower dosages) was Deca ... but, one problem I*

*ALWAYS had was I got the dreaded "Deca Dick", which for those that don't know, is ZERO sex drive. I had a limp dick and when you are in a relationship, this sucks.*

*Oxandrolone (Anavar) was another drug that I could take in lower dosages as well, but you don't see much of anything with it all by itself, not to mention, it's just too darn expensive. And of course, there is GH, but my body tends to need a high dosage for any drugs (whether it be steroids or Tylenol) and as we all know, high dosage GH is extremely expensive.*

*Anyway, to make a long story short, the products at HM Gear have been heaven sent for me! I mainly stack the Nandrolone-OH with the Testosterone-OH and it ROCKS! No side effects, no hairloss, no bloat, no bullshit. Just good, decent size. Yes, I do have to take more of it compared to "real" steroids to see similar effects, but I would say about 25% more. Hence, instead of doing 750 mgs a week of Deca Durabolin + Testosterone Cypionate, I now take 1000 mgs of Nandrolone-OH + Testosterone-OH.*

*Also, I usually add in about 100 mgs, twice weekly of the 4-AD Decanoate for extra sex drive and I feel and look good, hard and cut all the time without having to spend a ton of extra money on estrogen and DHT blockers, inhibitors, clomid, HCG, etc., etc.*

*I'm actually saving a lot more money now and hence, I'm very grateful and I wanted to show my appreciation by mentioning this company and their products! Hopefully for those in similar situations to mine, you will be just as happy with the results!*

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## Hardcore Muscle Max Androgen Phase Example

Below are some simple, yet very effective protocols you can use with the HM Gear products. Without the addition of any AAS, these products had been quite useful for creating several effective Max Androgen Phase utilized both as site-injection protocols and non-site-specific protocols. Frank has utilized them by following the schedule out-lined on the next few pages.

- Frank rotated sites daily in according to training.

### Hardcore Muscle Max Androgen Phase Example (Mass)

DAY	DRUGS
1.	4-AD Decanoate 200mg/Testosterone OH 100mg
2.	
3.	4-AD Decanoate 200mg/Testosterone OH 100mg
4.	
5.	4-AD Decanoate 200mg/Testosterone OH 100mg
6.	
7.	4-AD Decanoate 200mg/Testosterone OH 100mg
8.	
9.	4-AD Decanoate 200mg/Testosterone OH 100mg
10.	
11.	4-AD Decanoate 200mg/Testosterone OH 100mg
12.	
13.	4-AD Decanoate 200mg/Testosterone OH 100mg
14.	
15.	4-AD Decanoate 200mg/Testosterone OH 100mg
16.	
17.	4-AD Decanoate 200mg/Testosterone OH 100mg
18.	
19.	4-AD Decanoate 200mg/Testosterone OH 100mg
20.	
21.	4-AD Decanoate 200mg/Testosterone OH 100mg
22.	
23.	4-AD Decanoate 200mg/Testosterone OH 100mg
24.	
25.	4-AD Decanoate 200mg/Testosterone OH 100mg
26.	
27.	4-AD Decanoate 200mg/Testosterone OH 100mg
28.	

- Due to the effects of the 4-Hydroxytestosterone and 4-Hydroxynandolone upon estrogen no anti-estrogen layer was needed
- By adding Hardcore S.E.O., this protocol has been utilized as a Site-Injection Protocol quite effectively as well.

## Hardcore Muscle Max Androgen Phase Example (Cutting)

DAY	DRUGS
1.	Testosterone OH 200mg/Nandrolone OH 100mg
2.	
3.	Testosterone OH 200mg/Nandrolone OH 100mg
4.	
5.	Testosterone OH 200mg/Nandrolone OH 100mg
6.	
7.	Testosterone OH 200mg/Nandrolone OH 100mg
8.	
9.	Testosterone OH 200mg/Nandrolone OH 100mg
10.	
11.	Testosterone OH 200mg/Nandrolone OH 100mg
12.	
13.	Testosterone OH 200mg/Nandrolone OH 100mg
14.	
15.	Nandrolone OH 200mg/l -Testosterone Decanoate 50mg
16.	
17.	Nandrolone OH 200mg/l-Testosterone Decanoate 50mg
18.	
19.	Nandrolone OH 200mg/l -Testosterone Decanoate 50mg
20.	
21.	Nandrolone OH 200mg/l -Testosterone Decanoate 50mg
22.	
23.	Nandrolone OH 200mg/l-Testosterone Decanoate 50mg
24.	
25.	Nandrolone OH 200mg/l-Testosterone Decanoate 50mg
26.	
27.	Nandrolone OH 200mg/l -Testosterone Decanoate 50mg
28.	

- The combination of *Hardcore Testosterone-OH* ("Non-Aromatizing" Test) and *Hardcore Nandrolone-OH* ("No Side Effects" Deca) had a profound anti-estrogen value resulting in a decrease in body fat and an effective increase in lean mass tissue.
- High protein diets with moderate carbohydrate and fat intake significantly increased these effects ... obviously.
- The substitution of *1-Testosterone Decanoate* ("Burn Free" 1-Test) for *Testosterone-OH* resulted in a superior hardening effect.
- Those athletes whom were advanced enough doubled the listed dosages by following a daily administration schedule.

## Do You Hate Needles?

There was a rich individual who actually passed out cold at the sight of a syringe. So he utilized an all-oral Max Androgen Phase protocol that no one would consider liver "friendly". Though Primobolan Acetate tabs do not have a high liver toxicity problem (And Anavar is actually not too liver toxic), the use of Anadrol-50 and Winstrol tabs (Or Winstrol Depot orally) tended to be pretty liver toxic as the period of administration increased.

But what can be said except that he was 5'8 and 250 LBS? His estrogen control was 20 MG of Nolvadex 2 times daily on days #1-21. Then he layered in a Cortisol/Estrogen Suppression Phase beginning day # 22. He has also utilized an oral pancreatic insulin stimulator such as Glipizide 10 MG 2xd in a 2-days on/2- days off protocol beginning day #1, for the entire 28 days. I have to admit that his blood work was not as bad as one would think considering the number of times he has followed this protocol with minimal "off periods. Interesting, but scary.

### THE RICH MAN'S LIVER HATER ORAL MAX ANDROGEN PHASE (EXAMPLE)

DAY	DRUGS
1-7	AD-50 250mg/Andriol 240mg
8-14	200mg/Andriol 160mg/Winstrol 25mg
15-21	AD-50 150mg/Andriol 140mg/Winstrol 25mg/Anavar 25mg
22-28	Winstrol Oral 50mg/Anavar 25mg/Primobolan Tabs 50mg

### One Last Comment About HM Gear

I do wish to say that I include items in my books and articles from companies like Hardcore Muscle Gear simply because they work. In the case of HM Gear, I have had the fortunate experience to have worked with their products people (which is where I became fortunate enough to meet Sam...he is their marketing expert) on occasion and come away with a secure feeling that my readers and clients will receive the results they have a right to expect from anything they offer...so far.

The feedback from most of the users of the products has been extremely positive. This, coupled with the fact that my own mini-circle of test subjects has also realized great results is the reason I so openly endorse this company and their products. As stated already, I do not accept money for endorsements (though I was paid for the product designs) as I feel that would be a conflict of interests. And in truth, I am quite comfortable with the income I make without selling my soul. With that said...

## Pluggin' My Own Company T00...AGR Nutrition

Personally I am quite proud of the product projects we currently have completed and excited about several we are either researching or testing for HM. Naturally you are wondering why am I telling you all of this...

I thought that after all of these years that I have designed products for other companies, it was about time that the ones I really like are produced. I am fortunate enough to be one of the founders of a new company called Accelerated Growth & Research. One of the subdivisions is AGR Nutrition.

I do hope that you will enjoy our line as it develops and that each of you will be kind enough to give us some feed back about your results and interests. Please visit our under construction website at [www.agrnutrition.com](http://www.agrnutrition.com) and see what's new and how we can assist you in reaching your goals.

### Now, A Few of the upcoming HM Gear Products

1. **Primotren:** This is probably one of the most powerful orals there is. Ya, I know. It is somewhat of a cheesy name, but it is actually how this item is best described. The structure is a methylation product of the parent molecule 1-Testosterone. This means that in doing so the activity is increased several times over (some research suggest it is over 10 times more active once in the circulatory system, though I believe it to be a little less then 1 000%).

This is an orally active near 100% bioactive androgen similar in structure to both products that provides the hardening and strength qualities of trenbolone yet has the dry and tight effects upon musculature of Primobolan enanthate. There are no tren side-effects like "tren-dick" or progestin induced gyno (Trenbolone has progestin-like qualities and can trigger gyno and impotence when used in higher dosages or long term). Just good hard lean muscle mass gains with superior vascularity.

Primotren is soon available in bottles providing 60 10mg caps. The suggested daily use is 10mg 2xd.

2. **Methyl-4-AD:** There are some prohormones that have very high conversion rates into well known AAS. And, there are those that possess intrinsic activity that are already quite for effective anabolics unconverted. 4-AD is an example of this. As is, 4-AD is almost as effective as testosterone itself **when delivered into the circulatory system at effective dosages** (oral consumption would require about a gram ingested daily to be minimally effectual when compared to parental testosterones at low dosages due to first pass liver deactivation). And, as many are aware, 4-AD is

converted into testosterone itself by way of the 3b-hydroxysteroid dehydrogenase enzyme in the liver and peripheral tissues.

In the case of Methyl-4-AD we have a prohormone that is like super 4-AD in intrinsic activity. This means that in itself it is many times more anabolic and androgenic than 4-AD...and it converts to methyltestosterone at a very high rate in the body due to the same 3b-hydroxysteroid dehydrogenase enzyme. So a little goes a long ways. Due to methylation Methyl-4-AD resists first pass liver destruction thus being nearly 100% bioactive.

My experience has been that this is a reasonably effective oral product for mass and strength gains even used alone. However, the product also stacks well with HM Gear products like Testosterone OH for greater mass accrument, or with Nandrolone OH and/or Primotren resulting in a hardening value with good lean muscle gains. Due to its methylated structure it is always wise to also include milk thistle and rALA in the daily protocols. Methyl-4-AD is provided in bottles containing 90 10mg caps. I believe the suggested daily use is 10mg 3xd.

3. **Chiseled S.R.G.:** (Site Reducing Gel) with Yohimbine HCL, Octpamine, Formastane, and aminophylline. No joke! This is a topically applied "site-specific" fat reducing product. It works on the basis that certain areas experience fat loss better than others during cutting phases and calorie restricted periods. This is commonly due to a higher concentration of the receptors responsible for inhibiting fat loss called Alpha receptors (A2). Chiseled SGR penetrates the areas that it is applied to and saturates the localized area with substances known to increase fat expenditure and another that bocks the fat loss inhibiting A2 receptors to increase fat release. The addition of Formastane (a powerful suicide anti-aromatase) puts an end to localized estrogen formation resulting in a total anti-fat environment. Available in 4 oz bottles.

I would like to note that we use pharmaceutical quality raw materials in all of our products. This means that our puity of the active ingredients are unsurpassed anywhere in the supplemental industry and results are beyond compare.

## SHBG Control

### How To Elevate Free Testosterone Levels ... *Naturally!*

As the reader should be aware (and would be if "Chemical Muscle Enhancement" was read prior to this book), there are total, bound and free testosterone plasma levels. Bound testosterone, or any androgen/AAS, is inactive and bound by SHBG (Sex hormone binding globulin) and has no effect because it cannot merge with receptor sites. Free testosterone is the only active form and accounts for about 1-3% of total testosterone /Androgen plasma levels. So a total testosterone threshold of 100 MG would only provide 1-3 MG, approximately, of free and active testosterone.

\*Which is a lot of molecules when one considers normal endogenous total plasma levels of "about" 7 mg daily total. This would provide about 0.07 MG of free testosterone daily for a male athlete.

Brief AAS protocols were intended to get in, hit hard, and get out before the body's reaction of elevating cortisol/estrogen levels could catch up to elevated testosterone/AAS levels. This also meant that levels of cortisol failed to reach a dominant level post-cycle and greater lean mass was retained. A second benefit of brief protocols was the body's **inability** to catch up to quickly elevated plasma testosterone levels with an up-regulated SHBG synthesis (or the other sex hormone binding protein called albumin). SHBG also sometimes took a few weeks to down-regulate post-cycle, which means free testosterone levels were lower.

There are some OTC (over the counter) preventative supplements that aid in maintaining elevated free testosterone levels. Avena Sativa (wild oat extract), copper tartrate, magnesium aspartate, zinc aspartate, and vitamin-B-6 are the most common. There are products that contains Muira puama (potency wood) zinc, magnesium, and Vitamin B-6 in highly biologically active forms that have clinical studies supporting elevation of free testosterone by about 30%. There are several other products of this nature on store shelves.

## My Favorite

### Eurycoma Longifolia lack: AKA Long Jack

Tongkat Ali (Eurycoma longifolia jack) is a small tree found in the jungles throughout Malaysia and Southeast Asia. It is commonly known as Tongkat Ali in Malaysia and Singapore, Piak or Tung Saw in Thailand, and Pasak Bumi in Indonesia.

The tree can grow up to about 12 meters in height. Natives consider every part of the tree as medicine. Tongkat AM is used as a tonic, to treat malaria, and as an aphrodisiac.

There have been quite a few studies with this plant. The effects of eurycoma were studied on the libido of sexually experienced male rats after dosing them with 200, 400 and 800 mg/kg body weight twice daily for 10 days. Results showed that eurycoma produced a dose-dependent increase in mounting frequency of the treated animals. Further studies also showed that eurycoma promoted the growth of both ventral prostate and seminal vesicles. (Human studies showed the response at much lower dosages).

### **Use Them At The End**

Human studies have shown a fairly rapid decrease in SHBG and an increase in both total and free testosterone during relatively short-term use of this product. My personal experience has been that post-cycle SHBG elevation is quickly brought into a more effectual range. Naturally an interesting side effect is the fairly common increase in libido and erectile function. (Ya, got to love that one)

By beginning use of these products during the last active week of any AAS phase, Frank and other beasts realized that an increase in free testosterone was available to maintain post-cycle results and over ride cortisol and SHBG. 21-30 days use usually was enough to make a notable difference. And to be honest, it was just plain healthy.

*"Of course Insulin is an excellent SHBG controller, Frank. But health is paramount to progress. Sometimes the most benign appearing natural substances have the most profound long-term synergistic effects. A drug is not necessarily the best choice when the goal can be accomplished without potential additional negative Action/Reaction Factors to consider. Have we yet discussed 4-Hydroxy-Isoleucine, Lad?"*

*Coach*

## **CORTISOL/ESTROGEN SUPPRESSION PHASES**

Pretty much anyone who has ever used a supplement or drug for increased muscle mass has realized that post use much of the gains are lost. Naturally, in the case of AAS and other anabolics this greatly depends upon correct accounting for the actual Action/Reaction Factors relating to the chemistries employed and how each affects the body in positive or negative ways. This includes during and post use.

As you know the body maintains a state of balance called homeostasis. We both gain and lose protein-based tissue daily at a rate of bodyweight x 1.818 expressed in grams. This homeostasis is a balance between anabolism (tissue building/protein synthesis) and catabolism (tissue wasting/protein breakdown).

Several hormones and hormone-like-substances trigger catabolism. Glucagon does so by setting into motion a series of metabolic events that results in the release of fatty acids, amino acids, and glucose/glycogen from body tissue to restore blood circulatory glucose levels. However the group we are most concerned with are called corticosteroids.

This is a group of steroids that originated at the adrenal cortex. The most commonly discussed hormone of this group is cortisol. When a cortisol molecule merges with a muscle cell cortisol receptor-site, it triggers the release of amino acids from the cell. That probably does not sound like a big deal until one realizes that these amino acids come from the breaking down of the muscle cell proteins. This of course means all of that hard earned lean mass tissue begins to waste away.

Cortisol levels are elevated as a result of stress. Unfortunately, the body views stress stimuli such as increases in muscle tissue mass, training, sickness, and the spouse in a bad mood as reason to increase circulatory cortisol levels. Cortisol production can also inhibit endogenous GH and testosterone production.

The use of Androgens (AAS) of exogenous origin in excess of natural endogenous production triggers anabolism or tissue building /protein synthesis in excess of normal. This is a means of altering the balance or ratio between anabolism/catabolism in favor of anabolism.

During Max Androgen Phases (or any AAS protocol), like all other beasts, Frank's body attempted to re-establish the balance between anabolism/catabolism (homeostasis) in a variety of ways. First, after a few weeks, his body began to step-up cortisol production to trigger catabolism equal to AAS induced anabolism.

Second, through aromatization, his body attempted to create excessive estrogen levels that would induce a negative feed-back loop resulting in HPTA



suppression. The excessive unnatural estrogen levels would have also triggered female pattern fat deposits, gyno, and water retention, (which are counter-productive for the most part) Excess fat accumulation is also a major aromatase enzyme production site. (Yikes!)

Brief hard-hitting Max Androgen Phases did not increase cortisol production as much as the old longer AAS cycles. But, there was still an elevation to deal with... and if post AAS cycle cortisol levels are a little below normal...

Now, Cortisol /Estrogen Suppression Phases were a means of creating a state of "*protein sparing*". When we altered the anabolic/catabolic ratio by **decreasing** protein wasting, we realized a net gain in lean tissue mass. Let me explain.

If we gain and lose daily equal amounts of protein based tissue at a rate of bodyweight x 1.818 expressed in grams then we have the ability to increase protein based tissue mass either by addition (Anabolism) or by accumulation (Protein sparing/anti-catabolic/saving). This is like a checking account.

If we have a daily deposit of \$100 and withdrawal \$100 daily, then our account is in a state of homeostasis or "no change balance". But if we increase our daily deposits without also increasing or daily withdrawals we increase by addition (Anabolism). Now, how about if we left our daily deposits at \$100 but decreased our daily withdrawals? Yes, you are right. Our account would experience an increased balance daily due to accumulation.

In theory this would mean that a (Small) 200 lb bodybuilder could experience a net gain in lean mass tissue daily of 363.6 grams (200 lb x 1.818 = 363.6 grams) if the catabolism side of the anabolic/catabolic ratio were reduced 100%. That would result in a net gain of about 24 lbs of lean tissue in a month. Of course this is not only impossible, it would also be very unhealthy.

But... we did decrease the catabolic side of the ratio enough to not only put an end to cortisol induced post AAS cycle lean mass tissue loss, but in many beasts cases we actually augmented gains while "off-cycle".

My experience was that estrogen is an evil hormone during AAS protocol exit periods or when an athlete was attempting to maintain a reasonably lean musculature and significant muscle mass during off-periods. Most readers realize that estrogen seriously inhibits HPTA function and therefore inhibits natural/endogenous androgen synthesis in the male body (female don't have testes).

So it should be obvious to the non-cross dressing reader that elevated estrogen levels and suppressed androgen levels post-cycle resulted in far more than "shrunk nuts syndrome" and female pattern fat deposits.

"Normal or above endogenous androgen production and post-cycle lean mass tissue retention/addition (Yes, I said "addition") was dependent upon the suppression and subsequent system clearing of estrogen.

**Cortisol/Estrogen Suppression Phases** had helped to create the amazing monsters of the new era. Many of these monsters would not have been able to retain anywhere near the freak-status viewed today if not for the ability to arrest run-away catabolism and fat accumulation either naturally occurring or resulting from Action/Reaction Factors of chemical muscle enhancement.

### **Cortisol/Estrogen Suppression Phases Example #1 - Chart**

DAY	DRUGS	DAY	DRUGS
1.	Cytadren 250mg 2xd	15.	Teslac 250mg
2.	Cytadren 250mg 2xd	16.	Teslac 250mg
3.	Teslac 250mg	17.	Cytadren 250mg 2xd
4.	Teslac 250mg	18.	Cytadren 250mg 2xd
5.	Cytadren 250mg 2xd	19.	Teslac 250mg
6.	Cytadren 250mg 2xd	20.	Teslac 250mg
7.	Teslac 250mg	21.	Cytadren 250mg 2xd
8.	Teslac 250mg	22.	Cytadren 250mg 2xd
9.	Cytadren 250mg 2xd	23.	Teslac 250mg
10.	Cytadren 250mg 2xd	24.	Teslac 250mg
11.	Teslac 250mg	25.	Cytadren 250mg 2xd
12.	Teslac 250mg	26.	Cytadren 250mg 2xd
13.	Cytadren 250mg 2xd	27.	Teslac 250mg
14.	Cytadren 250mg 2xd	28.	Teslac 250mg

This protocol utilized multiple strategies. Cytadren is a biosynthesis inhibitor. This means that it inhibits endogenous synthesis of androgens, estrogens, glucocorticoids, and mineralcorticoids. So on one side the protocol (as outlined) this allowed for inhibition of estrogens and subsequent water retention inducing steroids, and on the other side allowed for the suppression of catabolic hormone production.

Unfortunately Cytadren also inhibits the production/synthesis of natural androgens (uh, like testosterone). Cytadren does this by preventing the enzymic conversion of cholesterol into the first step in all steroid biosynthesis...pregnenolone. This of course did not have any negative effect upon AAS activity (except inhibition of aromatization) nor prevented any conversion factors relating to prohormones or prosteroids.

So when this protocol was utilized as a layer beginning the 1 5<sup>th</sup> day of a Max Androgen Phase overall results improved significantly and post-cycle lean mass retention became something to brag about. Frank followed this protocol and added site-injection work using Protest fairly often.

As said prior, estrogen was a major enemy of post-cycle lean mass retention. This was only partly due to estrogen attempting dominance over declining AAS/androgenic levels. This also meant that water retention and female pattern fat accumulation as well attempting to over-ride muscle anabolism in the absence of control measures. Which of course sucked.

Cytadren inhibits estrogen production at its first biosynthesis step and prevents aromatization of most AAS. Teslac also inhibits estrogen, but is actually a relative of testosterone that has a low androgenic effect. Remember; it didn't take much to induce anabolism if catabolism was reduced. Teslac was one of the most effective anti-estrogens I had employed (rivaled only by Arimidex and Aromasin (exemestane)). This cycle was further enhanced for Frank at a later date by stacking 50-100 mg of Proviron daily. This went well with the second reason I liked Teslac.

Teslac has a profound direct influence upon the hypothalamus. This influence causes dramatic HPTA function up-regulation, which eventually leads to increased endogenous testosterone and sperm production as a positive rebound factor.

*“Look mom, post-protocol big nuts”*

*Frank*

Though this combination almost completely suppressed estrogens and catabolic hormones, there were a few side effects. Teslac had few side effects of course, though beasts had to avoid excessive calcium intake during use.

However, Cytadren did pose a few concerns. Strangely enough, long-term use has been noted to actually lead to increased stimulation of the release of catabolic hormones from the adrenal gland. The two day on - two day off protocol outlined (utilized for under 30 days) has of yet not done so. Joint soreness was common when high dosages were utilized for long periods, however.

The good new was that Cytadren can mangle cortisol receptor-sites long after discontinuance. Additionally, Teslac use has been reported to lead to permanent estrogen suppression (oh darn!). I doubt it needs to be said again, but Cytadren dosages were divided through out the day. (250-500mg each)

My problem concerning Cytadren was that it negated the body's ability to inhibit an inflammatory response. This meant a possibility of hemorrhaging from some types of injuries since it also inhibits blood clotting. (Avoiding knife fights is always a good idea) It could have also decrease our body's ability to fight infections.

However, this was unlikely and not noted at reasonable dosages and when utilized in a 2 on - 2 off administration schedule.

As stated prior, Cytadren does not inhibit 3 $\beta$ -hydroxysteroid dehydrogenase enzymes. This means that the prohormones 4-diols and 19 nor -diols and prosteroid 3-hydroxy androstanes converted to active androgens, but in truth they were already quite active as is.

As we have discussed prior, Action/Reaction Factors had to be anticipated and accounted for if the intent was maximum results with minimum negative side effects. Negative side-effects should have never been something mindlessly endured. They have also derail progress for the unknowing; significantly reducing positive results.

Cytadren (Aminoglutethimide) was utilized as a biosynthesis inhibitor by many competitive athletes as a means of inhibiting cholesterol conversion to pregnenolone, the first step in sex hormone biosynthesis. This means less or no (dosage dependant) production of glucocorticoids, mineralocorticoids, estrogens, and androgens occurred endogenously.

Sports use was intended to suppress the formation of cortisol and estrogen, thus decreasing the catabolic side of the anabolic/catabolic ratio and estrogenic activity. When layered into a Max Androgen Phase beginning day #1 5 there was still enough androgenic activity from AAS to counter-act the suppression of endogenous androgen production for about 21-28 days total, or about 7-14 days after the termination of a 30 day Max Androgen Phase.

However, when Cytadren was utilized in the absence of AAS, some form of exogenous androgen had to be utilized. Low dosages of Low-HPTA suppressing steroids such as Primobolan Depot or Anavar were Frank's options. Proviron was another option for androgenic support I had included in other beasts protocols.

Prohormones such as 4-Androstene 3,17-diol (4-AD) or prosteroids like 1-Test, 4-OH Testosterone or 4-OH Nandrolone (like those contained in some products by Hardcore Muscle Gear or Bio-Design Labs) were also useful. Remember, when the catabolic side of the ratio was reduced, even normal androgen levels were greatly effective.

However, we had negative feed-back loops to consider if Frank was to utilize the potential growth inducing properties of biosynthesis inhibitors. Prolonged high dosages of cortisol suppressing drugs such as Cytadren can induce a negative feed-back loop. My experience has been that when biosynthesis of cortisol production is nearly totally suppressed for more than 3 weeks, the body reacts by producing more ACTH (Adenocortico tropic hormone) which is released from the pituitary gland. This

in turn stimulates cortisol production/release by the adrenal glands. So Cytadren's action would be over ridden by the pituitary/adrenal glands as a reaction.

When Cytadren is prescribed for Cushings Syndrome patients, the dosage is 500-2500 mg daily. This strongly supported my belief that as little as 125 mg 2xd would have been moderately effective for bodybuilding purposes for a brief period. To counter act the possible negative feed-back loop, Cushings patients are given low dosages of hydrocortisone so some circulatory level of glucocorticoid steroids exists.

Again, this supported my belief that some form of cortisol had to exist in lower values and levels to fully utilize the benefits of Cytadren. And Cushings Syndrome victims produce far more cortisol than even the hardest training bodybuilder.

I have noted that once a negative feed-back loop begins, it slowly ramps up cortisol plasma levels until it reaches homeostasis or balance between anabolism /catabolism. In this situation, once Cytadren was discontinued, the existing elevated pituitary/adrenal produced cortisol levels were joined by normal endogenous cortisol production through biosynthesis. This obviously resulted in several steps back in the progress of the individuals whom erred in this manner.

The solution was simple: Utilizing a 2 day on - 2 day off protocol that allowed a ramping dosage of Cytadren for no more than 6 weeks (30 day maximum use was even more effective) did not allow the body's adaptive response to induce a concernable negative feed-back loop. This allowed for a very low level of circulatory glucocorticoid steroids to exist which in turn prevented triggering of adrenal hyper-function.

Another option utilized for very advanced athletes was a 30 day protocol with 2 day rotations of Cytadren and Metyrapone. Metyrapone inhibits 11-beta-hydroxylation in the adrenal cortex. (A light should go on above the reader's head now)

## Cortisol/Estrogen Suppression Phases Example #2 - Chart

DAY	DRUGS	DAY	DRUGS
1.	Trilostane 180mg/Proviron 50mg-150mg	12.	Nolvadex 30mg/Proviron 50mg-150mg
2.	Nolvadex 30mg/Proviron 50mg-150mg	13.	Trilostane 180mg/Proviron 50mg-150mg
3.	Trilostane 180mg/Proviron 50mg-150mg	14.	Nolvadex 30mg/Proviron 50mg-150mg
4.	Nolvadex 30mg/Proviron 50mg-150mg	15.	Trilostane 1 80mg/Proviron 50mg-150mg
5.	Trilostane 1 80mg/Proviron 50mg-150mg	16.	Nolvadex 30mg/Proviron 50mg-150mg
6.	Nolvadex 30mg/Proviron 50mg-150mg	17.	Trilostane 180mg/Proviron 50mg-150mg
7.	Trilostane 180mg/Proviron 50mg-150mg	18.	Nolvadex 30mg/Proviron 50mg-150mg
8.	Nolvadex 30mg/Proviron 50mg-150mg	19.	Trilostane 180mg/Proviron 50mg-150mg
9.	Trilostane 1 80mg/Proviron 50mg-150mg	20.	Nolvadex 30mg/Proviron 50mg-150mg
10.	Nolvadex 30mg/Proviron 50mg-150mg	21.	Trilostane 180mg/Proviron 50mg-150mg
11.	Trilostane 1 80mg/Proviron 50mg-150mg	22.	Nolvadex 30mg/Proviron 50mg-150mg

Trilostane inhibits 3-beta hydroxysteroid dehydrogenase delta 5-4 isomerase and was never utilized longer than 28 days (2 on/2 off) or with any related prohormone substance dependent upon 3-BHSD since it also inhibited their conversion. But the stuff did do a great job on inhibiting cortisol. Obviously, Trilostane was taken in 60 mg divided daily dosages.

Due to Trilostane's 3-beta inhibiting properties an "androgenic"-estrogen inhibitor such as Proviron or 4-OH Testosterone was utilized to elevate androgen levels daily, while 10 mg AM, 20 mg PM of Nolvadex suppressed estrogen activity with a 2 day on-2 day off protocol. When this protocol was utilized following a Max Androgen or AAS site injection phase, Frank began administration on the first day of the last active week of that phase.

By now, you are aware that this refers to the period when the AAS plasma level was ramping down. And that all Cortisol/Estrogen Suppression protocols have been successfully layered over any AAS cycle. But what about post-cycle feed-back loops? Simple: We began by the last week of activity.

## Cortisol/Estrogen Suppression Phases Example #3 - Chart

DAY	DRUGS	DAY	DRUGS
1.	Metyrapone 500 mg	15.	Metyrapone 500 mg
2.	Arimidex 1 mg	16.	Arimidex 1 mg
3.	Metyrapone 500 mg	17.	Metyrapone 500 mg
4.	Arimidex 1 mg	18.	Arimidex 1 mg
5.	Metyrapone 500 mg	19.	Metyrapone 500 mg
6.	Arimidex 1 mg	20.	Arimidex 1 mg
7.	Metyrapone 500 mg	21.	Metyrapone 500 mg
8.	Arimidex 1 mg	22.	Arimidex 1 mg
9.	Metyrapone 500 mg	23.	Metyrapone 500 mg
10.	Arimidex 1 mg	24.	Arimidex 1 mg
11.	Metyrapone 500 mg	25.	Metyrapone 500 mg
12.	Arimidex 1 mg	26.	Arimidex 1 mg
13.	Metyrapone 500 mg	27.	Metyrapone 500 mg
14.	Arimidex 1 mg	28.	Arimidex 1 mg

- Liquidex was a common replacement for Arimidex

Metyrapone (metopirone) is an inhibitor of 11-beta hydroxylation within the adrenal cortex so it was a notably good cortisol inhibitor that prevented or limited production. I preferred Cytadren, but Metyrapone worked well in a 2 day on - 2 day off protocol.

This short period of use prevented excessive production of ACTH and therefore no secondary negative feed-back loop. To put it differently, no negative reaction to the anabolic/catabolic ratio altering action.

Arimidex is fast acting as an estrogen inhibitor and possesses a long half-life (about 34 hours). So 1-1.5 mg daily worked very well when higher aromatizing AAS were in the layer, or 1 mg was sufficient if lower dosage or low aromatizing AAS were utilized. Obviously 0.5 mg of Arimidex was more than enough during non-AAS periods.

HCG has been successfully layered in utilizing 1000-3000 iu every 4 th day to boost endogenous androgen production. Additional estrogen control and HPTA up-regulation has been realized with the inclusion of Clomid: 100mg/d day 1-5 and 50mg/d day 6-15. (Arimidex has been documented to increase HPTA activity in males in the absence of AAS use)

It should be obvious that creatine use aided in post AAS cycle mass retention, but I thought I would point this out again. When, for some reason, 1 mg of Arimidex failed to create acceptable results for Frank (due to other metabolic factors), up to 2 mg daily had been utilized. Many have simply included the addition of 20 mg of Nolvadex before bed. 1.0 mg of Arimidex stopped most any degree of ramped estrogen production however in the absence of exogenous AAS use.

\*Recently there was a black market product called Liquidex that provided 1-2 mg of anastrozol (Arimidex) per ml of liquid. It came in a 20-100 ml vial. It was not an injection product but when used orally it provided excellent results. The lab claims a 10% over dose on each batch and thus far this was true. It was also WAY cheaper than Arimidex tabs.

### **Cortisol/Estrogen Suppression Phases - With HPTA Layer Example #4 - Chart**

DAY	DRUGS
1.	Remeron 7.5mg/Aromasin 25mg
2.	Remeron 7.5mg/Aromasin 25mg
3.	Remeron 15mg/Aromasin 25mg
4.	Remeron 15mg/Aromasin 25mg
5.	Remeron 15mg/Aromasin 25mg
6.	Remeron 15mg/Aromasin 25mg
7.	Remeron 15mg/Aromasin 25mg/HCC 1000iu
8.	Remeron 15mg/Aromasin 25mg
9.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
10.	Remeron 15mg/Aromasin 25mg
11.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
12.	Remeron 15mg/Aromasin 25mg
13.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
14.	Remeron 15mg/Aromasin 25mg
15.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
16.	Remeron 15mg/Aromasin 25mg
17.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
18.	Remeron 15mg/Aromasin 25mg
19.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
20.	Remeron 15mg/Aromasin 25mg
21.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
22.	Remeron 15mg/Aromasin 25mg
23.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
24.	Remeron 15mg/Aromasin 25mg
25.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
26.	Remeron 15mg/Aromasin 25mg
27.	Remeron 15mg/Aromasin 25mg/HCG 1000iu
28.	Remeron 15mg/Aromasin 25mg



**Remeron** (mirtazapine) tablets are prescribed as an antidepressant. Before thoughts of Frank on Prozac spin in your head realize that this is a section on cortisol and estrogen control.

Mirtazapine (Remeron) lowers cortisol and prolactin in normal subjects (like athletes are ever normal?) This appeared to an effectual alternative to Cytadren for those interested in lowering cortisol levels. It also lowers prolactin resulting in a decrease in fatty tissue and HPTA inhibition.

Additionally there was a mild (low dose) antidepressant effect as well that some welcomed post-cycle. Kind of like low dose Prozac, Cytadren and bromocriptine or cabergoline all in one. It is noteworthy that many have reported improved appetite during Remeron use, but several have also reported sleeping 12 hours the first few days of use even with the stepped initial dosages.

In studies 15mg/d resulted in a significant decrease in cortisol. What is really interesting is the method of inhibition. ATCH is a hormone that tells the adrenal glands to release corticoid steroids. Naturally most are catabolic to protein tissue (like muscle). However the suppression of ATCH leads to less cortisol being produced without inhibition of sex steroid synthesis.

This means a significant increase potential for LH/FSH production and the resulting increase in natural androgen production. Yup, that is right. Remeron inhibits ATCH secretion. Of course the anti-prolactin effect is nice in the bedroom as well.

**Endocrinological effects of mirtazapine in healthy volunteers.**  
**Prog Neuropsychopharmacol Biol Psychiatry 2002**  
**Dec;26(7-8):1253-61**

## OUR FRIEND ESTROGEN ...AND ITS EVIL COUSINS

So far I have made a sincere effort to avoid techno-geek stuff for the most part. However, it seems paramount at this point to include some that was not included in "Chemical Muscle Enhancement". Frank gets pretty bored during techno-geek discussions. I, on the other hand, am amazed at the human body.

Estrogen is not a single function, single form or structure hormone. Most bodybuilders assume estrogen is the stuff that gives women great racks and weird (but fun) mood swings. Or they view estrogen as only an evil hormone that makes males bloated, soft, and gyno prone. Did you know a males woody would be a **wood-have-been** without estrogen? How about estrogens IGF-1 stimulating and glucose transport mediating properties explained earlier?

The term "estrogen" is a catch-all term utilized to describe a group of biologically active hormones. Just as most doctors and the media have grouped all AAS into one single term, so have most abused the word estrogen.

*"So, Frank. You wonder at the power of propaganda? You have a point. As to most doctors and the media grouping all AAS into one group, try this on for size: "Arguments must therefore be crude, clear and forcible, and appeal to emotions and instincts, not the intellect. Truth was unimportant and entirely subordinate to tactics and psychology." Sound familiar? The quote was from Joseph Goebbels and his thoughts on WW II. Created perception is all that matters to most. A few think for themselves before they act. In that lies success"*

*Coach*

*\*Think about that and read on.*

The amounts and ratios vary or differ for men and women, but the body produces several different estrogens. 4-Hydroxyestrone, 2a-Hydroxyestrone, 16a-Hydroxyestrone, estrone, estriol, and 17b estradiol for example. Each has different metabolic pathways for production and each has a different level of estrogenic activity. This means that the body has a purpose for each estrogen and some are stronger than others.

1 7b-estradiol is the most powerful estrogen and has strong estrogenic effects upon tissue. 1 7b-estradiol can convert or metabolize into estrone, which in turn can convert into 2a-hydroxyestrone, and/or 16a-hydroxyestrone. This conversion process is mediated by enzymes. In fact, in some cases, the enzymes the body uses to convert androgens (Such as androstenedione and Androstenediol) into stronger hormones (Such as testosterone) can also effect estrogen production. Obviously the body needs a balance of all hormones and hormone-like substances. This of course means that alterations made through chemical muscle enhancements can have a non-synergistic effect if not properly structured.

Several studies have shown that the ratio of 2a-Hydroxyestrone to 16a-Hydroxyestrone is a deciding factor in the development of estrogen related tissue and cancer. For this reason the medical community is utilizing Nolvadex, Arimidex, Aromasin, Faslodex and others as a treatment for, and prevention of, breast cancer.

In rare cases, male gynecomastia has developed into full-blown breast cancer so readers be aware. Research strongly suggests that a higher level of 2a-Hydroxyestrone and lower level of 16a-Hydroxyestrone is beneficial for prevention.

Estrogen control protocols have been described in depth in "Chemical Muscle Enhancement" and in the present text. Most of these examples utilized drugs that were prescription only in many countries.

However there are a few studies that support the use of OTC products to effectively control estrogen to a respectable degree. Di-indolyl-methane (DIM), Indol-3-carbinol (1-3-C), lycopene, and ground flax seed (And flax seed oil) have studies supporting their beneficial effects upon decreasing bad estrogens while increasing the good.

DIM and 1-3-C both potentially aid in estrogen deactivation and evacuation. They do so by inhibiting the conversion enzyme responsible for 16a-Hydroxyestrone formation. This then forces the body to utilize the C-2 pathway and increase the production of 2a-Hydroxyestrone. The body then excretes the inactive metabolite of 2a-Hydroxyestrone rather quickly and effectively. This results in less total circulatory estrogens as well.

The good side of AAS aromatization for Frank was the fact that estrogen passing through the liver increases the production and release of IGF-1. Estrogen also positively affects the hypothalamus-pituitary-axis only in that it triggers the release of GH.

As you know by now, IGF-1 triggers the up-regulation of satellite-cell production, and the newly formed cells (In the presence of elevated androgen levels) possess a higher number of androgen receptor-sites (Meaning that the new cells potentially have a higher capacity to respond to AAS)

Since IGF-1 also increases the inclusion of satellite-cells into both existing and newly formed muscle fibers, growth potential is further enhanced. Theoretically, by aiding in a better 2a-Hydroxyestrone - to - 16a-Hydroxyestrone ratio, and the subsequent conversion to and excretion of 2-Methoxyestrone at a faster rate, more IGF-1 would be produced. So the use of DIM and 1-3-C before, during, and after AAS cycles did have interesting benefits. I believe this may have been true for both sexes.

Personal experience has shown that the inclusion of DIM, 1-3-C, and flax seed powder or oil is surprisingly effective during high estrogen level periods for any athlete. In addition their inclusion during phases such as Absolute Anabolic Phases has had a certain synergistic effect.

500mg/d DIM, 500mg/d 1-3-C, and up to 3 tablespoons of flax seed oil per day has been effective for several athletes employing low dosage AAS protocols.

## **Prolactin...Another Evil Cousin**

When a woman's body prepares to give birth it begins to produce a hormone called prolactin. Its job is to trigger an increase in breast and glandular tissue to produce milk for the coming baby's sustenance. Men produce prolactin as well!

Prolactin is a single-chain protein hormone that is closely related to growth hormone (GH). It is secreted by so-called *lactotrophs* in the anterior pituitary gland. It should be noted however that it is also synthesized and secreted by a broad range of other cells in the body, most prominently various immune cells, the brain and the decidua of the pregnant uterus.

## **Natural Prolactin Control**

In opposition to what we normally see with all of the other pituitary hormones, the hypothalamus predominantly suppresses prolactin release from the pituitary gland. In other words, there is usually a hypothalamic "Stop that" order set against the lactotroph, and prolactin is released only when the order is released.

A note of interest is that if the pituitary stalk is severed, prolactin release increases, while secretion of all the other pituitary hormones decreases dramatically due to loss of hypothalamic releasing hormones. But this is an unlikely scenario for most athletes and should obviously be avoided nonetheless.

The neurotransmitter Dopamine appears to act as the top dog prolactin-inhibiting factor. Dopamine is secreted into portal blood by the hypothalamic neurons. Next it binds to receptors on lactotrophs, and inhibits both the synthesis and release of prolactin. So chemicals and drugs that interfere with dopamine release or receptor binding also increase the release of prolactin. These are called antagonists. Drugs and chemicals that either increase, act as, or potentiate dopamine are agonists.

Of course there are other chemicals in the body's Action/Reaction Factor closet that positively regulate prolactin. The major ones are GnRH, TRH (thyroid Releasing Hormone) and VIP (Vasoactive Intestinal Polypeptide). By the way, hyper-stimulation of the nipples may have a stimulatory effect upon prolactin release as well. But that is one we will leave alone.

## **So Why Do Non-Cross Dressing Men Produce Prolactin?**

As a man ages his body begins to decrease the amount of androgens that it synthesizes. In fact many studies have shown that an average 40-year-old male produces about half of the testosterone that he did when he was 18. So he possesses a lower rate of muscle anabolism yet a higher rate of fat anabolism.

Many researches have claimed that this is due to normal physiological changes that occur as we progress through the years. In truth this is bullshit and supposition based upon average sedentary individuals.

I monitor the physiological indicators of athletes for a living. I can say conclusively that almost any otherwise healthy male that remains in peak condition and eats a proper diet will retain a superior androgen production profile. So this is more so a matter of choice than pre-programmed physiological events. With that said let's get on with the why of prolactin.

Estrogen is a primary promoter of prolactin release. Of course there are other factors to consider (which we will discuss in a moment) that may trigger excessive prolactin secretion, but the normal trend toward increased prolactin release is due to increased estrogen synthesis.

## **More Action/Reaction (*For More Action*)**

The clinical term for excessive release of prolactin is *hyperprolactinemia*. It is actually a relatively common disorder in humans. There are many causes that initiate the condition including prolactin-secreting tumors and therapy with certain drugs.

Interesting is that the use of progestin-like AAS such as nandrolone and trenbolone will usually result in an increase in prolactin secretion. The employment of drugs such as cabergoline (0.25-0.5mg 2xW) bromocriptine (2.5-5.0mg/d) or mirtazapine for 2-4 weeks post use normally brings prolactin levels back to normal or below. This results in increased HPTA function, improved determent from accumulative female pattern fat and significantly heightened libido.

Males that experience hyperprolactinemia commonly develop hypogonadism (the shut down of the HPTA) with decreased sperm production, decreased sex drive and impotence. Those affected normally show breast enlargement (gynecomastia), but very rarely actually lactate. The gyno can initially manifest itself as an increase in fatty tissue under the lower pectorals and a puffy appearance to the areola and nipple.

A simple blood test for serum prolactin levels is commonly employed to evaluate the degree of potential feminization a male can or is experiencing. The lab results are quite simple to read, though a trained professional should interpret the results.

Normal Levels:

- A. Adult: <20 ng/ml (including males)
- B. Newborn: 100 to 300 (falls below 20 after 6 weeks)
- C. Pregnancy
  - 1. First Trimester: <80 ng/ml
  - 2. Second trimester: <160 ng/ml
  - 3. Third Trimester: <400 ng/ml

\*References: Bakerman (1984) ABCs of Lab Data, p. 342

## **BIOSYNTHESIS INHIBITORS and THYROID FUNCTION**

Another Action/Reaction factor that was considered during protocols utilizing biosynthesis inhibitors was possible inadequate thyroid hormone levels/activity. In some cases (Usually due to prolonged high dosage use) biosynthesis inhibitors have decreased thyroid hormone levels due to another negative feed-back loop.

As the reader knows already, or will soon learn, inadequate T-4/T-3 levels will severely decrease muscle protein synthesis due to poor nutrient turn-over and a sluggish metabolism.

Again the solution was simple. Frank used protocols such as Cortisol/Estrogen Suppression Phases for 30 days or under, and followed a 2 day on-2 day off schedule for those that included these drugs.

The one time his post-cycle thyroid function was low we utilized Guggulsterones and another TSH stimulator listed in "Chemical Muscle Enhancement", to normalize thyroid function. This was not necessary in most cases when phases were cycled as explained later in this discussion of Frank's story.

## LOW DOSE AAS PHASE?

The idea behind a low dose AAS Phase was to maintain or even add some high quality muscle tissue, allow vascular and nerve tissue growth to catch up with the mega growth from serious weight/mass gain cycles (and site injection protocols) while not inhibiting HPTA function significantly.

This meant using high anabolics such as Primobolan Depot, 4-OH Testosterone, 4-OH Nandrolone, Equipoise, Oxandrolone and/or Nandrolone in an off set androgen alternating protocol with HPTA function chemistry. Total AAS dosages equaled about 1 mg per pound of bodyweight per week, and always pivoted upon very clean chemistry. This is often considered bridging by many.

After reviewing several blood test results, I had come to the realization that when done properly, low dose AAS phases could run 4-12 weeks with little decrease in HPTA function and few variances in liver enzyme profiles. To be effective, these phases included Action/Reaction time periods.

Simply stated, the body began to adapt and react to most chemistry or stimuli in 2-3 weeks. So each androgen /HPTA function period could not exceed 2 weeks.

Most high anabolic injectable AAS did not cause significant decreases in HPTA function and were very liver friendly. Nandrolone and Boldenone did cause *some* HPTA suppression even at 1 mg per pound of body weight weekly when utilized for more than 2 weeks consecutively. So mixing or alternating the three AAS was possible and was employed dependant upon the beast.

Few orals, except Primobolan tabs, could have been utilized since all others were cl7alkylated structures that could negatively affect liver function. Oxandrolone is a cl7alkylated AAS oral but at reasonable dosages failed to induce significant liver enzyme alterations.

Of course, just Primobolan Depot, oxandrolone or Equipoise had been utilized on numerous occasions. Fast acting Testosterone suspension was a useful androgen choice (if at all) since it cleared the system in a few days. This approach kept Frank easily within the 2 week alternating time frame.

## LOW DOSE AAS PHASE EXAMPLE (250 LB Man)

DAY	DRUGS	DAY	DRUGS
1.	200 mg. Primo D/50 mg. Test. Sus.	23.	200 mg. Primo D/50 mg. Test. Sus.
2.		24.	
3.		25.	
4.		26.	
5.		27.	
6.		28.	
7.		29.	
8.	200 mg. Primo D/50 mg. Test. Sus.	30.	200 mg. Primo D/50 mg. Test. Sus.
9.	HCC 500iu	31.	HCG 500iu
10.	HCG 500iu	32.	HCC 500iu
11.	HCG 500iu	33.	HCG 500iu
12.	HCC 500iu	34.	HCG 500iu
13.	HCG 500iu	35.	HCC 500iu
14.	HCC 500iu	36.	HCG 500iu
15.	HCG 500iu	37.	HCC 500iu
16.	HCG 500iu	38.	HCG 500iu
17.	HCC 500iu	39.	HCG 500iu
18.	HCG 500iu	40.	HCG 500iu
19.	HCG 500iu	41.	HCG 500iu
20.	HCC 500iu	42.	HCC 500iu
21.	HCG 500iu	43.	HCG 500iu
22.	HCC 500iu	44.	HCG 500iu

- Frank continued in same manner or mixed AAS from this point on for up to 12 weeks. Cycle /Phase always ended with an HPTA function period.
- Low dose AAS phases had also been utilized as part of a site injection protocol by creating a dosage equivalent Max Mix for maintenance periods.
- The addition of Clomid 50-1 00 mg/d during the last 1 4 days of a low dose protocol was very beneficial.
- Bromocriptine 2.5mg/d on listed HCG days resulted in an increase in HPTA activity due to inhibition of prolactin. (And an increase in libido is always nice)



## ORAL AAS ARE NOT ALL BAD?

*"It is often that in the building of perfect beasts that the issues of health and longevity become a primary concern. Oddly enough many, not of my design, opt to introduce health concerning factors at a phenomenal rate as a result of misinformation or critics. Misinformation in itself appears to be a commodity at times. Perhaps this is why I am criticized by some for my distinct lack of long term oral anabolic/androgenic steroids utilized in cycle outline discussions. For the critics I say: "Like I care what you think."*

*Coach*

Oral AAS did possess a few distinct advantages and did have some risk-to-effect value when utilized with responsible common sense. As you know from the first "Chemical Muscle", most oral steroids were usually noted to be very liver toxic due to their c17-alkylated structures. (Primobolan Acetate tabs was not c17-alkylated) Remember; If a beast had screwed up their liver, serious bodybuilding (and health) would have been pretty much history.

The good effects of orals were elevated liver release of IGF-1 due to a liver detoxification (and deactivation) Action/Reaction, no extra injections, and convenience. When used for brief periods of 3-6 weeks liver enzyme values usually returned to normal within a 4-8 week period after discontinuance. But for the wiser athlete limited use was best. Let's briefly look at some common oral AAS and the possibilities we had employed, again. Each has been successfully layered into various phases.

**ANADROL-50 (Oxymetholone)** Anadrol was probably the all time favorite mass and strength building oral AAS. At dosages of 100-250 mg/d, many realized weight gains of 15-25 lbs...n only a few weeks. Users raved about unbelievable strength and recovery as well. Unfortunately, the majority of weight gain was water retention and to a much lesser extent increased red blood cell count. Post-cycle lean mass retention sucked unless a high anabolic AAS such as Nandrolone, Equipoise, Winstrol-Depot, or Primobolan Depot were stacked with it, or in my experience more wisely utilized in a Max Androgen Phase. (And Anadrol was discontinued at least 10-14 days before the high anabolic of course)

Anadrol was commonly used the last few weeks pre-contest also. This was done to increase training intensity, post work-out recovery, increase vascularity, and induce a distinct fullness/hardening effect. When used pre-contest, Anadrol required the use of Nolvadex or other estrogen receptor antagonist.

Even with the use of a finasteride-type drug, Anadrol tended to cause balding for some. It also gave many athletes raging gyno in the upper dosage ranges. Anadrol does not actually aromatize but does possess progesterone like qualities.

Progesterone is an estrogen that causes gyno as well as serious water retention. Faslodex (fulvestrant) has been the better estrogen antagonist during administration of any AAS with progestin activity. Why, as I said prior, the drug works by down-regulating estrogen and progesterone receptor counts. Less receptors means less activity.

There was a debate as to whether or not Anadrol did, or could convert to, a DHT or DHT derivative. I now feel both sides are unlikely true or right. Anadrol was such a potent androgen that it attached itself to sex tissue specific androgen receptors in the scalp and prostatic areas. Finasteride did seem to possess some receptor-site blocking qualities and therefore was helpful.

Interestingly, Anadrol did not induce strong activation of androgen receptor-sites in muscle tissue. But it did cause impressive muscle mass gains. Ironic, but obviously Anadrol worked.

Sadly, Anadrol was seriously liver toxic. Only Methyltestosterone rivaled it for toxicity and Halotestin beat it for liver destruction.

Due to serious water retention during Anadrol use pre-contest, a diuretic would be necessary the last 2-3 days pre-contest. Remember Anadrol causes serious HPTA suppression.

**ANAVAR (Oxandrolone)** Anavar was actually a very potent AAS. I can almost hear you laughing, but milligram for milligram, Anavar caused a greater anabolic response than Anadrol. Oddly enough, even though Anavar was a c17-alkylated structure and elevated liver values, it did not seem to aggravate liver problems. Anavar did not convert to DHT, nor did it aromatize to estrogen.

Pre-contest, Anavar significantly increased hardness and aided in lean mass retention during calorie-restricted periods. During use, some strength gains were realized but weight gain was slow even with a higher calorie intake. However, the weight was pure lean muscle and was mostly retained post-cycle even without HPTA stimulation. This was due to Anavar's distinct lack of HPTA suppression qualities and low androgenic value.

An interesting use for Anavar and other non-HPTA suppressing AAS was lean mass retention after a heavy AAS cycle. This was very effective while HPTA stimulators kicked in endogenous androgen production and catabolic hormone levels normalized.

Elevated ATP levels significantly aided in post-cycle lean mass retention. And guess what? Anavar increased creatine phosphate synthesis. I know of several fitness competitors who stacked 25 mg of Anavar daily with 20-30 mg of Nolvadex and either 80-120 mcg of Clenbuterol and/or an ephedrine /caffeine stack, daily. They got lean, hard, and often didn't need thyroid drugs if a good diet plan was followed.

Several hard-core male bodybuilders commonly used 40-80 mg/d of Anavar during the last 3-4 weeks of contest prep to increase hardness and preserve lean body mass. Like I said, "interesting". The down side was expense. It should seem apparent that Anavar has been successfully utilized by Frank N. Steroid as a replacement for non-HPTA suppressing AAS during Low Dose AAS Phases. The dosage equaled 0.25 mg per lb/d.

There was a black market version of Oxandrolone circulating that allowed for injection type administration. The problem was that not all AAS are effective to a great degree when the liver deactivation factor is removed to a significant degree...and this was one.

**DIANABOL (Methandrostenolone)** Dianabol has had a long history in sports of all types, like since the '50's, in fact. In most cases, 50 mg/d of Dianabol caused the same results as 1 50-200 mg/d of Anadrol-50 and was somewhat easier on the liver.

Usually a dosage of 5mg/25 lb of bodyweight daily promoted excellent strength and mass weight gains for any hard-core bodybuilder. Personally, I just did not believe a dosage above this was more effective or had risk-to-effect value. (If the Dianabol utilized was real and dosage per tablet/liquid in gel caps was accurate.)

I felt Dianabol was excellent at aiding in a faster androgen plasma level during the first 7-10 days of a Max Androgen Phase. And most reported a sensation of feeling "Great" during employment.

Dianabol did aromatize significantly, and did possess some DHT receptor stimulatory qualities. In this case, Finasteride did allow for some receptor antagonist value and was fairly effective for inhibition of DHT formation.

Aromatization was usually handled with most aromatization inhibitors such as Cyclophenil, Arimidex, Proviron, or Teslac. However, a greater benefit was realized with Nolvadex. This was due to Nolvadex being an estrogen antagonist. Since antagonist only "block" estrogen receptor-sites, the elevated circulatory estrogen levels caused IGF-1 production increases. Which was cool if there was a sufficient androgen level, of course!

However, when Dianabol was layered into an AAS cycle or utilized alone for more than 21 days, an aromatase inhibitor needed to be added at least 7 days before discontinuance of all AAS. If not, post-cycle estrogen levels became a problem.

It should be realized that Dianabol was most potent in its effects as an oral, not injection form AAS. This was due to remarkable IGF-1 release from the liver during detoxification of its molecule, which was of course a c17-alkylated structure. Unfortunately, Dianabol also had serious HPTA suppressing effects.

Since Dianabol caused high water retention, it was not normally utilized as a contest prep drug. For mass cycles with good post-cycle lean mass retention, Dianabol was commonly stacked with, or in the case of my beast, phased with a high anabolic drug such as Nandrolone, Equipoise, or Primobolan Depot. These high anabolic drugs had a respectable affinity for androgen receptor-sites and solidified Dianabol induced mass into high quality lean muscle tissue...when the protocol was properly structured.

A plus for Dianabol was its distinct cortisol suppression qualities. In fact, 50-70% of cortisol's effects were blocked at a dosage of 5mg/25 lb daily. About 10 mg daily was noted in available literature as equal in activity to an average male's endogenous androgen production.

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***Editor's Note:** At the time of this writing, the research team has discovered an oral "pro-Dianabol" compound that HM Gears should be making available within the next few months. It works EXACTLY like Dianabol, but you'll just need to take more milligram wise. Cool part is that it'll be fairly cheap, about \$40 or so for a month's supply (which is the equivalent of a 15-20 mg daily dosage of REAL dianabol!. We are finalizing the patent on this compound and trying to get a good price for our first initial batch run (first runs always end up costing more due to lack of volume, setup costs, etc.). By the time this book is published and you are reading this, it will hopefully be available for sale at [www, hmgear. Com](http://www.hmgear.com)*

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**HALOTESTIN (Fluoxymesterone)** Personally, I hated the method of use most employed with this drug. Though its high androgenic characteristic increased hardness pre-contest, and fueled serious training intensity, it ate livers for breakfast.

Halotestin did little at the androgen receptor-sites in muscle tissue. However, it did do serious activation at steroid receptor sites in the brain in comparison. The result was hard-core intensity and a bad attitude problem for most athletes who were susceptible.

As I mentioned prior, I did not think of the normal 8-12 week progressive dosage administration schedule as "best utilization". Later I will explain the approach my beast used to decrease liver stress. I have noted that Halotestin 10mg 2xd is a common prescribed androgen therapy as I write this story.

Halotestin is a DHT derivative. Blood test results confirmed a significant elevation in DHT during administration. But stock in Proscar probably went up! Gyno was not a problem with Halotestin due to a lack of aromatization. In fact, some individuals have reduced their existing gyno due to the use of this drug. It also did not cause water retention, another pre-contest plus.

Halotestin was not a great or mass gain drug, definitely not in the car with Anadrol or Dianabol. But some results in lean mass were realized and strength

maintenance during calorie restriction was a plus. I could not find a main stay use for it except possibly during the last 3-4 weeks of contest prep... and then, only as a last choice of a high androgen.

**METHYLTESTOSTERONE** Don't look for any good words for this liver killer, gyno causing, headache inducing drug from me. Funny irony to think about. Last year while researching a possible pro hormone structure, I discovered a way to introduce 4-Androstenediol into the system in a way that induced GI track conversion to Methyltestosterone and absorption at a very high rate. (And it was legal)

**WINSTROL Orals (Stanozolol)** I believe milligram for milligram Winstrol was the most effective oral. I have written and said it several times; when compared to Anadrol, Dianabol, or Anavar, Winstrol kicked butt. Unfortunately it was an alkyl 17 oral.

Winstrol was not a "great" strength drug but lean muscle tissue growth was far superior. Winstrol seriously stimulated androgen receptors and aided in hardness to some extent due to low water retention. Obviously, Winstrol increased protein synthesis on two levels. First as an anabolic steroid and second as an oral IGF-1 stimulator.

Winstrol did not convert to DHT or aromatize to estrogen. However, it did cause some hair loss. Since it has been noted to attach easily to scalp androgen receptor sites this was not a major surprise. It was much milder on the liver than Dianabol or Anadrol. And certainly was more liver friendly than Halotestin. But then, so is an ice pick.

Another plus for Winstrol was its structural similarity to progesterone. This means it was noted to bind with progesterone receptors and yet inhibit progesterone's effects. This was obviously like Nolvadex in that it blocked receptor sites. For this reason it has been successfully been utilized as part of an oral stack (When orals became necessary) with either Dianabol or Anadrol. As a mass cycle the protocol required using 25-50 mg/d of Winstrol and 5-10 mg/50 lb bodyweight daily of Dianabol or 100-200 mg/d of Anadrol. 50-150 mg/d of Anadrol for pre-contest phases during the last 3-4 weeks was fairly effective as well.

Winstrol orals or injectable stacked well with Anavar for brief periods following longer AAS protocols and during initial HPTA restimulation. Winstrol had little effect upon HPTA function. So this had some real benefit in aiding post-cycle lean mass retention. Winstrol was regularly stacked with most other AAS as a synergistic component of any protocol by a few successful beasts.

Oral Winstrol tabs and Winstrol Depot /V were actually identical. Orals were quite expensive at 25 mg/d or even 50 mg/d. As I explained in the first "Chemical

Muscle", simply orally ingesting the injectable version (1/2 m = 25 mg) was far more economical. (Duh!)

## **ABSOLUTE ANABOLIC PHASES**

As we have seen so far, Frank was able to significantly increase his lean muscle mass as a result of chemically induced alterations in the Anabolic/Catabolic Ratio.

We know the body both gains and loses protein based tissue equal to bodyweight x 1.818 expressed in grams daily.

We have discussed two methods my beasts had utilized for altering either side of the equation in favor of increasing lean muscle mass. After a recap, we will look at a third option employed by Frank and other beast and then put it all together for maximum results.

**Max Androgen Phases** significantly and rapidly increased strength, bodyweight, and lean muscle mass. They did so through an increase in anabolism or increased protein synthesis. While it is true that all AAS possess some anti-catabolic qualities, their action is predominantly the result of an alteration in the Anabolic/Catabolic ratio in favor of anabolism. This is similar to a checking account.

If we made daily deposits equal to withdrawals, we would not realize a change in balance. However, if we made daily deposits in excess of withdrawals, we would realize an increase due to addition. Unfortunately the body catches on to this alteration in normal homeostasis (balance) and begins to react with catabolic counter-measures after 2-3 weeks. As you know, these counter-measures come in the form of hormones such as cortisol, glucagon, and estrogen.

Due to a ramp type increase in cortisol, a maintained AAS threshold would no longer induce significant anabolism beyond elevating catabolism after about 6 weeks. This in turn results in a near equal anabolic/catabolic ratio. If the AAS threshold /level were decreased or discontinued, the elevated cortisol levels would overwhelm even well above normal endogenous androgen induced anabolism. Which of course would result in excessive post-cycle lean muscle mass loss. The use of AAS also resulted in another negative feed-back loop.....

### **Estrogen Levels Elevate During AAS Protocols For 2 Reasons:**

- (1) Almost all AAS aromatized to some extent. This reaction was obviously due to the aromatase enzyme that converts AAS into estrogen.
- (2) The body elevated estrogen biosynthesis in an attempt to re-establish the body's natural testosterone/estrogen ratio.

In both cases, the excess estrogen caused a negative feed-back loop that told the hypothalamus to shut down endogenous androgen production. If the estrogen levels remained elevated post-cycle the inhibition of the HPTA remained significant and the athlete's body developed female pattern fat deposits. It also significantly reduced post cycle-lean mass retention.

But we know one way Frank turned these negative feed-back loops into another growth phase. Right? Frank would have been foolish to administer any AAS protocol continuously anyway. If health concerns were not reason enough to utilize brief Max Androgen Phases, long-term growth potential certainly was. Frank became a beast by the motto "Get in, grow hard, and get out before the body counter attacks."

**Cortisol/Estrogen Suppression Phases** increased total lean muscle mass by altering the anabolic/catabolic ratio in favor of protein sparing. They were also a method of destruction for both elevated cortisol production/levels and HPTA negative feed-back loops induced by elevated estrogen levels. The checking account analogy applied here in reverse. If we decreased the total amount of daily withdrawals, but still maintained daily deposits, we would realize an increase in total holdings.

Therefore, Cortisol/Estrogen Suppression Phases greatly increased post-cycle lean mass retention or gains. They have also been utilized as a stand-alone means of increasing total lean mass to some degree. When layered into a Max Androgen Phase during the last 7-10 (or day #15) days of activity, Cortisol/Estrogen Suppression Phases destroyed post-cycle blues quite well. (Gee, Ya Think?) They also turned "off periods" into growth periods. Obviously, they had value when utilized during the first 7-15 days of a Max Androgen Phase as well. But there was a better protocol...

**Absolute Anabolic Phases** were constructed without the use of AAS. Though in some cases they may have been a layer of **Low Dose AAS Phases**. Since **Low Dose AAS Phases** did not significantly inhibit HPTA function or cause excessive cortisol and estrogen production this was certainly quite beneficial. The functions of Absolute Anabolic Phases were:

1. Induced growth through an alteration in the Anabolic/Catabolic ratio in favor of protein synthesis.
2. Induced an increase in lean muscle mass by inhibiting cortisol and glucagon production without negatively affecting HPTA function or estrogen levels. This means they also altered the Anabolic/Catabolic ratio in favor of protein sparing.

Absolute Anabolic Phases were another method of utilizing "AAS off periods" for quality growth. It should also seem evident that they were additionally utilized as a means of destroying cortisol induced lean muscle mass loss post AAS cycle. But what if I had constructed a protocol in which Frank was to begin an Absolute Anabolic Phase at an appropriate time that would allow its last 7-15 days to layer over a Max Androgen Phase initial 7-15 days? It would have delayed his body's catabolic attack an additional 7-15 days while significantly increasing the results realized from the Max Androgen Phase.

Now, what if I had layered a Cortisol/Estrogen Suppression Phase into his Max Androgen Phase beginning the last 7-15 days of its activity? Yup, we would have only allowed Frank's body 14 days at most to mount a counter attack against new growth. Remember; it took the body 2-3 weeks to begin adequate counter measures? This means Frank would have been in a near continuous state of growth while following a 28-30 day on/28-30 day off protocol for actual AAS use. It was simply far more logical and productive.

As a point of fact, ponder this. We know the human body both gains and loses protein based tissue equal to bodyweight x 1.818 expressed in grams daily. So a (little) 200 lb bodybuilder both gains and loses about 363.6 grams of protein based tissue daily ( $200 \text{ lb} \times 1.818 = 363.6 \text{ g}$ ). If through whatever means we altered the Anabolic /Catabolic ratio by increasing anabolism 50% and decreasing catabolism 50 % our small 200 lb bodybuilder would gain almost a pound in lean tissue mass daily.

Oh ya, and by the way, this near pound of growth daily would be without training. Strange, but true. Of course this would require adequate calories and obviously major protein intake. Before anyone checks into couch potato status, our 200 lb bodybuilder would get quite fat, as well as sissy weak.

***"The fat could be mediated with appropriate chemistry, but sissy status can only be over come with hard-core training, you weenie".***

## FIRST A BRIEF RECAP

As with any chemical muscle enhancement drugs, those out-lined in this section had Action/Reaction effects that absolutely had to be considered. All drugs and chemicals create an adaptive response within the body.

Some are growth inducing by altering the ratio of anabolism/catabolism in favor of anabolism or retention, while others have secondary reactions which will instantly put a halt to progress. Some can seriously hamper long term potential. As example, Insulin has been noted to make an athlete huge, fat, or dead.



## INSULIN

Insulin is **the** most anabolic hormone, yet it is anti-catabolic as well. Since this is a storage hormone, anabolism is initiated by inducing storage intracellularly of glucose, amino acids, and fats, as well as electrolytes. Remember, creatine is an amino acid structure as are several growth inducing chemicals. As well, there is a great deal of evidence that by creating a state of cellular hydration, supraphysiological insulin induces a secondary anabolism and cellular hypertrophy.

Insulin has anti-catabolic qualities simply because the presence of Insulin in the blood stream at high enough levels prevents catabolic hormones such as cortisol and glucagon from becoming elevated. This obviously creates a state in which amino acids and glucose cannot exit cells.

As noted in the treatment of type II diabetics, insulin receptor-sites can be desensitized from over use of exogenous Insulin. Ever notice how fat diabetic couch potatoes get? This is because the lack of exercise allows muscle cell insulin receptor-sites to become insulin resistant and the fat cells become the main point of storage. Which of course sucks! For this reason, (and others) long protocols with exogenous Insulin could have greatly reduced long term potential.

There were some supplements employed that enhanced Insulin sensitivity and allowed for lower dosages to be more effective. This in turn helped to prevent Insulin resistance. Alfa Lipoic Acid, Chromium, Corosolic Acid, D-Pinitol, Selenium, 4-Hydroxyisoleucine, L-Phenyl Alanine, and Courdin which is a fraction from bitter melon (*Momridica Charantia*).

These supplements made a huge difference in results both during and post-Insulin use. Frank always included a mixture of 200 mg Lipoic Acid, 1000 mg L-Phenyl Alanine, and 50 mg of D-Pinitol twice daily with Insulin, and twice daily for 14 days after with 4-hydroxyisoleucine added at a dosage of 200mg 2xd.

Over the years, I have noted many athletes obtained the same results utilizing "up to" half of their normal exogenous Insulin dosages with this supplement schedule. They also stored less fat.

Another addition said to lower fat storage was HCA (Hydroxycitric Acid) which is supposed to decrease conversion of carbohydrates to fat by inhibiting its conversion enzyme. 1000 mg 2-4 times daily. My personal experience has been that HCA is **not** effective.

The essential fatty acid supplement CLA was probably the best OTC product for fat synthesis inhibition during exogenous insulin use. (We are currently patenting a fat that tells the body to not store fat and to burn fat at a higher rate. This is like super CLA x 10)

Misuse of Insulin and the normally required 10 g of carbs or appropriate super substrate per i.u. has been known to lead to coma, beta cell burn out, pancreatic damage, and blindness among other things such as cataracts.

Insulin significantly and quickly reduces blood sugar and can create a state of hypoglycemia. You will need to know that later. (There will be a test) This means Insulin is a major storage hormone, right?! So it was absolutely vital for Frank or any of the beasts to protect against Insulin insensitivity. GH release was inhibited by elevated blood sugar by the way. Think about it.

The examples that follow often list Humalog or Humulin-R insulin because they are fast/short acting and easier to control. Going to sleep with supraphysiological Insulin levels could have killed. So by utilizing a fast acting Insulin, the system was mostly clear within 1.5-3 or 6-8 hours respectively after injection.

Obviously a meal before sleep was a must. For Frank, or any beast, a single injection of Humulin-R that exceeds 1 i.u. per 10 lbs. of body weight would have significantly increased risks. This was simply because metabolic processes would not have been able to keep up with necessary glucose supplies in most cases.

## **THINGS TO THINK ABOUT...**

I realize I have pointed this out several times, but Action/Reaction principles applied for greatest growth potential rely on synergy. This meant not only strategic use of drugs that interacted to induce a more powerful stimuli, but also anticipating and responding to the negative feed-back loops drugs can produce.

In the applications I had utilized with Frank and other beasts, this required atleast a basic understanding of drug Action/Reaction Factors as well as drug interaction. Using Insulin and GH as an example, let's look at some factors taken into consideration.

## **GH and Insulin Action/Reaction Factors**

1. Insulin is hypoglycemic - GH is hyperglycemic.
2. Insulin is fat storing - GH is fat releasing.
3. Insulin promotes cellular uptake of about half of the amino acids needed for repair and growth - GH promotes the uptake of the other half.
4. Insulin increases T-4 conversion to T-3 - GH decreases liver conversion of T-4 to T-3.

## Specific Intent Considerations

GH use without an androgen or Insulin would have been anti-catabolic yet not as anabolic. GH use alone would not have increased muscle fiber contractile proteins significantly. This is due to GH lacking the ability to induce uptake of "all" essential amino acids and therefore most growth occurs in structural proteins.

Since GH can also decrease T-3 levels, protein synthesis decreases and growth would have occurred due to a stronger alteration in the catabolic side of the Anabolic/Catabolic ratio. We added insulin and growth occurred in both contractile and structural proteins due to greater *complete amino acid profile* **and** *ratio* availability and adequate T-3 levels that allowed elevated protein synthesis. This is greatly simplified but an adequate explanation of Action/Reaction Synergy.

Another considered and included point of synergy between Insulin and GH was this: Insulin and IGF-1 each potentially stimulate the other's receptor sites when plasma levels are supraphysiological. GH converts to IGF-1 in the liver, and stimulates the release of IGF-1 in muscle cells. Stacking GH and Insulin obviously resulted in a great deal of IGF-1 receptor-site activity for beasts. Interesting huh?

There are several forms and origins of exogenous Insulin used to treat diabetics. The options were numerous but experience had taught me that the fast acting (short acting) insulin's had benefits and a greater control safety (no, Insulin is not "safe" by any means without proper medical guidance) over long acting Insulin. Just as different AAS we had employed had different half and active-lives, so did Insulin's. And just like AAS, longer acting Insulin's took a longer period to become active.

The use of insulin with an active period of more than 6-8 hours without stacking it with GH was not the best approach in most situations. First, due to long periods of possible and dangerous hypoglycemic activity, and second due to a decrease in endogenous GH release in the presence of elevated insulin levels, the benefits did not out-weigh the loss. Remember synergy and Action/Reaction?

I have noted that athletes who utilized very-short or short acting insulin were about the same weight as those who favored long-acting insulin's. However, those who used the short-acting insulin carried greater lean mass and less fat in comparison. But it seemed to be equal regardless of insulin type administered when 1-2iu of GH was layered in 2-5 times daily during insulin protocols.

## EXAMPLES OF INSULIN

Name of Insulin	Start Activity	Highest Activity	Ends Activity	Low Blood Sugar
Very short-acting (Humalog)	10 Minutes	1.5 Hours	3 Hours	2-4 Hours
Short-acting (Regular/-R)	20 minutes	3-4 hours	8 hours	3-7 hours
Intermediate acting (Nor L)	1.5-2 hours	4-15 hours	22-24 hours	6-13 hours
Long-acting (Ultra Lente)	4 hours	10-24 hours	12-28 hours	12-28 hours
Combination: 70%N/30% R	0-1 hour	3-13 hours	12-20 hours	3-12 hours
Combination: 50%N/50% R	0-1 hour	3-12 hours	12-20 hours	3-12 hours

- Humalog was administered about 15 minutes before an appropriate meal
- Regular Type-R was administered 30 minutes before an appropriate meal

It would seem obvious that when Frank had utilized a protocol that required 30iu of insulin per day with 2iu of GH 3-5xd he could have theoretically administered 30iu of Combination: 70% N/30% R subcutaneously upon waking in the morning (About 10 minutes before a meal), if a meal had been ingested just prior to retiring the night before. But there is a problem with that. Go re-read GH and Insulin Action/Reaction Factors.

This would have been applicable only during a mass phase. This is due to the fact that insulin stops the fat burning effect of GH. So the better choice was GH first and a fast acting Insulin 30-60 minutes later.

**NOTE:** A fragment of the GH molecule stimulates the B-3 receptors on fat cells. This then triggers lipolysis (fat burning) while simultaneously blocking fat storage.

Some have stated that they believe an elevation in insulin during GH activity would result in a decrease in anabolic value. This is interesting in that both GH and insulin must be present in the liver and available to tissues for the synthesis of IGF-1. As most are aware IGF-1 is by far a superior anabolic on a dose dependent basis and all three are necessary for the natural synergy need for maximum growth.

## Growth Hormone (GH)

Alone GH was not as anabolic as some individuals may think. However, the positive effects of GH went well beyond any term or period of use. Let me explain that before we continue.

Muscle protein synthesis (anabolism) is triggered as a result of muscular contractions (Lifting weights) and subsequent up-take or absorption of amino acids and glucose (for glycogen synthesis) by muscle cells.

Muscle proteins are separated into two broad categories: Contractile (Myofibrillar) and structural (Sarcoplasmic) muscle proteins. Obviously contractile proteins are the working proteins and the structural proteins are the load-bearing proteins.

"Initially" the utilization of GH (In the absence of elevated androgen levels) has been noted to result in larger but not "immediately" stronger muscles. This was GH acting in an anabolic manner to increase predominantly (but not solely) structural muscle proteins far more than contractile muscle proteins. Therefore initial protein synthesis induced by GH administration occurs mostly within structural muscle proteins. This is not to say that the muscle was not structurally stronger... it simply failed to result in an increase in actual bigger weights *mowed...initially*.

Why am I repeating myself? Tendons, ligaments, muscle fibers, and other soft tissues such as cartilage contain structural proteins, too. Can you say injury repair and more muscle tissue to make bigger and stronger? (Much bigger and with exceptional post-cycle retention)

The reader should recall that supraphysiological levels of androgens resulted in an increase in protein synthesis within both categories of muscle proteins, but most significantly growth occurred within the contractile muscle proteins. At rest, structural protein synthesis was about 25% greater than contractile proteins.

However, this equation reversed due to training stimuli favoring contractile muscle protein synthesis. Both muscle proteins had to increase in mass to alter the so-called genetic limitations for growth in favor of freak status.

NOTE: Another point for the reader to remember as this tale continues is that IGF-1 had noted similar effects upon muscle proteins.

GH is converted into IGF-1 (Insulin-like Growth Factor-1 and other growth factors) within the liver and muscle cells in the presence of insulin. It should be noted

that some cells, such as muscle cells, can synthesize IGF-1 as a result of certain stimulus such as stretching and lactic acid build-up.

However, GH also contributes to the synthesis of other growth factors such as Fibril Growth Factor, Nerve Growth Factor, Vascular Growth Factor, and MGF (Mechano Growth Factor). Each of these factors contributes to the growth of vascular tissue. Obviously there was a necessary synergy required to this biochemistry that maximizes potential freakiness. And knowledge of these Action/Reaction Factors was needed if optimum long-term progress was the intent. And to think that Frank thought GH simply reduced body fat stores by forcing fatty acid release. (Geez!)

## **GH and Synergy**

During their heavy growth years, children experience periodical GH release surges. These surges, or pulses, can be as impressive as 2i.u. pulses 7 times daily for about 5 days. Note that this is not to say that the pulses always run for 5 days consecutively. (Ever buy clothes for a child twice in the same summer?) These growth surges are mediated and/or prolonged by other hormones and hormone-like substances. Take a look at any healthy growing child and tell me that Mother Nature doesn't know a thing or two about effective synergistic cycling intended for growth!

An average adult pituitary gland releases about 0.5-1.5 i.u. of GH daily and, of course, declines as we get older. (The lion's share of GH is released during the first 4 hours of sleep.) The reader should be aware of the fact that it is not that the adult pituitary can not release more GH daily without any endocrinology stress, it is that the HPA (Hypothalamus-Pituitary-Axis) sees no reason to do so.

This was explained so that the point of fact that 2-4 i.u. of GH administered daily by a first time user has induced respectable results can be fully appreciated. Of course an advanced athlete would not realize significant progress, as a rule, until daily GH dosages reached 6 i.u. or more. In fact I know of several that administer 9 i.u., or more, daily for months before realizing obvious results.

I do have to point out that there is a great deal of available Black Market GH such as Serostim (by Serono) that has been relabeled as a higher dosage than the vials actually contain. As example there are many who offer 1 2 i.u. vials that are labeled 1 8 i.u. This in itself may suggest that poor results some athlete's have experienced.

However, this did not correlate with Mother Nature's plan. Androgens (AAS), estrogens, thyroid hormones, insulin, Interleukins, prostaglandins, and other bio-substances play a vital role in natural GH growth induced synergy. For this reason different protocols were utilized to produce Frank's desired results. All of which contributed to long-term growth potential.

An example of synergy is when GH was stacked with insulin. GH is anabolic, anticatabolic, fat burning and hyperglycemic. Insulin is very anabolic (To fat cells, also) anti-catabolic and hypoglycemic. For beasts this synergy resulted in a total anabolic and nutrient storage period with fat synthesis inhibitive qualities.

When T-3 thyroid hormone was added, with or without Insulin, anabolism was significantly increased due to thyroid hormone action upon both macronutrient turn over/assimilation and due to increased protein turn over rate (PTOR). When we added to this metabolic rate increases from T-3 use, and increased fatty acid (from fat stores) use as energy (due to GH activity), and increased muscularity resulted. Remember; GH suppressed natural T-3 and Insulin release!

I wrote extensively on the effects of GH upon hyperplasia in ***Chemical Muscle Enhancement***, but briefly let me say that more type-I fibers increased in growth than type-II from GH use. However, heavy androgens "converted" type-I fibers into type-II strength/size fibers over a period of time. This is another example of long-term synergy that resulted from GH use.

## Counter-Parts and Activity

Okay, every hormone has its counter part that protects homeostasis. Testosterone has sex hormone binding globulin (SHBG) which binds and deactivates it, IGF-1 (for a period) and GH have somatostatin. All have cortisol to deal with, of course.

Somatostatin is a hormone the body utilizes to shut down GH and IGF-1 production and receptor activity in an attempt to maintain homeostasis or the anabolic/catabolic ratio. Short burst cycles utilizing GH did not significantly elevate somatostatin levels. However, somatostatin was easily reduced by simple use of L-Arginine and its analogues.

**A point of interest:** GH and most other hormones are amino acid sequences. Since Insulin acts to increase cellular absorption and use of amino acids (and other nutrients) it is probable that Insulin also increases the induction of GH into cells.

***"Synergy Baby! Without it nothing is maximally progressive in a desired direction. It's like sex without foreplay. You may satisfy an initial desire, but some factor for continued progress will be left unmet. And you may not get a second chance to do it right if you fail the first time"***

Coach

## IGF-1 (Insulin-Like Growth Factor-1)

rIGF-1 is not majorly anabolic on its own. As a by-product of GH however, it did play a very potent role in the growth long-term equation. The main problems with exogenous rIGF-I were first, its instability. Loud noises, shaking, dropping, and other life realities (like the spouse in a bad mood) will destroy it. This is why the body makes its own IGF-1 at the last moment from GH. The stuff is fragile!

Second is the fact that the intestinal areas and organs have more IGF-1 receptor-sites than muscle cells do. This is the reason so many of today's larger pro's have distended abdominal areas. Which absolutely sucked (Ya, corny but someone had to suffer the pun)!

### Which IGF-1?

However, IGF-1 was quite useful for Frank when the right form was employed (Yes, I am going to talk about Long R-3 IGF-1). And hey, someone else was footing Frank's bill in the end anyway! Remember IGF-1 is an amino acid sequence. When stacked together, the combination of IGF-1 and GH is documented to be at least twice as anabolic as either alone.

I do not believe either worked "best" for growth without exogenous insulin. But IGF-1 was better than GH alone when dosages were above 60mcg daily. Since there have been numerous studies performed with children employing as much as 9mg (9000mcg) of IGF-1 daily, 60-1 20 mcg per day was not a major issue in my opinion.

Long R-3 IGF-1 is a technological wonder. This is similar to DES (1-3) IGF-1 in that it is at least 10 times as anabolic as rIGF-I and it is resistant to liver destruction. As I have explained in ***Chemical Muscle Enhancement***, most IGF-1 exists in a bound or non-active form within our bodies regardless of origin.

It is bound by a protein called IGF-1 BP-3 (Insulin-Like Growth Factor-1 Binding Protein-3). When it is freed much of the IGF-1 is destroyed before it can even tickle one cell into growth. This means much of an administered dosage of rIGF-I is lost before it can have any fun, as well.

Long R-3 IGF-1 is resistant to binding by IGF-1 BP-3 yet it is self protective of liver destruction. Additionally the use of this anabolic actually decreases the body's production of the binding protein. Oh ya, though some still tell their lady's otherwise, Long R-3 IGF-1 is not as instable by a long shot as rIGF-1. (I had a client whom once



actually told his wife "**Hush! The IGF-1 is sleeping in the refrigerator**" and managed to get her to speak and walk quietly in the kitchen for several days.)

## Thyroid Hormones

No matter how strong the anabolic signal is that a muscle cell receives, it cannot grow unless nutrients are available at the site when the message is sent. The rate at which nutrients become available depends on diet, and metabolism. Even if an athlete ingests enough protein, carbs, and fats, it is possible, due to digestive capabilities and metabolic rate, that not enough nutrients will be available at the cell for significant increases in growth.

Thyroid hormones control most of the metabolic rate and therefore the assimilation of protein, carbs, and fats (which greatly influence protein synthesis). Thus the protein turn over rate (PTOR) is influenced as well. GH can decrease thyroid hormone activity by inhibiting the conversion of T-4 thyroid hormone into the more active T-3 hormone, so muscle protein synthesis and growth slows down.

A note of interest is the fact that many athletes have shown low levels of phenylalanine, tyrosine (which are amino acids) and iodine when tested. The body uses an enzyme called phenylalanine-hydroxylase to convert phenylalanine into tyrosine. Some athletes suffer from a disease called phenylketonuria (PKU) which is a lack of this conversion enzyme. This leads to excess unconverted phenylalanine, and therefore insufficient available tyrosine for the production of such things as catecholamine neurotransmitters from L-dope to adrenaline. Another problem is that PKU prevents tyrosine from reaching the thyroid gland. Who cares? Well, tyrosine is the amino acid part of thyroxin (T-4) and iodine is the co-factor which allows it to be manufactured.

Low tyrosine, phenylalanine, and/or iodine means low thyroxin T-4 production. This means...low conversion of T-4 to T-3 and slow metabolism, growth decreases or halts, protein synthesis is a joke, and athletes get fat. It also means there is inadequate nutrients at the muscle cell when the anabolic message arrives.

Often supplementing with 1000 mg (1 gram) each of Tyrosine and phenylalanine 3 times daily (In addition to a high protein diet) solves the problem of decreased metabolic rate. Amino acid tests such as a QUAAST can find the shortage. PKU sufferers should not take supplemental phenylalanine. Three grams total daily of tyrosine would be a better choice for PKU sufferers.

To grow, Frank had to **eat and absorb** massive amounts of nutrients so it would be at the cell and able to get in when the anabolic message arrived. This eating/absorbing factor had to keep up with the anabolic growth factor. Getting the idea? Of course you are.

So now we know that thyroid hormones were actually quite anabolic. However, the difference between anabolic and catabolic was adequate supplies of nutrients and training stimuli.

Thyroid hormone levels decide metabolic rate to a great extent and therefore dictated post workout recovery time. The higher the metabolic rate, the quicker recovery... the more calories were necessary. This was the most frequent deciding factor or choice for a chemically assisted beast such as Frank.

**Simply stated:** chemically *created* *ectomorph*, endomorph or mesomorph.

## **MGF (Mechano Growth Factor)**

Many studies support my belief that IGF-1, whether of endogenous or exogenous origin, was the primary **synergy** hormone responsible for muscular growth and repair. I have had several clients who had below average endogenous testosterone production and average or above IGF-1 levels.

Yet most experienced gains near equal to other natural bodybuilders. I believe this was due to the synergy between IGF-1 and another hormone called Mechano Growth Factor, or MGF for short.

MGF is a hormone locally produced in muscles in response to both stretching and over-load training, especially due to heavy over-load training. (Heavy Weights) This is true only when there is an atmosphere of extensive muscle fiber damage from high weight/high traumatic sets. The more severe the heavy stimuli, the more MGF the muscle fibers produce. This is quite "similar" to localized endogenous IGF-1 production and stimuli.

Recent studies used engineered genes that increased the expression of MGF in mice muscle. The researchers injected the genes and found a 20% muscle mass increase in only 14 days. WOW! Picture a 250 LB bodybuilder achieving 300 LBS in only 14 days! MGF is far more potent than IGF-1 or/and GH.

Currently, MGF research is geared toward treating various muscle diseases and weakened musculature occurring due to the lack of exercise and age. Researchers have found humans who continue to train as they age, may be able to maintain optimum MGF production.

However, some surgery or disabled patients cannot. For this reason, researchers are experimenting with MGF and IGF-1 and/ or GH for muscle wasting prevention. For now, watch out for big mice.

I think it is safe to theorize that MGF will be one of the next genetic expanding chemistries for most sports. I have noted two genetic research companies are

presently manufacturing MGF for research use. Soon it will show up on the black market, I think.

By the way, researchers have also already created a testing protocol to detect MCF use. So much for some of the lying so-called natural competitors being too happy. I am not saying all naturals are liars, just the majority of the top competitive ones. Ya, I said that and know it to be true.

Novice athletes who reported utilizing chemical muscle enhancement during research for Chemical Muscle Enhancement had no need for Absolute Anabolic Phases. And in truth, many intermediate athletes did not either.

In my opinion the goal should have been to utilize steps to realize maximum potential, not limit possibilities. (More on that in a moment) Using Frank's creation as an example (at different levels of development) the following rough guide-lines were followed for dosage adjustments. Remember; calorie intake had to exceed calorie expenditure for growth augmentation... and thyroid hormones increased metabolic rate.

### **INTERMEDIATE**

Insulin: 5-7 i.u. 2xd (Humalog only)  
Growth Hormone: 2 i.u. 2-3 xd.  
'Thyroid T-4: 100-200 mcg/d.

### **ADVANCED**

Insulin: 20-30 i.u. total daily. (Prefer Humalog or Humulin-R)  
Growth hormone: 2 i.u. 3-4 xd.  
Insulin-Like-Growth-Factor-I (Long R-3): 20 mcg 2-4 xd.  
Prostaglandin as PGF-2: 2 mg 2-4 xd. \*Thyroid T-4: 200-300 mcg/d (or T-3). 'Thyroid T-3: 50-125 mcg/d.

### **VERY ADVANCED**

Insulin: 24-40 i.u. total daily. (Prefer Humalog or Humulin-R)  
Growth Hormone: 2 i.u. 4-5 xd.  
Insulin-Like-Growth-Factor-I Long R-3): 20-40 mcg 3-5 xd.  
Prostaglandin as PGF-2: 2 mg 3-5 xd.  
Thyroid T-4: 350 mcg maximum (or 200 mcg T-4 with 1 00-1 25 mcg T-3/d)

\*During contest prep, T-3 and / or T-4 dosages were slightly higher for brief periods.

We will discuss Frank's DNP (2,4-dinitrophenol) use later. However, there was a natural occurring substance that had reported similar effects (supposedly) called Usnic acid. It was suppose to increase metabolic rate 100-200 %, but I have not had

"great" success with it or the green tea that contained a similar substance. I have not given up on it yet as it does have potential.

## T-2

We will also discuss how and why some beasts used triacana later as well due to its thyroid hormone elevating effects. But for now I would like to point out that there was another natural occurring thyroid-hormone-degradation-compound called T-2 (diiodothyronine).

T-2 acted as an uncoupler of oxidative phosphorylation by effecting cytochrome-c oxidizers and cytochrome-c reducers. This in turn increased metabolic rate and the PTOR. Interesting that research suggests that T-2 increases brown fat and subsequently fat expenditure as heat.

My experience with this substance had been favorable in that the result was greater lean mass and less fat. It appears that in action the chemical increased protein synthesis in a greater respective ratio to protein degradation which resulted in a sort of anti-catabolic effect superior to that experience with T-3 or T-4.

This was likely due to the cAMP up-ramping effect fostered by T-2. The dosage adjustments were also more easily accomplished. I had also experienced profound results when I had employed DNP and T-2 during a cutting phase. Except for the usual "everything I wore had yellow pit stains" from the body's excretion process of DNP, the results were without side effect, (short-term)

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*Editor's Note: Personally, I think a combination of ALL 3 thyroid hormones, in lower dosages (T-2, T-3 and T-4) work better for retaining muscle and burning fat then taking "high" dosage of just one thyroid (i.e. most bodybuilders just take a ton of cytomel, T-3).*

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## A Note On DNP Dangers

It should be noted that I am aware of one individual who suffered severe side-effects while employing multiple DNP protocols back-to-back. He began personal experimentation at a bodyweight of over 260LBS. Once he discontinued self-administration of the substance his bodyweight continued to decline even after the initiation of 6iu of GH 3xd.

He finally sought medical intervention that resulted in a group of the AMA's finest scratching their heads and no answers as to why. Eventually the lad stopped losing body mass and leveled out at 170-plus pounds. Though I was never asked, it

seems likely that the problem was extreme cyto-toxicity. After all, DNP is essentially a bug spray.

## Absolute Anabolic Phases Example #1 - Chart

DAY	DRUGS
1-7	Lutalyse 2mq-3xd / Humalog 7iu-2xd
8-14	Lutalyse 2mg-4xd / Humalog 7iu-3xd
15-21	Lutalyse 2mg-5xd / Humalog 7iu-4xd

- In earlier stages of his development when Frank had less prior insulin use he had cut dosages down to 4-6 iu per injection.
- Lutalyse (PGF-2a) and Humalog were utilized by Frank, site-specific and mixed together in a syringe.
- This example was created and utilized for a very advanced athlete. At daily insulin dosages of above 20-25 i.u. total, T-4 or T-3 inclusion was necessary to allow adequate macro-nutrient turn over rate. 1 50 mcg T-4, or 50mcg T-3 was commonly enough.
- Insulin injections before a work-out, regardless of calorie intake, would have resulted in hypoglycemia and serious health threat. Since Humalog was outlined in this example, it should be noted that Frank needed to ingest food at least 1 5 minutes before an injection.

## Absolute Anabolic Phases Example #1 - Details

Lytalase is a prostaglandin PGF-2a and, in my opinion, really cool stuff. Where as insulin was the most non-specific anabolic, PGF-2 was the most site-specific anabolic hormone-like chemical Frank used for chemical muscle enhancement. PGF-2 was best utilized site-specific, meaning that it was most effective when injected directly into a muscle for localized growth. (Gee, you think so?)

In short it was perfect for bringing up lagging body parts and contest prep symmetry adjustments. There is a black market preparation that is a form of PGF-2a that is more powerful and in an oil suspension. This allows it to remain at the administration site for a prolonged period exerting its anabolic effects.

There was another reason prostaglandins had to be utilized site-specific. PGF-2 had a brief active-life that reduced effective response periods for **total body** stimulation... though some did migrate to the vascular system and trigger anabolism through out the body. And this was where problems arose.

Every muscle in the body responds to PCF-2, including smooth muscle tissue such as intestines, and stomach. An injection too near these areas would have resulted in serious cramping. As you know, cramping is a form of contraction. This means that in some cases concerns of "**projectile waste**" was possible.

Reports showed that in some individuals, flu like symptoms occurred during prostaglandin use. Women mostly were wise enough to avoid PGF-2 use. (Though a few had utilized the drug effectively)

The localized growth induced by site injection PGF-2 triggered highly effective protein synthesis. The few beast who had utilized PGF-2 properly, in respect to the intent of muscular augmentation, added 1 -2 " to their arms in a 3 week period. Some Beasts had to rotated injection sites on a schedule dictated by training for multiple body-part stimulation.

Some did so simply because PGF-2 injections left the target muscle quite sore for a couple of days and the pain hindered intense training. This was not sore like a testosterone suspension injection site. It was far more painful. Kind of like the pain resulting from 15-20 sets of negative-only straight bar curls.

For this reason, Frank did not inject PGF-2 into a target muscle until after it was trained that day. This required Frank to either train first thing in the morning, or to localize sites trained the prior day until after the new day's work-out was complete. Of course multi-sites spread out over multiple days resulted in less growth per part, but an athlete needs to be able to train. Did I mention the soreness some experience?

Another interesting effect of PGF-2 was fat loss. This was fully discussed in the first "Chemical Muscle Enhancement". But to briefly touch on this phenomenon... PGF-2 triggered a chain of metabolic Action/ Reaction Factors that is in part dependant upon fatty acids. Fatty acids were rapidly released from adipose tissue (fat cells) to fuel these actions. COOL!

*"There will always be individuals who claim something is not true due to what someone else has said. Those who say PGF-2 does not induce dramatic effects simply have not used it or failed to procure the real deal."*

*Coach*

PGF-2 protocols dramatically increased androgen receptor-site count and site sensitivity. This effect was so dramatic in some beasts that even the elevation in endogenous testosterone production created from HCG use caused muscular pumps so intense that benching an empty bar was enough to bring tears to (some) eyes. These individuals would have been obviously unwise to have utilized AAS with PGF-2. However, "most" advanced athletes did not react this intensely.

Since PGF-2 had this effect upon androgen receptor-sites, protocols utilizing it was perfect for leading into a Max Androgen Phase or other AAS protocol. This was due to multiple reasons.

First, even lower plasma thresholds of AAS brought significantly better results. Second, the synergistic factors realized when the last 7-15 days of PGF-2/insulin use overlapped (layered over) the first 7-15 days of a Max Androgen Phase were quite impressive. There were other reasons this was so effective, but I will wait until the end of this chapter for better understanding.

Frank utilized the properties of insulin to act synergistically with PGF-2. Since PGF-2 was notably protein synthesis magic, a great deal of amino acids (from protein) had to be available at the muscle site when anabolism was triggered. Inadequate availability of nutrients would have produced poor results.

To utilize PGF-2 without assuring building materials would have been like telling the wife and her sister a threesome would rock: Message sent and received, but it is highly unlikely anything good would result. Only very bad things, including a huge waste of money on divorce in most cases. Of course, as I have said countless times prior, some have responded positively to almost any stimulus. We are talking muscle chemistry of course.

Supraphysiological levels of insulin facilitated the metabolization, transport, and storage of supraphysiological levels of macronutrients. Obviously, only because massive amounts of protein, carbohydrates, and fats were first ingested and absorbed.

Unfortunately exogenous insulin use alone will trigger anabolism in all tissues including fat stores. However, when insulin was stacked with a specific-tissue anabolic stimulator such as PGF-2, amazing muscular growth occurred. And in this case, fat stores were reduced in proportion to total calorie needs (metabolic) and ingestion.

Frank always ingested at least 10 grams of carbohydrates or glucose substrates per I.U. of insulin injected. He also avoided overlapping injections of insulin since, due to active-life, it would have induced an up-ramping effect of insulin plasma levels. (Bad idea!) He never went to sleep during a insulin high-activity period and always had a meal before sleeping.

Using days listed as examples, Frank's timing sequence usually followed this schedule:

**Day#**

**#1-7:** 7am, 2 pm, 9 pm (trained at 1 pm or between 5-9 pm). Slept at 11 pm.

**#8-14:** 7am, 11:30am, 4pm, 9pm (trained at 10 am or between 7-9 pm). Slept at 11 pm.

**#15-21:** 7am, 10 am, 1pm, 6 pm, 9pm (trained at 5 pm or moved 6pm injection to 5 pm and trained at 8 pm.)

In so many evaluated cases, even 21 days of continuous insulin use was pushing it. Frank never used any insulin protocol for more than 28 days, or repeated use without at least a 28 day insulin-free period.

*"Becoming a diabetic is not a good thing or the goal anyone would seek intelligently. That is the equivalent of cutting a finger off to collect insurance. Let me count your fingers, Frank...now the other 9."*

*Coach*

**Absolute Anabolic Phases  
Example #2A - Chart**

DAY	DRUGS
1.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
2.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
3.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
4.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
5.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
6.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
7.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
8.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
9.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
10.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
11.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
12.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
13.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
14.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
15.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
16.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
17.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
18.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
19.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
20.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
21.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.



## **Absolute Anabolic Phases Example #2A - Details**

This was a very straight-forward protocol utilizing the theoretical growth protocol set by nature for GH. The liver had a limited capacity for conversion of GH into growth factors such as IGF-1 during a given period of time. I have noted that GH dosages of 4 iu did not significantly increase circulatory IGF-1 levels above that which were realized from injections of only 2-3 iu. And IGF-1 (as well as other GH fractions) has a very brief half-life.

This meant that four 2iu injections spaced through out the day had obviously produced significantly greater, total amount of IGF-1 than would 2-4 iu or one 8 iu injection.

It was important to avoid administration of GH just before sleep and right after training as these are two natural periods of endogenous production/pulses. Why would Frank given up the extra help? I had also noted "slightly" better results by injecting directly into muscles trained the day prior until after that day's body part was trained. (At which time we switched to that muscle group for injections sites. Duh!)

Humulin-R is a fast-acting human insulin with a half-life of 3-4 hours and, obviously, an active-life of about twice that. Frank's first 10 iu injection was administered upon waking since Frank did not train until about 6-hours later. And the second 10 iu injection was administered immediately following training. GH injections were spaced in between.

So Humulin-R was at 8am and 4pm with GH at 10 am, 2pm, 6pm, 10pm. Frank utilized less insulin due to the activity of IGF-1 produced from GH conversion and because he was using the supplement mix I described earlier. More would have been totally unnecessary at this time (and not necessary in the future because insulin receptor sensitivity was maintained. More on that later...of course).

In fact, many other beasts did do just as well with 6 iu at 8 am and 4 iu at 4 pm. But Frank had used insulin several times prior (miss used). So now that Frank had the ability to trigger anabolism and to transport/store nutrients, he had to accelerate nutrient supply and turn over rates.

Cytomel was a T-3 thyroid hormone. It was necessary to cut 25 mcg. tabs in half on some days to achieve the listed mcg. Frank ingested 100g of carbs with each insulin administration (10 g per iu) with 50 g of protein. So the increased metabolic rate from the T-3 administered in the morning had additional nutrients to force-feed growth stimulated muscle cells.

Frank ate/drank meals every 2 hours (10 meals per day) containing 625 calories each for a total of about 6250 calories daily or about 25 calories per LB of bodyweight. This was not as difficult as some may think. Insulin made all beasts very hungry and a T-3 enhanced metabolic rate needed constant feeding.

It is true that GH was not very effective for mass gains of the immediate nature without the synergistic effects of insulin and T-3 thyroid hormone. Regardless of the synergists that had been layered, successful progress realization from GH administration required a dramatic caloric increase.

It was also a fact that GH had a more dramatic effect when layered with high androgenic AAS. This protocol was easily layered with testosterone. The most dramatic weight and strength increases were with Testosterone suspension. 25-50 mg with each insulin or GH injection provided impressive results. The intent was two 25 mg or 50 mg injections administered daily to assure superior androgenic activity. (Or 200 mg of testosterone propionate on non-GH days.

GH's anabolic activity was mostly due to conversion to IGF-1. When higher dosages of Testosterone Propionate were utilized, the liver produced significant levels of IGF-1 in response. Higher dosages of many oral 17-alkylated AAS also had this effect when the liver attempted to detoxify them.

For estrogen control, Faslodex or Clomid were the better choices since they blocked estrogen receptor-sites rather than inhibiting aromatization and production. This was important because estrogen levels, when high, also increased IGF-1 production.

Another option employed for non-AAS protocol use was the elevated androgen levels created endogenously from Clomid, HCG, or Male Mix to aid in activity. This was truly versatile in application. And the long-term effects from GH use were certainly obvious for Frank.

Humatrope, Serosim or Jintropin was preferred due to their correct 191 amino acid sequence. If Long R-3 IGF-1 was to be layered with in this protocol (and no prior IGF-1 had been utilized) I incorporated a beginning dosage of 20 mcg about an hour after each insulin administration and one either in between or before bed at night depending upon when I trained.

rIGF-1 has a very short active-life of only minutes. (Long R-3 has a very long half-life) However, when bound to its primary binding protein, IGFBP-3, it has a blood-life of about 12 hours. Only free IGF-1 is active, so when bound to IGFBP-3 it is inactive. This is similar to testosterone in that testosterone's binding protein is sex hormone binding globulin (SHBG). Both are controlled by deactivating proteins in blood. As the reader must know by now, bound hormones have been freed or unbound due to synergistic use of other muscle chemistry.

## Absolute Anabolic Phases Example #2A - Chart

The below is an obvious alteration in application for Absolute Anabolic Phase Example 2A, but I thought that I would add it nonetheless.

DAY	DRUGS
1.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
2.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
3.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
4.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
5.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
6.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
7.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
8.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
9.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
10.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
11.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
12.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
13.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
14.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
15.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
16.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
17.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
18.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
19.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg
20.	GH 2 iu 4xd/Humulin-R 10 iu 2xd/Cytomel 50-75 mcg.
21.	Humulin-R 10 iu 2xd/Cytomel 50-75 mcg

## POOR MANS ABSOLUTE ANABOLIC PHASE?

Clonidine HCL was a drug utilized for the treatment of high blood pressure. However, when 0.15-0.30 mg was taken on an empty stomach, it really kicked up endogenous production / release of growth hormone (GH). This was not comparable to 2iu 2xd of exogenous GH, but it did about double normal GH release for a protracted period.

So when the drug was taken just prior to bed and first thing in the morning (on an empty stomach) with 5-10 grams of L-glutamine and the same amount of L-Arginine, it was possible to establish a level of GH release about twice normal. This effect was possible for only about 2-3 weeks before the body caught on.

I had mentioned an individual earlier who was seriously scared of a needle. I outlined his past oral Max Androgen Phase in that section as well as his prior use of pancreatic insulin secretion stimulators (which stimulate the islands of langerhans to release excessive insulin).

He used Glipizide or Glyburide orals and Clonidine or Sermorelin in a 2 day on /2 day off schedule for 28 days. He then began his oral Max Androgen Phase on day #1 5 of his Poor Mans Absolute Anabolic Phase. This was then layered for continued synergy by beginning a Cortisol/Estrogen Suppression Phase (again orals) on day #1 5 of his Max Androgen Phase. He was able to control his blood pressure pretty well too!

Of course there are other interesting GH releasing goodies...

## SERMORELIN

**Dose:** *by intravenous injection*, 1 microgram/kg in the morning after an overnight fast.

### **Can It Induce Antibodies?**

Serum antibodies to GHRH develop in most children receiving treatment with subcutaneous sermorelin. Antibody titres decline sharply 3 months after cessation of therapy and are almost undetectable after 9 months. The clinical relevance of these antibodies is uncertain as there is no correlation between their presence and the effects of sermorelin on growth or on the frequency of adverse effects. Similarly, up to one-fifth of children developed anti-growth hormone antibodies after treatment with recombinant growth hormone preparations. Again, the clinical relevance is unclear.

### **Less Effective than Somatropin?**

Increases in height velocity from baseline values were less in children receiving sermorelin than in those receiving somatropin in 2 studies. Patients were randomised to receive subcutaneous sermorelin (30 ug/kg/day by constant infusion pump delivering a pulse every 24 minutes or as 3 divided doses) or subcutaneous somatropin 30 ug/kg once daily for 6 months in these trials.

The recommended regimen of sermorelin (30 ug/kg once daily) has not yet been directly compared with once daily somatropin, although sermorelin was given at the correct daily dosage.

### **Diagnosis: Sermorelin Specific and Rapid**

Typically, provocative tests of growth hormone secretion with pharmacological stimuli, such as insulin, glucagon, levodopa, clonidine, arginine or propranolol, are used to confirm growth hormone deficiency. The minimum normal peak serum growth hormone response to these tests has been arbitrarily set at 10 ug/L. However, these provocative tests have a number of limitations including sparse normative data, unclear mechanism of action and high variability of growth hormone responses.

Because of the variable responses with these conventional tests, it is recommended that at least 2 provocative tests be performed to confirm the diagnosis of growth hormone deficiency.

### **Starting from the Top**

Sermorelin is a synthetic analogue of growth hormone-releasing hormone (GHRH) that specifically stimulates growth hormone secretion from the anterior pituitary. Since the majority of patients with idiopathic growth hormone deficiency have a deficit in hypothalamic GHRH synthesis or release rather than in growth hormone itself, treatment with sermorelin appears to be a logical approach in the management of these patients.

Sermorelin has been shown to be well tolerated and suitable for use as a provocative test of growth hormone deficiency. Although sermorelin does not appear to be as effective as somatropin in promoting growth in some children with idiopathic growth hormone deficiency, it may be a less costly alternative to somatropin. Sermorelin is compared with somatropin in the Differential features table.

## Benefits

Reverses the effects of aging	Reverses muscle wasting
14% Reduction in Weight and body fat after six months, without dieting	Lowers blood pressure and cholesterol
9% increase in lean muscle after six months, without exercise	Increases energy level, endurance and exercise capacity
Increases cardiac output and athletic performance	Reduces stress and enhances immune system
Growth hormone accelerates wound healing and skin regeneration	Improves memory retention and cognitive functions
Increase recovery from athletic injury	Improves vision
Achieve a younger, tighter, thicker skin	Improves kidney function
Reverse osteoporosis	Improves sleep

### Interesting?

Some beasts have used Somorelin as a pituitary regeneration drug resulting in significant GH secretion after long GH administration periods. Others have used the GHRH preparation as a means of inducing significant GH release as a layer in a Max Androgen Phase or Absolute Anabolic Phase. In both cases 1 -2 mg 3xd resulted in very obvious progress.

## CREATINE AND INSULIN

We know that Frank's insulin use facilitated metabolism, transport, and storage of amino acids (proteins), glucose (carbohydrates) and fatty acids (fats) as well as electrolytes and minerals. We also know that insulin triggered anabolism through multiple pathways.

One form of anabolism (referring to muscle growth) is hypertrophy triggered by cellular osmotic response. This is a state of intracellular hydration in excess of normal. This supra-osmotic state allows for a greater cellular content of macro and micro nutrients. Which in itself triggers an anabolic response. However, this supra-osmotic state also increases immediate available building blocks for protein synthesis and inhibits catabolism. This is a growth environment of a potentially grand scale.

Creatine in any form is an amino acid. The body stores creatine in the form of creatine phosphate/phosphocreatine in the liver and in muscle cells for the purpose of regeneration of adenosine tri-phosphate (ATP). Tissue building requires a great deal of ATP. Limited ATP and ATP regeneration means limited tissue building. (This is too obvious, huh?)

Several studies have proven supraphysiological levels of creatine increase muscle cell hypertrophy and aid in cellular hyperplasia as well as satellite-cell up-regulation.

Several companies market great creatine product that they claim is 880% more effective than regular creatine monohydrate. It is more effective due to 75 grams of glucose to trigger increased endogenous insulin release and 200 mg of alfa lipoic acid to aid in insulin receptor-site sensitivity. They also added a few other minerals necessary for ATP regeneration. Good product (for those who do not fear fat accumulation).

The key fact here is that insulin secretion must be elevated to significantly increase cellular stores of creatine for ATP regeneration (... amino acids to build contractile and structural proteins, glucose as glycogen to fuel hard-core training, and fatty acids to fuel formation of prostaglandins). Add electrolytes for nerve function and ATP formation, as well as increased cellular hydration, and the environment is set for growth.

Gee, do you think Frank's creatine use during Insulin protocols could have been beneficial for muscular augmentation? And pure dextrose or sugar was not necessary to make it all happen. Just protein, carbs and creatine. (The glycemic value of the carbohydrate was mostly a matter of choice and body type)

Frank utilized a creatine-amino acid-carbohydrate shake several times daily during Absolute Anabolic Phases. I had a personal favorite mixture I felt was significantly more effective than any other (of course).

"Human Profile" was a peptide amino acid structure that provides the proper ratio of amino acids, and it absorbed nearly 100%. The issue here is "**oral ratio**", since we are not discussing clinical I.V. introduction. Also a product called "D.E. 20" or whole-wheat flour (we will talk about the latter, later) worked very well for a carb.

### **TO MIX:**

50 grams 50/50 Whey Concentrate & Isolate  
10 grams D.E. 20 per iu of Insulin (Powdered complex carbs)  
10 grams Creatine Monohydrate  
50 milligrams D-Pinitol or / and 200 milligrams of Lipoic acid  
(or 3 grams of ground fenugreek seed. It contains 0.9% 4-hydroxyisoleucine)  
1 teaspoon PG oil mix described earlier

Frank ingested this mixture for the first 5 days at a rate of 3 times daily. Then dropped the amount of creatine per mix/shake to 5 g each, 3 times daily until the final day of his Absolute Anabolic Phase. This resulted in a synergistic response during the following phases as well. Personally, I enjoyed the significant increase in pumps, strength, and total weight gains.

Absolute Anabolic Phases have been utilized as part of an HPTA regeneration period also. Let me explain how that was advantageous.

Insulin suppressed cortisol release while inhibiting circulatory cortisol activity. It also increased androgen activity and down regulated SHBG. This resulted in an elevated plasma level of free (unbound) testosterone and lower cortisol levels/activity to inhibit androgenic induced protein synthesis. So it obviously required very little endogenous or exogenous testosterone to stimulate anabolism.

Therefore, the testosterone level realized from HCC stimulation was far more productive at positively effecting post Max Androgen Phase lean mass retention and growth.

This also has been utilized by layering Humulin-R or Humalog into the "Estrogen Control/HPTA Regeneration Example #1" explained in post AAS Cycle (lean mass retention). Humulin-R or Humalog was utilized in a 2 day on-2 day off protocol. When exogenous insulin had been layered into the prior Max Androgen Phase, an athlete did not utilize exogenous insulin for at least 30 days after discontinuance.

In this case, an oral pancreatic stimulator of endogenous insulin release (such as Glipizide) was a better choice. This potentially aided in regeneration of pancreatic function while significantly increasing lean mass retention and growth potential. Again, a 2 day on /2 day off protocol was utilized with 10 mg of Glipizide 2x daily.

Another option employed was IGF-1 or Insulin layered into Frank's Low Dose AAS Phase Example #1. (2 days on-2 days off). Read the "**Orals**" section with this in mind. Options for controlling negative feed-back loops or utilizing their effects were far more effective when synergy was considered. (Duh!)



## ABSOLUTE ANABOLIC PHASES Example #3 (Options)

Day#	A	B	C	D
1.	GH/Insulin	GH / IGF-1	GH/Insulin	IGF-1
2.	GH/Insulin	GH/Insulin/IGF-1	GH/HCG	IGF-I/GH
3.	IGF-1/HCG	Insulin/IGF-1	IGF-1/Insulin	GH/Insulin
4.	IGF-1	HCG	IGF-1	Insulin
5.	GH/Insulin	GH/IGF-1	GH/Insulin	IGF-1
6.	GH/Insulin	GH/Insulin/IGF-1	GH/HCG	IGF-I/GH
7.	IGF-1/HCG	Insulin/IGF-1	IGF-1/Insulin	GH/Insulin
8.	IGF-1	HCG	IGF-1	Insulin
9.	GH/Insulin/100 mcg T-4	GH/IGF-1/100 mcg T-4	GH/Insulin/100 mcg T-4	IGF-1
10.	GH/Insulin/100 mcg T-4	GH/Insulin/IGF-1/100 mcg T-4	GH/HCG/100 mcg T-4	IGF-I/GH/100-200 mcg T-4
11.	IGF-1/HCG	Insulin/IGF-1	IGF-1/Insulin	GH/Insulin/100-200 mcg T-4
12.	IGF-1	HCG	IGF-1	Insulin
13.	GH/Insulin/100-200 mcg T-4	GH/IGF-1/100-200 mcg T-4	GH/Insulin/100-200 mcg T-4	IGF-1
14.	GH/Insulin/100-200 mcg T-4	GH/Insulin/IGF-1/100-200 mcg T-4	GH/HCG/100-200 mcg T-4	IGF-I/GH/100-200 mcg T-4
15.	IGF-1/HCG	Insulin/IGF-1	Insulin/IGF-1	GH/Insulin/100-200 mcg T-4
16.	IGF-1	HCG	IGF-1	Insulin
17.	GH/Insulin/100-200 mcg T-4	GH/IGF-1/100-200 mcg T-4	GH/Insulin/100-200 mcg T-4	IGF-1
18.	GH/Insulin/100-200 mcg T-4	GH/Insulin/IGF-1/100-200 mcg T-4	GH/HCG/100-200 mcg T-4	IGF-I/GH/100-200 mcg T-4
19.	IGF-1/HCG	Insulin/IGF-1	IGF-1/Insulin	GH/Insulin/100-200 mcg T-4
20.	IGF-1	HCG	IGF-1	Insulin
21.	GH/Insulin/100-200 mcg T-4	GH/IGF-1/100-200 mcg T-4	GH/Insulin/100-200 mcg T-4	IGF-1
22.	GH/Insulin/100-200 mcg T-4	GH/Insulin/IGF-1/100-200 mcg T-4	GH/HCG/100-200 mcg T-4	IGF-I/GH/100-200 mcg T-4
23.	IGF-1/HCG	IGF-1 /Insulin	IGF-1/Insulin	GH/Insulin/100-200 mcg T-4
24.	IGF-1	HCG	IGF-1	Insulin
25.	GH/Insulin/100-200 mcg T-4	GH/IGF-1/100-200 mcg T-4	GH/Insulin/100-200 mcg T-4	IGF-1
26.	GH/Insulin/100-200 mcg T-4	GH/Insulin/IGF-1/100-200 mcg T-4	GH/100-200 mcg T-4	IGF-I/GH/100-200 mcg T-4
27.	IGF-1	Insulin/IGF-1	IGF-1/Insulin	GH/Insulin/100-200 mcg T-4
28.	IGF-1		IGF-1	Insulin

- The option of utilizing T-2 or T-3 was applicable.

Layers upon layers for synergistic manipulation of growth mechanisms constantly presented themselves for the beast with a long-term plan. Any plan absolutely had to include specific intent, long-term potential, and a keen focus upon health. This means optimum progress can only be made at optimum rates if the entire action/reaction equation is considered. And damage to tissue and organs is intelligently avoided.

No doubt some readers of Frank's story will smirk with disdain thinking that health and AAS in the same sentence creates an oxymoron. The vast majority of the information in this story and on these pages is based upon a great deal of research, survey data and interviews, facts gather through years of athlete monitoring by way of viable lab tests results, and personal experience.

So I believe the summery would be this: Are drugs dangerous? Absolutely! But, like guns, only in the hands of idiots and those who lack the mandatory knowledge. This is of course not an endorsement of drug use. It is simply what it is: A fact.

**Frank's Absolute Anabolic Phase Example 3 had four different possible sequences (A,B,C,D) and each had a specific intent of it's own.**

**A:** Was pretty much a one size fits all protocol for constant and significant IGF-1 elevation. It consistently resulted in good lean mass with serious declines in adipose tissue.

**B:** Frank best used this protocol by training 3 days on /1 day off (HCG days were non-training days) allowing for maximum rotation of site-specific injections and growth over the complete body. Due to the insulin sequence, maximum macro/micro nutrient storage-transport-and use was possible before training each 3 day sequence again.

## OPTIONS

Insulin use in these examples was limited to a total of about half of their total length, or 14 days of insulin use during the 27-28 day protocol. This significantly reduced pancreatic negative feed-back loops and rebound issues while in no way limiting results.

### EXAMPLE #3 A

**INSULIN:** Frank has utilized a single injection of combination: 70%/n/30%r (30 iu), first thing in the morning to cover the complete day on scheduled days. This was impossible due to the fact that there were no days that called for both Insulin and IGF-1, without GH to mediate their combined potential hypoglycemic activity.

**GROWTH HORMONE:** Frank used 2-4 2iu GH injections through out each scheduled day. He did not inject during endogenous GH pulses. He has also ramped dosages by beginning at 2 iu/2xd and increased to an additional 2 iu injection every 9 days until reaching 2 iu/4xd. Personally, I started at 2 iu/4xd and "ramped-down." This assured high circulatory IGF-1 levels as the body adapted and down regulated GH conversion to IGF-1.

**INSULIN-LIKE GROWTH FACTOR-1 (IGF-1):** The effective dosage for IGF-1 is listed in research materials as 0.10 mcg/kg 3-5 times daily. The reader should realize however that in child research dosages of 3mg/d (Yes, that is 3 milligrams daily) are not uncommon. So Frank ramped this over the 28 day period: Day # 3,4,7, and 8 = 3xd. Days #11,1 2,1 5,1 6,andl 9 =4xd. Days # 20,23,24,27,and 28 = 5xd. This "up-ramp" counter acted any possible down-regulation of GH conversion to IGF-1. So Frank trained first thing in the morning so that maximum muscular chemical stimulation could be achieved though out the day on freshly traumatized muscle tissue. And when Frank trained:

Day #1 -back/traps/bi's

Day #2- chest/delts/tris,

Day #3-quads/hams/calves

Day #4 -rest while localizing each specific body part trained each day, results were significant.

**C:** Frank best utilized this sequence by training 1 day on/1 day off. Some athletes simply recovered too slowly to train more often. In this example, Frank trained on insulin days only and planned to train lagging body parts specifically on IGF-1 /Insulin days so that he could more efficiently localize for symmetry adjustments. An example was:

Day #1 -legs/back/traps

Day #2 off;

Day #3-chest/delts/arms

Day #4 off.

- This should seem obvious when reviewing Example #3 C.

**D:** This was another protocol that worked best utilizing a 3 day on/1 day off training sequence. As outlined, it allowed for over lapping of weak body part localization. As example, Frank's arms and lateral delts were lagging. The adjustment was made so that he could train:

Day #1: back/chest/traps

Day #2: shoulders/bi's/tri's

Day #3: legs

Day #4: rest .

- He has localized arms and lateral delts on both day #1 and 2 for optimum growth.

By now, the reader has probably realized several other possibilities. Taking into account each chemical's qualities, any training protocol had to be accommodated and Action/Reaction Factors planned for.

**T-4 Thyroid Hormones:** The body normally produces about 76 mcg of T-4 hormone daily. About one third is converted by the liver into the much more active T-3 or about 26 mcg T-3 daily. During GH use there have been cases where body had down-regulated T-4 production and T-4 conversion to T-3. Additional T-4 supplied exogenously has been clinically shown to increase both T-4 and T-3 circulatory levels. However, the process became more effective for beasts with the addition of 25 mg of Ephedrine/325 mg Caffeine/ 2xd on T-4 administration days. I preferred T-3 exogenous sources as a rule, but T-4 tended to have a lower negative feed-back loop potential.

**HCG:** HCG was optional of course. However, there was additional synergy realized with its limited use due to elevation of endogenous androgen production. We have discussed the employed synergy between androgens, Insulin, GH, and IGF-1 and will again. But there was an additional benefit when an Absolute Anabolic Phase was layered into the first 7-15 days of a Max Androgen Phase: The effects of HCG further negated down-regulation of HPTA function. (More on that later) The dosage of HCG administered in this case was 500 iu 3-5 xd on listed days. Remember HCG is actually a female hormone so more was not always better. (But that too had synergistic possibilities.) The addition of Clomid at a dosage of 50 mg/d for 2-3 weeks was also noted to be beneficial by some beasts.

**CLOMID/CYCLOFENIL:** The use of Clomid at a dosage of 50-100 mg/d or Cyclofenil at a dosage of 400-600 mg/d for the last 14 days of an Absolute Anabolic Phase further enhanced the HPTA synergistic factor as well as transitional value when Phase Cycling into and out of a Max Androgen Phase.

## **ABSOLUTE ANABOLIC PHASES EXAMPLE #3B**

**INSULIN:** Humalog 10 iu 3xd on GH/Insulin/IGF-1 days and 7 iu 3xd on Insulin/IGF-1 days. The decreased Insulin dosage was due to the combination of IGF-1 and Insulin without GH to aid in mediating their combined hypoglycemic response. (GH is Hyperglycemic) This meant greater metabolism, transport, storage, and utilization of macro and micro nutrients at lower Insulin dosages. Humalog was a short / fast acting Insulin with a greater activity and control capacity.

**GROWTH HORMONE (GH):** Frank utilized the same dosages and sequence of ramping as explained in **Example #3 A.**

**INSULIN-LIKE GROWTH FACTOR! (IGF-1):** Again the same as **Example #3 A** (HCG and T-4 points applied for all 4 examples.)

**EXAMPLE #3 C:** Frank utilized Humalog or Humulin - R (10 iu 3xd) on GH/Insulin days or combination: 70 % N/30%R (30 iu 1 xd AM) However, a 1/3 reduction in total daily Insulin dosage and only Humalog or Humulin-R was wise for Frank on **IGF-1 /Insulin** days.

\*The same ramp / dosage issues applied to **example #3 C** as outlined in **Example #3 A**.

**EXAMPLE #3 D:** Go re-read Example #3 A. The issues and dosages applied here as well. We had simply altered the sequence to accommodate training and site localization goals. Cool huh?

## **ABSOLUTE ANABOLIC PHASES: An Interesting Discussion**

### ***Truncated Insulin and Bug Spray = The Poor Mans IGF-1 ?***

In the world of the Chemically enhanced athlete exists the really strange truth that someone somewhere has tried it, whatever "it" may be. AAS (anabolic/androgenic steroids)? Yup. Growth Factors? Duh! Bug spray? Oddly enough, yes...and insulin to complete the stack. Why?

Picture an average athlete gaining 20 LBS of lean tissue mass while losing half that amount in adipose tissue without the use of any AAS. Hmmm? Big deal? Have you seen what 20 lbs of lean meat looks like in the grocery store? Now cut away 10 lbs of fat and rethink that big deal idea.

Thankfully many athletes have realized that maximum progress occurs due to working **with** instead of **against** the body's physiological **Action/Reaction Factors**. This means a planned protocol that correctly anticipates and responds to the body's negative reactions to chemically induced positive actions. Anything else simply becomes a matter of attempting to maintain a degree of the positive gains achieved through progressive dosages while negative side-effects mount to a chronic level yet unknown. Ponder that as you read on.

### **Insulin**

Insulin is notably the most anabolic substance there is in the average hard-core beast's chemical muscle enhancement arsenal. Unfortunately insulin's anabolic action is not lean tissue specific. Yup! This means that the employment of insulin includes an anabolic response to fat cells leading to an increase in adipose tissue growth almost equal to lean tissue growth for many wanna-be beasts.

Some claim not to mind the Krispy Creme addict appearance (as they hide within layers of sweatshirts) and unnecessarily view it as the lesser of two evils. (The word "small" is the vilest word spoken in the gym and the bedroom alike.)

### **Warning: Insulin Science Geek Stuff**

Insulin stimulates the all-important activity of the cell Sodium/Potassium Pump (SPP) which in turn increases the rate and level of nutrient transport into cells. (Cell feeding mediator) Though the three macronutrients: Protein (in the form of amino acids), fats (in the form of fatty acids) and carbohydrates (in the forms of glucose and glycogen) are all paramount to the growth process, it is amino acid (AA) transport potential that we are most focused upon in this article. The reason is obvious in that the growth rate or hypertrophy of muscle cells is proportionate to the rate and level of AA entry into them. No one makes freak status with a low AA transport potential.

Insulin and other anabolic substances increase the process of protein synthesis (anabolism) and RNA activity. Insulin is believed to affect DNA in a way that causes an up-regulation in DNA initiated protein synthesis. Of all anabolics, insulin is far more powerful and important in this respect due to the Sodium/Potassium Pump factor and, specifically, BCAA (Branch Chain Amino Acids) up-take as a result of its actions.

Insulin inhibits protein break-down (catabolism) by directly interfering with the activity of certain structures called lysosomes. Lysosomes are intricate to the action of breaking down cellular proteins into amino acids so that they may be used as energy. In case you missed the point, these proteins are cannibalized from muscle tissue. By acting as an anti-catabolic substance, insulin inhibits muscle loss. Unfortunately insulin also inhibits fat loss as well.

For a moment imagine the freaky potential insulin would possess for lean tissue growth if it did not inhibit fat loss while remaining anabolic to muscle tissue? Yup, a poor mans IGF-1 would be the result!

*(The following is an excerpt from Chemical Muscle Enhancement)*

## **DNP (2,4 DINITROPHENOL)**

This was truly reported as being chemical exercise. Normally the mitochondria process that converts ADP (adenosine diphosphate) into ATP (adenosine triphosphate) is about 60% efficient, which means there is a great deal of energy wasted. Those who have read the creatine section ahead of this are well aware of our good friend ATP. When we exercise, this process accelerates and raises our metabolic rate. (More calories are burned as a result) The process is called oxidative phosphorylation. Since ATP is the high-energy chemical our bodies utilize for intense training, anything that compromises this process will make cellular mitochondria work harder and expend more energy as heat. (Body temperature rises)

DNP is an oxidative phosphorylation uncoupler. It makes the process only about 40% efficient by uncoupling a high-energy phosphate molecule from ATP and therefore turning ATP into ADP. To maintain an adequate supply of ATP, the body must step-up production. For this reason metabolism is significantly increased and an incredible amount of calories are burned. During this accelerated metabolic state, and due to the need for ATP production, most of the calories come from fatty acids (adipose/fat tissue). So little or no muscle is lost (With adequate protein intake).

Users experienced elevated body temperatures and perspiration even while sitting around. Simply stated, metabolic rates elevate 100-200% in only a few hours. **Sounds** great, but DNP can be deadly. It has even been used as a component of bug spray. Since increased energy is dissipated as body heat, too high of a dosage of DNP for too long of a period can actually COOK ORGANS! No joke, I mean medium well done. I cannot stress enough how dangerous the use of this chemical can be. The

body usually possesses receptors or pathways to shut down as a means of survival from the potential life threatening dangers and damage of most chemicals. In the case of DNP there are none.

The issue of body temperature is of interest here and is a relevant point to discuss further. Clenbuterol and ephedrine are fairly easy to chart for effective results by checking body temperature. However, DNP is much different in this sense. When an athlete (or anyone) used DNP, increased respiration, heart rate, and skin dilation occurred. Thus heat is quickly dissipated.

This means that a person using DNP could feel warm but a thermometer can fail to show an increase in body temperature. According to available literature, in most cases a body temperature of near 100 degrees indicates a metabolic rate of about twice normal. It also means that the individual is in the very near the danger zone. This is wholly unnecessary, and it is the low cellular ATP level induced by high dosage DNP use that was most dangerous.

The temperature or heat issue is secondary by comparison. Most reported users of DNP ingested a daily dosage of 6-8 mg per kilogram of body weight. Realistically speaking, I can say from personal experience that this is not only an uncomfortable experience, but dangerous and unnecessary as well. My experience has been that 3-5mg/kg daily provided better results and did so even without a calorie decrease. Personally I felt a body temperature of 99.5-99.7 degrees was preferable also.

Before going on, I would like to say a few related points. Now we know that the mitochondrial process of converting ADP into ATP is called oxidative phosphorylation and that the process is normally 60% efficient. We know DNP is an oxidative phosphorylation uncoupler that will reduce the process efficiency to 40% and that this burns fat while raising metabolic rates 100-200% while increasing body temperature. (We also know misuse can cook our guts!).

***\*So now we have insulin and its "anabolic to muscle and fat" alike qualities and DNP that increases calorie expenditure primarily from fat stores. It would seem that we have applied Action/Reaction Factors correctly to create an increase in lean tissue mass at the expense of adipose tissue stores. But here is where it gets really interesting...***

## ***Warning: More Science Geek Stuff Interesting***

### **Studies Sometimes Validate What We Have learned**

\*Rapid stimulation of glucose transport by mitochondria! uncoupling depends in part on cytosolic Ca<sup>2+</sup> and cPKC  
Z A Khayat, T Tsakiridis, A Ueyama, R Somwar, A Kiip  
AMERICAN JOURNAL OF PHYSIOLOGY, 275(6 Pt 1):C1487~C1497 1998



2,4-Dinitrophenol (DNP) uncouples the mitochondria! oxidative chain from ATP production, preventing oxidative metabolism. The consequent increase in energy demand is, however, contested by cells increasing glucose uptake to produce ATP via glycolysis. In skeletal muscle cells, DNP rapidly *doubles glucose transport*, reminiscent of the effect of insulin. However, glucose transport stimulation by DNP does not require insulin receptor substrate-1 phosphorylation and is wortmannin insensitive.

Overnight treatment with 4-phorbol 12-myristate 13-acetate down-regulated cPKC isoforms alpha, beta, and gamma and partially inhibited (45.0 +/- 3.6%) DNP- but not insulin-stimulated glucose uptake. Consistent with this, the PKC inhibitor bisindolylmaleimide I blocked PKC enzyme activity at the plasma membrane (100%) and inhibited DNP-stimulated 2-[3H]deoxyglucose uptake (61.2 +/- 2.4%) with no effect on the stimulation of glucose transport by insulin. Finally, the selective PKC-beta inhibitor LY-379196 partially inhibited DNP effects on glucose uptake (66.7 +/- 1.6%). The results suggest interfering with mitochondria! ATP production acts on a signal transduction pathway independent from that of insulin and partly mediated by Ca<sup>2+</sup> and cPKCs, of which PKC-beta likely plays a significant role.

*So now we know that insulin is not the only mediator of glucose transport into muscle cells. We also have validated the increase in potential muscle glycogen synthesis during the employment of DNP is about twice that of insulin. Hmmm, not getting it yet? Be patient. You will in a minute.*

\*Effects of cellular ATP depletion on glucose transport and insulin signaling in 3T3-L1 adipocytes  
E Heart, J Kang, C K Sung  
*American Journal of Physiology - Endocrinology and Metabolism*, 280(3):E428-E435 2001

Glucosamine induced insulin resistance in 3T3-L1 adipocytes (fat cells), which was associated with a 15% decrease in cellular ATP content. To study the role of ATP depletion in insulin resistance, researchers employed sodium azide (NaN<sub>3</sub>) and dinitrophenol (DNP), which affect mitochondrial oxidative phosphorylation, to achieve a similar 15% ATP depletion.

Unlike glucosamine, NaN<sub>3</sub> and DNP markedly increased basal glucose transport, and the increased basal glucose transport was associated with increased GLUT-1 content in the plasma membrane without changes in total GLUT-1 content. These agents, like glucosamine, did not affect the early insulin signaling that is implicated in insulin stimulation of glucose transport. In cells with a severe 40% ATP depletion, basal glucose transport was similarly elevated, and insulin-stimulated glucose transport was similar in cells with 15% ATP depletion.

In these cells, however, early insulin signaling was severely diminished. These data suggest that cellular ATP depletion by glucosamine, NaN<sub>3</sub>, and DNP exerts differential effects on basal and insulin-stimulated glucose transport and that ATP depletion per se does not induce insulin resistance in 3T3-L1 adipocytes.

DNP aids in inducing an environment of insulin resistance in adipose sites thus decreasing the ability for fat cells to get food. So DNP increases calorie expenditure as heat, increases glucose transport into muscle cells but decreases fat cell gluttony. Gee, do you think the combination of the super anabolic insulin and DNP just may be very pro-muscle growth and fat loss?

## **Closing Thoughts and Other insanity**

When athletes have employed the DNP/Insulin Protocol in the past there has been a noted dramatic increase in lean tissue mass and a lack of hypoglycemia in almost all cases. This in itself was an exciting issue to research as one of the many negative side effects possible from non-medically supervised administration of insulin in hypoglycemia and coma...and death. (All of which suck) There are two possible explanations for this:

1. The insulin molecule is experiencing N-terminal truncation when coming into contact with circulatory DNP. When the N-terminal is removed from IGF-1 the resulting growth factor is called Des (1-3) IGF-1. The result is an anabolic far more powerful than even IGF-1 itself. This is true of IGF-2 and other growth factors including insulin. The possibility strongly suggests that the truncated insulin molecule would more readily fit into and activate the muscle cell IGF-1 receptors as well.
2. The insulin and IGF-1 receptors themselves are truncated by the presence of DNP. In this case the truncation is effecting the COOH-Terminal response thus altering the hypoglycemic and anabolic effects of insulin positively toward that of IGF-1 in both function and action.

\*There are several approaches that have been employed for Insulin and DNP but one of the more effective examples follows. **Please do not try this at home.** (Even with proper medical supervision and a professional exterminator.)

## WARNING!

This is intended as a discussion example only and not meant as a guide for use. DNP and insulin can both be very dangerous chemicals. Insulin use must be medically supervised and DNP has not been a legal supplement since the 20's.

### Insulin & Bug Spray Example Protocol

DAY	DRUGS
1.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
2.	
3.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
4.	
5.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
6.	
7.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
8.	
9.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
10.	
11.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
12.	
13.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
14.	
15.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
16.	
17.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
18.	
19.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
20.	
21.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
22.	
23.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
24.	
25.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
26.	
27.	Humulin-R 8-10 iu 3xd/DNP 4-5mg/kg Daily
28.	

- Optional layer: Avandia 2mg 2xd increases muscle cell insulin receptor sensitivity and facilitates an improved IGF-1 profile.

## **HORMONE SYNERGY (More things to think about)**

Before we continue the story of how a beast was constructed, I would like to discuss hormone synergy. Synergy was the introduction of two or more hormones in such a way that the total effect is greater than the sum of the individual hormone at higher dosages. (More is not always better. Better is better.)

Over the years, we have observed bigger and leaner athletes. It seems to come in spurts, or periods of adjustments. The top competitors will come in 6-10 LBS heavier and a little leaner, then one arrives far larger, and the rest suddenly catch up the following year. This is due to individuals who pioneer new chemistry and learn how to use it in synergy with the rest of their gear for optimum progress. This synergy is not all that difficult to understand for the most part.

As we know there is bound (inactive) testosterone and free (active) testosterone. Only the unbound free testosterone is capable of inducing an anabolic/androgenic action. Sex hormone binding globulin (SHBG) binds about 98-99% of total testosterone within the blood stream. So a daily plasma threshold level of 100 mg of testosterone would only allow about a 1-2 mg active or free testosterone level at any given time. And what amazing things that 1-2 mg have done. Testosterone also suppresses fat storage enzymes.

Insulin is both anabolic and anti-catabolic. We know about insulin's ability to shuttle amino acids and glucose into tissues, unfortunately it also is the main fat storage hormone. Research has also shown insulin affects storage of only about half of the essential amino acids. But did you know Insulin also reduces SHBG? Yup! So when AAS were stacked or layered with Insulin in a protocol there was an existing increase in free testosterone.

Second, due to AAS ability to suppress the fat storage process, insulin had a superior ability to shuttle nutrients into muscle instead of fat cells. A third synergistic event happened when insulin induced increases in both glucose and amino acids storage made possible significant cellular protein synthesis induced by AAS.

Last, we know in the presence of elevated circulatory insulin levels, lower plasma cortisol levels existed. Therefore this allowed a significant alteration in the anabolic/catabolic ratio in favor of anabolism. Too bad insulin was Lipogenic (fat synthesis promoting).

GH is Lipolytic (fat burning) and acts to increase muscle structural proteins, induce hyperplasia, and increase the cellular uptake of about half of the essential amino acids. Guess what? It is the complimentary half of essential amino acids to those insulin increased cellular uptake of. Without a complete and properly ratioed

amino acid profile, growth is significantly retarded regardless of hormone source. A single deficiency in any one amino acid will limit growth in proportion to its limit. GH stacked with testosterone induced an increase in cell/fiber count in muscle tissue while significantly increasing muscular hypertrophy.

It also increased half of the amino acid uptake and use to produce growth, while joining together to prevent fat storage. And the fat was better utilized for energy needs. Since GH and Insulin stacked together supported uptake of **all** essential amino acids, significant protein synthesis occurred. Add an AAS and a stronger anabolic message was realized... And again, synergy was improved.

Researchers have proven that gross **muscular** weight gains are modest with either GH or Insulin. The same researchers have also proven combined synergistic stacks of Insulin and GH create enormous gross and lean mass weight gains (Duh!)

*" The future will be strangely interesting, my Lad. With the addition of IGF-1, PGF-2, PGE-I, INTERLEUKIN-15, myostatin inhibitors, and MGF to the synergistic pool, athletes will compete at 350-400 LBS. And it will not be just the genetic gifted individuals."*

*Coach*

But what about lagging body parts? (More things to think about later!)

## PHASE CYCLING

I often read articles concerning genetic limitations. It is true that genetics play an important role in growth and body symmetry potential. However, many beasts have significantly altered their genetic predisposition because they were aware of Action/Reaction Factors.

I have personally witnessed individuals go from 150 LB 6 foot "ectomorph" to a 300 LB "mesomorphs". Ya, I know, cannot be done, right? **Wrong!** Though skeletal structure and height cannot be significantly altered, pretty much everything else has been by athletes that learned how.

. So what does science have to say about genetic body types? There are three basic body types, though any individual can be a mixture of two by having qualities ranging between two types.

**Ectomorph:** Usually a person considered an ectomorph is small boned or framed and has difficulty adding body weight because their metabolic rate is very high. We know thyroid hormones have the greatest effect upon metabolic rates and we also know this also means a very high PTOR. Add training, reduce catabolism, add major calories, and this person grows very quickly.

Since an ectomorph has an unusually high thyroid hormone levels they also heal very quickly between workouts and can train more often. But must use brief intense work-outs. Ectomorphs tend to need 8-12 meals per day. What is really cool about these individuals is that they show every pound of muscle they gain as if it were 2 pounds or more due to smaller joints and bones.

And if they choose to add significant mass, they look like Flex Wheeler. The interesting fact is that ectomorphs have metabolic rates many endomorphs and mesomorphs have attempted create with synthetic thyroid hormones. So the wiser ectomorphs avoided exogenous thyroid hormones use for prolonged periods when mass gain was the goal.

**Endomorph:** Though several fat people swear they are endomorphs, the truth is that endomorphs are simply large boned. They are usually heavy set and have fairly slow metabolic rates. Again, thyroid hormones control metabolic rate for the most part. Since they have slower metabolic rates, they put on weight easily.

Unfortunately this is also a predisposition for fat accumulation because they recover from work-outs much more slowly. Think about that. If a person cannot increase training intensity there is a poor nutrient partitioning effect stimulus for protein-based tissue such as muscle. Increased metabolic rate and induced significant anabolic signals would result in this endomorph becoming a monster rather quickly.

And their fat problems have been noted to commonly disappear. Endomorphs usually require 7-10 days to recover between each body part work out. Back and legs can take even longer.

**Mesomorph:** These are considered the lucky ones. Most of the best natural bodybuilders are mesomorphs in fact. They have few problems with maintaining body mass and have average size bone structure. They are leaner than endomorphs yet carry slightly more body fat than ectomorphs. So it should be apparent that they have what would be considered a normal metabolic rate. Therefore recover from work-outs within 3-5 days as a rule.

We often discuss fiber types but not muscle density. Muscle density is simply a matter of possessing a higher number of muscle fibers. More experienced athletes have greater muscle density, as a rule, due to increased fiber count induced by chemical and training stimuli.

Do you remember our friends the muscle satellite-cells? Gee, do you think an athlete can improve muscle density? Just something to consider. Now on to Phase Cycling.

## Phase Cycling?

Phases may were utilized totally independent of each other with quite profitable results when negative feed- back loop counter measures were employed. However, there was synergistic value to "**Phase Cycling**".

Phase Cycling allowed for greater results and adjustments for ***specific intent***. Of course there was the prolonged overall "***most effective period***" to consider as well. Possibilities were endless when Action/Reaction Factors were accounted for.

Frank chose Max Androgen Phases that terminated high activity within 30 days, 28 day Cortisol/Estrogen Suppression Phases, and 28 day Absolute Anabolic Phases. There were 3 basic sequences employed for maximum progress while inhibiting negative feed-back loops.

### **EXAMPLE A**

Day #1-30:	Absolute Anabolic Phase
Day #15-44:	Max Androgen Phase ( <i>Added 30-60 mg/d Nolvadex day #15- 31</i> )
Day #31-58:	Cortisol/Estrogen Suppression Phase

#### ***OPTIONS UTILIZED:***

- \*10 mg 2xd Glipizide on days #30,32,34,36,38,40,42.
- \*50 mg 2xd Proviron on days #45-58 (Protest is an alternative)
- \*400-600 mg/d Cyclofenil on day #1 5-30.

This sequence was exceptionally beneficial for ectomorph beasts since they needed all of the cortisol control they could get post-cycle, but can't we all.

### **EXAMPLE B**

Day #1-30: Max Androgen Phase (*Added 30-60 mg/d Nolvadex day #1-15*)  
Day #1 5-42: Cortisol / Estrogen Suppression Phase.  
Day #31-58: Absolute Anabolic Phase

#### **OPTIONS UTILIZED:**

- \*10 mg 2xd Glyburide on day #2,4,6,8,10,12,14, or #1-3,7-9,1 3-1 5.
- \*4ml/d Protest (w/prohormones) on days #31-51.
- \*50-100 mg/d Clomid on days #45-58.
- \*500 iu 4-6 xd HCC on days #45,48,51,54,57.

This allowed endomorphic beasts to greatly reduce fat stores after a massive weight gain period while exiting the Phase Cycle in a very anabolic environment. Frank's body type was between an endomorph and a mesomorph. For those that had low thyroid hormone levels, naturally this was great.

### **EXAMPLE C**

Day #1 -30: Max Androgen Phase (*Added 30-60 mg/d Nolvadex day #1-30*)  
Day #1 5-42: Absolute Anabolic Phase.  
Day #31-58: Cortisol/Estrogen Suppression Phase

#### **OPTIONS UTILIZED:**

- \*400-600 mg/d Cyclofenil on days #16-30 (or)
- \*1.0-2.0 mg/d Arimidex on days #16-30.
- \*50 mg 2xd Proviron on days #31-58 (or)
- \*10-25 mg/d Clomid on days #55-68 w/500 iu 4-6 xd HCG on days #55,58,61,64,67.

This worked well for those beasts that were endomorphs with an above average active estrogen level and a near normal active thyroid hormone level. The term "active" was of importance due to the fact that endomorphs tended to possess high "total" circulatory estrogen and thyroid hormone levels, but not necessarily high levels of "active" or unbound estrogen and thyroid hormone levels.

As the reader realizes by this time, only active/unbound/free hormones can merge with their receptor-sites and trigger a response. By utilizing the structure of Example C, endomorphs were able to exit a Phase Cycle with low estrogen and cortisol levels. This obviously meant little or no post-cycle lean mass tissue loss for them.



Mesomorphs responded well to most Phase Cycling techniques but did best when they avoided cycle exits utilizing thyroid hormone type drugs when pure mass gain was the goal. They usually had average thyroid hormone profiles.

It should seem evident that there were many other options utilized such as low dosage AAS protocols, site-injection protocols, HPTA regeneration protocols, and shorter layers of Absolute Anabolic or Cortisol / Estrogen Suppression Phases. It simply depended upon specific-intent and Action/Reaction Factors. The idea was to avoid long protocols of a single type of phase so as to be able to properly respond to negative feed-back loops.

We have had great results using the original Protest in a Phase Cycle I will outline. Most were amazed at the results of this type of Phase Cycle when one considers the chemistry used.

#### **EXAMPLE D:**

Day #1-30: Max Androgen Phase (*Added 30-60 mg/d Nolvadex*) Protest  
Day #20-48: Max Androgen Phase. Cortisol/Estrogen Suppression  
Day #15-36: Phase Example 1 Absolute Anabolic Phase Example  
Day #37-57: 2. 50 mg 2xd Clomid.  
Day #42-55: Day #42,44,46,48,50,52,54 take 500 iu 4-6 xd HCG.

## **GROWTH PLATEAUS (Poor Mans IGF-1/Androgen Cycle)**

Some athletes reached a plateau (of size and weight) and seemed unable to get over it. This was almost always due to misuse of cycles and a lack of proper negative feed-back loop suppression. Action/Reaction, remember?

Sometimes the problem was a lack of "support networks" (Huh?). Support networks was a term describing vascular tissue to supply blood and nutrients (as well as hormones) and a neurological network to communicate with the tissue.

Site-injection protocols were an excellent method utilized to induce site-specific growth. Simply stated, an increase in area was created with a space occupier. Over a period of time vascular and nerve tissue filled the space. An increase in muscular tissue was then realized due to fiber expansion into a greater available space. This was great for lagging body parts. But what about total body mass?

Have you ever noticed what happens when a fat dude becomes a hard-core bodybuilder? They diet off the fat and then seem to grow like a politician's bank account. This is simple to explain.

Fat is a space occupier and fat needs support networks to exist. Once the fat is gone, the space still exists and the support networks remain. Thus allowing unobstructed growth of newly stimulated muscle mass. Their body's need for balance, or homeostasis, actually fights to gain body weight and mass. It is the ex-fat dude's choice what the mass consists of.

This offered insight into beating growth plateaus and a few choices or ways to do it. Get really fat, drop the fat, and fill the space with muscular mass?

Intracellular and extracellular water weight is a space occupier. And intracellular increases in cellular volume trigger anabolism. So did a significant increase in androgen levels and IGF-1 production.

Estrogens increased water retention and liver production of IGF-1. They also made androgen receptor-sites very sensitive. Creatine and insulin increased intracellular volume, nutrient storage, and ATP regeneration while adding to receptor-site sensitivity. Can you say synergy?

Testosterone obviously aromatizes to estrogens. But Methandriol Dipropionate (MD) did all the estrogen stuff due to its chemical structural similarities to estrogen. Several Aussie AAS were either MD, or have a high level of MD with low dosages of either nandrolone or boldenone (EQ).

Humalog or Humulin-R are short acting Insulin analogues. They unfortunately suppressed pancreatic function when utilized too frequently.

Glipizide (GZ) is an oral drug medically prescribed for non-insulin dependent diabetics and is provided in 5-10 mg tabs. It works by inducing pancreatic release of insulin and therefore had some post exogenous insulin use synergy. Since it induces pancreatic insulin release, GZ potentially aids in regenerating suppressed pancreatic function caused by exogenous insulin use.

\*So does 4-hydroxyisoleucine.

The below was a well-timed testosterone cycle utilizing: Sustanon-250, testosterone enanthate, and Testosterone Propionate. **Gyno withstanding**, very low estrogen control was employed utilizing Nolvadex as needed. Some beasts called it "The Poor Man's IGF-1/Androgen Cycle". I simply called it *effective*.

### Poor Mans IGF-1/Androgen Cycle Example #1 - Chart

DAY	DRUGS	DAY	DRUGS
1.	MD 50 mg/SUS-250 (500 mg)/Insulin 10 iu 2xd	16.	Test. E. 250 mg/Insulin 10 iu 2xd
2.	MD 50 mg/Insulin 10 iu 2xd	17.	Insulin 10 iu 2xd
3.	MD 50 mg/SUS-250 (500 mg)/Insulin 10 iu 2xd	18.	MD 50 mg/Insulin 10 iu 2xd
4.	MD 50 mg/Insulin 10 iu 2xd	19.	
5.	MD 50 mg/Sus-250 (500 mg)	20.	
6.	MD 50 mg	21.	MD 50 mg/Test. P. 100 mg
7.	MD 50 trig/Test. E. 500 mg	22.	GZ 10 mg 2xd
8.	GZ 10 2xd	23.	Test. P. 100 mg/GZ 10 mg 2xd
9.	MD 50 mg/GZ-10 mg 2xd	24.	GZ 10 mg 2xd
10.	Test. E. 500 mg/GZ 10 mg 2xd	25.	Test. P. 100 mg/GZ 10 mg 2xd
11.	MD 50 mg/GZ-10 mg 2xd	26.	
12.		27.	Test. P. 50 mg
13.	MD 50 mg/Test. E. 250 mg	28.	
14.		29.	Test. P. 50 mg
15.	MD 50 mg/Insulin 10 iu 2xd	30.	

- 0.30 g of creatine per kg of bodyweight daily was a utilized option on listed Insulin days
- This cycle normally required a Cortisol/ Estrogen Suppression Phase of 14-30 days beginning day #27.
- Blood pressure was monitored frequently. Most healthy bodies can withstand short periods of elevated blood pressure. Long periods could have possibly damaged Frank or any of the beasts. Blood pressure was not something to be taken lightly!

## Poor Mans IGF-1/Androgen Cycle Example #1 - Details

It was a fact that only through adequate increases in support networks, and proper Phase Cycling to induce them, did an athlete realize significant permanent increases in ***optimal lean body mass***.

Just as Frank began at a genetic weight threshold or range, he had to also establish new genetic weight thresholds that the body will interpreted as normal. At each step of progress the body then resumed homeostasis at an increased genetic weight due to perceived normalcy. To retain this perceived genetic normalcy or new level of homeostasis, adequate nutrient/calorie support and training stimulus was required.

Negative feed-back loops inhibited progressive levels of homeostasis as much as they destroyed significant progress. Through correct anticipation of action/reaction factors and subsequent proper response to them, maximum increases in genetic weight/favorable body composition were supported and maintained by homeostasis.

I know of several individuals who had for years maintained an "on cycle" weight of 230-240 lbs and an "off cycle" weight of 200-210 lbs at one time. Through proper Phase Cycling and protocol structure, they eventually reached a level of 285-300 lbs "on cycle", and 250-270 lbs "off cycle".

No matter how many times I witnessed this metamorphosis, it never failed to amaze me. However, what "thrilled me" was the health factors of these individuals. They had significantly *improved* when compared to only a couple of years prior.

*"There will always be those individuals who, when faced with overwhelming facts to the contrary, will continue to believe a lie simply because it is the lie that they are most comfortable with. Or perhaps the lie merely justifies their actions no matter how erroneous. Such is the destiny of the AAS gunslinger whom refuses to learn and grow for the experience."*

*Coach*

## ALL OUT PSYCHO LAYER CYCLE Example #1 - Chart

DAY	DRUGS
1.	Test. S. 100mg/CH 2iu 4xd/Humulin 10iu 2xd/Cytadren 500mg
2.	Test. S. 100mg/GH 2iu 4xd/Humulin 10iu 2xd/Cytadren 500mg
3.	Test. S. 100mg/GH 2iu 4xd/Humulin 10iu 2xd/Teslac 250mg/Nolvadex 30mg
4.	Test. S. 100mg/IGF-I 20mcg 4xd/Teslac 250mg/Nolvadex 30mg
5.	Test. S. 100mg/IGF-I 20mcg 4xd /Cytadren 500mg
6.	Test. S. 100mg/IGF-I 20mcg 4xd /Cytadren 500mg
7.	Test. S. 100mg/IGF-I 20mcg 4xd/Teslac 250mg/Nolvadex 30mg
8.	Test. S. 75mg/Win. D . 25mg/GH 2iu 4xd/Humulin 10iu 2xd/Teslac 250mg/Nolvadex 30mg
9.	Test. S. 75mg/Win. D 25mg/GH 2iu 4xd/Humulin 10iu 2xd/Cytadren 500mg
10.	Test. s 75mg/Win. D 25mg/GH 2iu 4xd/Humulin 10iu 2xd/Cytadren 500mg
11.	Test. s 75mg/Win. D 25mg/IGF-I 20mcg 4xd /Teslac 250mg/Nolvadex 30mg
12.	Test. s 75mg/Win. D 25mg/IGF-I 20mcg 4xd /Teslac 250mg/Nolvadex 30mg
13.	Test. s 75mg/Win. D 25mg/IGF-I 20mcg 4xd /Cytadren 500mg
14.	Test. s 75mg/Win. D 25mg/IGF-I 20mcg 4xd /Cytadren 500mg
15.	Test. s 50mg/Win D . 50mg/GH 2iu 4xd/Humulin 10iu 2xd/Teslac 250mg/Nolvadex 30mg }
16.	Test. S. 50mg/Win D . 50mg/GH 2iu 4xd/Humulin 10iu 2xd/Teslac 250mg/Nolvadex 30mg
17.	Test. s 50mg/Win. D 50mg/GH 2iu 4xd/Humulin 10iu 2xd/Cytadren 750mg
18.	Test. s 50mg/Win. D 50mg/IGF-I 20mcg 4xd /Cytadren 750mg
19.	Test. s 50mg/Win. D 50mg/IGF-I 20mcg 4xd /Teslac 250mg/Nolvadex 30mg
20.	Test. s 50mg/Win. D 50mg/IGF-I 20mcg 4xd /Teslac 250mg/Nolvadex 30mg
21.	Test. s 50mg/Win. D 50mg/IGF-1 20mcg 4xd /Cytadren 750mg
22.	Test. s 25mg/Win. D 75mg/GH 2iu 4xd/Humulin 10iu 2xd/Cytadren 750mg
23.	Test. S. 25mg/Win D . 75mg/GH 2iu 4xd/Humulin 10iu 2xd/Teslac 250mg/Nolvadex 30mg
24.	Test. S. 25mg/Win D . 75mg/GH 2iu 4xd/Humulin 10iu 2xd/Teslac 250mg/Nolvadex 30mg
25.	Test. s 25mg/Win. D 75mg/IGF-I 20mcg 4xd /Cytadren 750mg/Nolvadex 30mg
26.	Test. s 25mg/Win. D 75mg/IGF-I 20mcg 4xd /Cytadren 750mg/Nolvadex 30mg
27.	Test. s 25mg/Win. D 75mg/IGF-I 20mcg 4xd /Teslac 250mg/Nolvadex 30mg
28.	Test s 25mg/Win. D 75mg/IGF-1 20mcg 4xd /Teslac 250mg/Nolvadex 30mg

- Test. S. =Testosterone Suspension
- Win. D. = Winstrol Depot
- Humulin = Humulin-R
- By some standards the listed dosages for Long R-3 IGF-1 were conservative. (But cost effective as is)
- IGF-1 employed was Long R-3 IGF-1

## ALL OUT PSYCHO LAYER CYCLE

### Example #1 - Details

This was an All Out Psycho Layer Cycle. Frank did not use this type of cycle more than once or twice a year simply because his (or any beast's) body would have had difficulty enduring utilization more often. The only addition this protocol had also benefit from was the inclusion of Lutalyse (PGF-2) at a dosage of 2mg 3-5 times per day injected into weak body parts (Site-specifically) on listed IGF-1 days.

**Note:** Some people felt "crampy" (The shits) during PGF-2/androgen co-administration.

**Testosterone suspension** was a fast-acting drug with powerful effects. Utilizing Test. S. in a down-ramping protocol was necessary due to its brief half and active-life. Testosterone suspension was notably more powerful milligram for milligram than Sustanon-250, by the way. Due to this drug possessing such a brief active-life (of about 24 hours) this protocol established a 100mg plasma level threshold by day #2.

**Winstrol Depot** was a highly anabolic and fast-acting AAS. Unfortunately it was also quite dirty due to its 17 alkylated structure. However, 21 days of continuous administration was not a problem for Frank.

I realize that I have said this prior, but the reader should be aware of the fact that the listed dosages for IGF-1 were rather conservative by some athlete's standards. The truth is that the listed dosages had been proven effective while maintaining some sense of cost effectiveness. GH and IGF-1 were alternated in this example to allow for a synthesis up-regulation of the liver enzymes that facilitate GH conversion into highly active growth factors (Like IGF-1). This was effective at the cellular level as well.

**Insulin** was utilized for a total of 12 out of 28 days. This protocol therefore had little detectable negative effect upon Frank's pancreatic function. The goal was to turn Frank into a beast, not a diabetic. In this protocol insulin was administered first thing in the morning and directly following Frank's work-out.

Naturally the normal 10 grams of carbohydrates (Minimum) per iu of insulin administered was ingested also. Two of the 2iu GH injections were spaced and administered after the A. M. insulin injection. The remaining two 2iu injections were spaced and utilized after the afternoon work-out. My experience has been that in most individuals the co-administration of androgens (AAS) with insulin resulted in increased insulin receptor-site sensitivity. So, initially, less was more effective. When Frank was a first-time insulin user, he adjusted his dosages to 4-6iu each.

**Note:** The reader should note again the fact that insulin must be present or the liver fails to maximize GH conversion to growth factors.

The most effective method of IGF-1 and GH use was to inject directly into the muscle that was trained that day. This was a site-specific application. As with any injection there was a procedure to adhere to.

1. After cleaning the injection site with alcohol and a sterile cotton ball, Frank inserted the needle deep into the muscle.
2. Pulled back gently on the syringe plunger and checked for blood filling the syringe. (Hitting a vein was a bad idea!)
3. If no blood appeared, Frank slowly injected contents.
4. Pressed an alcohol dipped cotton ball against the injection site for a few seconds.
5. All beasts were careful to never administer an injection of IGF-1 near the abdominal area. There is a high concentration of IGF-1 receptors in abdominal organ tissue and no one needed enlarged guts.

### **(Back To Frank's) ALL OUT PSYCHO LAYER CYCLE**

**Cytadren** was used in lower dosages due to the anti-catabolic effects of GH, IGF-1, and Insulin. **Some** cortisol was necessary for health and immune system function. Since Cytadren administration inhibited endogenous sex hormones and hormone biosynthesis at the very beginning, it also acted to prevent estrogen production. It was a fact that GH and IGF-1 needed some estrogenic and corticoid activity to reach full potential.

**Nolvadex** 10 mg a.m. and 20 mg p.m. handled estrogenic activity at the receptor-site while Teslac prevented excessive aromatization and production for the rest of Frank's estrogen control. Normally, Teslac would have been enough to keep HPTA function from totally shutting down. But it was necessary to utilize some HCG and/ or Clomid for a couple of weeks post cycle for some beasts.

When a beast had a natural low metabolic rate, Cytomel (T-3 thyroid) was added at a dosage of 37.5-75 mcg daily, or Synthroid (T-4 thyroid ) at a dosage of 100-200 mcg daily, starting low and progressing slowly up in dosage. Again, adequate circulatory thyroid hormone levels were necessary for GH and IGF-1 to reach full potential. (Ephedrine increased T-4 conversion to T-3)

**Frank weighed 275 LBS at this point, so this cycle was utilized as follows the first time:**

**Day #1-3**, 8 am -10 iu Humulin-R with 100 g of whole wheat flour and 50 g of Human Profile /9am-2 iu CH /50 mg Test. Susp. injected directly into the muscle trained the prior day/250 mg Cytadren 2-3xd or 250 mg Teslac 1xd and 10-20 mg Nolvadex 3xd (see example).

1 pm: 2iu CH. 3pm:

Training, one hour

4pm: 10 iu Humulin-R, 100 g whole wheat flour and 50 g Human Profile

5pm: 2iu GH injected into muscle trained at 3pm

8pm: 2iu GH injected into muscle trained at 3 pm/250 mg Cytadren or 20 mg Nolvadex/50 mg Test. Susp. injected with GH

**Day #4-7**. 9 am-20 mcg of IGF-1/50 mg Test. Susp. injected into muscle trained prior day/250 mg Cytadren or 250 mg Teslac, 10 mg Nolvadex (see example)

3pm: Training, 1 hour

4 pm: 1 2.5 mcg IGF-1/50 mg Test. Susp. injected into muscle trained at 3pm

8 pm: 250 mg Cytadren or 250 mg Teslac -20 mg Nolvadex.

And the rest of the cycle ran similar with a down-ramping schedule for Testosterone Suspension and an up-ramping schedule utilized for the administration of Winstrol Depot. The additional 250 mg of Cytadren was ingested about 4:30 pm..

At a later date when PGF-2 was layered into this cycle, 2 mg co-administered with each IGF-1 injection resulted in impressive gains due to synergy...of course. As mentioned prior, some athletes experienced severe muscle cramps and flu-like symptoms from PGF-2 use. Especially when stacked with AAS.

Any Max Androgen Phase or site injection protocol could have been structured as an All Out Psycho Layer Cycle simply by properly layering in the appropriate chemistry. Initially less was best. More was always added for future cycles only once results became inadequate.



## NUTRITION - *Mass Phase*

As any successful mass monster will tell you that there was no miracle muscle chemistry that induced their best significant gains in quality muscle mass without the inclusion of adequate nutrient/calorie intake to support maximum new growth. I am talking about serious amounts of macro-nutrients containing complete sources of micro-nutrients, and in the proper ratios.

### PROTEIN

Since lean muscle tissue is predominantly made of protein, we will start there. Protein is made up of several different amino acids in varying sequences and chains (As is lean muscle tissue). Therefore an athletes dietary protein sources contain the correct amino acids, and in the proper ratios if the goal is maximum potential growth rate.

Chemical Muscle Enhancement explained this in detail so I will get to the actual necessary amino acids, their proper ratios, and maximizing amounts quickly. However, it is absolutely paramount that the reader realizes that any shortage in any single or multiple amino acid(s) will break the chain in the protein synthesis equation.

I often explain the importance of using QUAASTs to evaluate circulatory amino acid deficiencies, but there is also a basic oral ingestion ratio that is quite close to the amino acid ratio profile of human muscle growth requirements.

#### (Expressed in %)

Alanine	2.5	**Leucine	9.0	Tyrosine	2.5
Arginine	9.0	*Lysine	10.0		
Aspartic acid	2.0	*Methionine	5.0		
Cysteine	0.5	*Phenylalanine	2.5		
Citrulline	1.5	Proline	2.0		
Glutamic acid	10.0	Serine	5.0		
Glutamine	10.0	*Threonine	4.0		
Glycine	3.5	*Tryptophane	3.0		
Histidine	2.0	Taurine	1.0		
**Isoleucine	6.0	**Valine	5.0		

\* Essential Amino Acid

\*\* Essential Branch Chain Amino Acid (BCAA)

(Bet that amino acid profile sent you running to read the label on your favorite "Big Nuts" protein powder).

The body can make or synthesize non-essential amino acids from other amino acids and proteins ingested (Or stored as muscle tissue), but it cannot synthesize essential or branch chain amino acids (BCAAs). The problem with that is the fact that the body uses dietary and circulatory proteins/amino acids for tissue repair and growth. But robs the body's protein based tissues (Uh, like muscle) for essential amino acids to utilize as raw materials for non-essential amino acid synthesis when a shortage occurs.

In addition, when a shortage of available glucose occurs (For energy needs) the body will convert BCAAs and glutamine into glucose. This is where the hype from supplement companies selling BCAAs came from.

As you know, the average individual's daily protein turn over rate (PTOR) is body weight x 1.818 expressed in grams. The PTOR will increase in favor of catabolism when dietary protein intake fails to provide adequate amounts or proper ratios of amino acids. (Yes, protein is still made up of amino acids)

So, it is logical that a 200 LB (small) bodybuilder would remain at a maximum anabolic or protein synthesis level if he/she were to consume their bodyweight x 1.818 (200 lbs x 1.818 =363.6g) in protein daily if the basic amino acid profile outlined on the prior page were met.

I can tell you this: I have clients who are over 300 LBS and growing, as well as those who are far smaller and not growing. Guess which ones adhere to this protein intake and profile schedule? The difference is flat amazing, and both groups follow the same protocols in all other areas. Those who finally wise up are pretty amazed when the lean mass gains possible result.

So how does a wanna-be beast eat to get this profile? Well, there are 2 ways. Consume at least half of your daily protein through protein shakes and divide the rest between meals consisting of red meat, fowl, fish, eggs, and low fat dairy products utilizing a 2-4 gram per pound of body weight daily schedule.

Or you can try to eat 400-1000 grams of protein daily supplied from meat, fish, chicken and eggs. Personally I have a thing about buffalo meat and lots of protein drinks. (Almost no bloating)

When looking at the amino acid profile shown on the prior pages, note the high percentages of L-Arginine. Arginine is used as an immune stimulator and is required for tissue regeneration. Though the body can make this non-essential amino acid, it cannot make it in great enough quantities to keep up with a hard-core athlete's training schedule. Worse yet is the fact that the body will rob other amino acids to create/synthesize Arginine... which leaves a shortage of the necessary amino acid profile needed for optimum repair and growth.

Low Arginine levels will result in poor recovery due to a break down in the protein synthesis chain, low sperm counts, low nitric oxide production, and impaired GH production. I have noted significant improvements in post work-out recovery, training pumps, and sex drive, (ya, I do like it) when 0.5-1 gram of Arginine per 20 LBS of bodyweight is ingested daily by athletes.

## **CARBOHYDRATES**

Carbohydrates are said to be the main source of energy providing nutrients. Complex and low glycemic sources such as rice, grains, sweet potatoes, whole wheat, pasta, and veggies are the better choices. The slower burning/low glycemic carbs are best because they provide continuous fuel for training and healing as well as the raw material for replenishment of liver and muscle glycogen stores.

The three slowest burning carb sources are Maltodextrin 20 (D.E. 20), brown rice, and oat fiber. A hard-core mass monster needs 1-3g of carbs per pound of bodyweight daily to grow at maximum rates. (Those whom replace carbs with gluconeogenic amino acids obviously do not)

Low carb intake will result in poor muscular pumps and a lack of energy for high intensity or volume oriented training. Carbs also aid in preventing protein from being utilized as an energy source, so they do have an anticatabolic quality.

## **FATS**

Fats are necessary for optimum gains and health. Low ingestion/absorption levels of essential fatty acids (EFAs) results in low prostaglandin production. One very important prostaglandin is PGF-2. I wrote at length upon this in Chemical Muscle-The BDR, but let me say a full 20-40 % of daily calories should come from fats (Especially EFAs). Remember; at 9 calories per gram, fats are also a valuable energy source, and therefore possess anti-catabolic properties.

## NUTRITION "KISS"

"Keep it simple stupid" is the motto I use concerning nutrition. Follow a diet protocol that provides 2-4 g protein/1-3 g complex carbs per pound of bodyweight daily divided into 6-10 meals while avoiding excessive fat intake (normally 20-40%) and growth will happen. But there are ways to make it easier and more affordable.

By the way, yes KISS strategy often must exceed 25 calories per pound of bodyweight for some, while others would get very fat ingesting this amount of calories daily. Adjusting the carbs is the key.

### (Mass Phase Continued)

Most serious athletes own a Nutritional Almanac and a gram scale, or should. Using an Almanac, look up the food source, divide or multiply protein, carbs, and fats listed by the amounts necessary, weigh it and eat. But this too can be made easier and more beneficial.

Meats provide 6-7 g of protein per ounce, carbs are weighed by the gram only if dry such as grains, (rice oats, etc) and pasta, and let fats fall where they may (be reasonable). Add the essential fatty acid mixture described earlier.

I buy a great deal of whole grain bread at the day old bake store. There is one in most areas. I buy wholesale meat and cut my own, usually a whole round (the hind leg and ass of a cow), and cut steaks or stew meat. Veggies, in some areas, can be bought cheaply at roadside farmer stands and frozen in smaller parcels.

I usually have clients eat 4 meals per day, and drink 2-6 more. The protein shakes contain 30-50 grams of Human Profile or whey/ casein protein powder, and D.E. 20 or whole wheat flour for a carb source in a ratio/amount appropriate to their carb requirements.

What the hell is wheat flour? Whole wheat flour is what whole wheat bread and pasta is made from. It provides about 130 calories and 23-27 g of carbs per ounce. It is also a cheap and a major source of glutamine peptides. In fact, some special whole wheat flours contain as much as 50% glutamine peptides by weight, though common available sources contain about 20%. It is very cheap, by the pound, at most health food stores and has little flavor.

Whole wheat flour can be very useful for ectomorphs and mesomorphs...and hazardous to some endomorphs if they accumulate fat tissue easily. The only problem occurs when shakes are made in advance. The flour tends to thicken and tries to become bread in the shaker. So mixing and drinking immediately is necessary. Some individuals have trouble eating/drinking this amount of calories daily. A quick fix is to

leave a shake by the toilet (on the counter, preferably), then mix and drink it when you get up at night to take a leak.

**WARNING: Don't try to mix and pee at the same time. (However, my spouse swears this is an interesting sight.)**

It is paramount to totally prepare for every aspect of a protocol before even the first rep or set is done. Before an athlete begins any protocol all components necessary for the best results must be stock piled. This means a plan is written and itemized before beginning a protocol.

Personally I purchased all I need and in quantities that slightly exceeds planned needs a month ahead. This greatly aids in mentally ramping-up for each stage of success. It also makes success much less stressful to achieve and total focus on the task much easier.

**Note:** Undigested food packed around the colon allows for poor nutrient absorption. An average mature adult has 8-25LBS of undigested food in his/her colon. Colonic irrigation solves this (Ouch!)

## **Is a high protein diet unhealthy?**

Researchers at the University of Mississippi and Wake Forest University in North Carolina tested the theory that high protein diets can lead to high blood pressure and cardiac risk. 13,935 adults ranging in age from 45-66 were monitored from 1987-1995.

The results showed that both animal and total protein intakes were inversely linked to diastolic and systolic blood pressure. Yet vegetable proteins had no effect on either blood pressure measurements. The study's conclusion? " Higher protein intakes are related to lower blood pressure". Ya, we knew that.

Also the superiority of animal protein over vegetable protein (again) shines through. It seems obvious that those who often write about how "...high protein diets can lead to high blood pressure due to harmful effects of protein metabolites on particular kidney structures" will need to cry new lines of B.S. Cool!

But of course, they will still whine for years anyway.

## **NUTRITION - Contest Prep**

***"It seems that there is almost a mystical quality to the transition a well trained physique under goes during contest prep. The human body is the most beautiful art form... which can illicit the most profound emotional response we know of. But the truth of the matter is that there really is no mystery to how it is done." Coach***

There were simple goals to contest prep. Obviously coming in hard, dry, and tight was paramount. This took a well thought out plan and the heart to follow through with it.

### **Frank's Contest Prep Goals:**

- Maintain or increase lean muscle mass.
- Increase vascularity.
- Burn body fat stores.
- Improve separation and striation of musculature.
- Monitor and adjust symmetry.

Seems almost stupid to read, doesn't it? You would be quite surprised how many hard-core bodybuilders knew these goals but failed to achieve them. Many simply decreased calories, added aerobics (yes, I said the "A" word) and hoped for the best.

Worse yet, the new trend was becoming symmetry adjustments made at the last moment with several hundred ML of Synthol (Oh, but that is against the rules now!). Usually this results in a ripped physique with obvious oil bags in certain areas. How many times have we seen a truly gifted and strong-hearted 280 lb beast diet to 215-225 LBS only to appear soft and stringy?

### **DIET STRATEGY**

It seems an obvious point of common sense that muscle-changing chemistry played a major role in the contest prep plan for beasts, but diet was even far more important. As example, the commonly endorsed diet approach focusing upon an ever-decreasing daily calorie count was the surest path to muscle loss and failure.

After a 10-14 day lay-off from training and a month break total from all chemistry, Frank was ready to begin. About 4 weeks prior to his diet or contest prep period, Frank's daily calorie count was reduced about 500 calories per day in weekly intervals. So Frank began at 6000 calories daily. Week #1 equaled 5500 calories daily, week #2 equaled 5000 calories daily, etc., etc..

## ...AND THE SCULPTING BEGAN

**Body Weight:** 308 LB, 12 % bodyfat, Lean Mass Weight 271 LBS.

**Measurements:** Chest 59.2 ", Arms 23.2 ", Legs 33.25 ", Waist 36.5".

Frank has made excellent progress and holds more adipose tissue near his waist with decent abs still showing, oddly enough. Frank has 37 LBS of fat and about 15 LBS of water to lose. The latter will be lost, in part, the last 3 days prior to contest.

Realistically speaking, Frank will not lose the entire 37 LBS of bodyfat. But the weight loss is a base for this diet. If all goes perfect, Frank will weigh 250-256 LBS when he steps on stage and be about 4% body fat. Of course, very freaky also. Our goal will be to bring him in at over 270 LBS contest ready next year, but lets get this one done with first.

*If* Frank were a natural bodybuilder, the diet parameters would differ from a chemically enhanced athlete. He would begin by determining his basal metabolic rate (BMR). BMR is the number of calories a body requires to maintain homeostasis at rest.

One method of dieting assumes a BMR of 6.5 calories per pound of lean mass weight. For Frank this would be 271 LBS x 6.5 cal = 1761.5 calories required at rest. Remember, "at rest" means the number calories metabolically active tissue burns daily with zero movement. We do not count fat as par of the equation because fat does not burn calories.

Next, I would account for Frank's calories burned daily due to activity. A weight training work-out burns between 250-450 calories, depending on training intensity and how long each work-out last.

There are also normal daily activities to account for since they too induce a calorie expenditure. As an example, a couch potato may only need an additional 300 calories daily above his/her BMR, but an active individual may require over 750 additional calories above their BMR to maintain homeostasis. These additional calories (required for normal daily activities) are where Frank would initially create a calorie deficit. The math on that is pretty simple... BMR plus the high intensity work-out calories required would equal 2211 calories daily (1761c for BMR + 450c for each work-out=2211 calories).

So utilizing a 3 day staggered calorie count around this base figure would allow Frank a stagger of day #1 -2500c; day#2-2350c; day #3 2200c, and then repeat. Since Frank is moderately active, he utilizes about 500 calories daily for normal activities, so his net calorie deficit would be about 2500 calories total every 7 days.

This would result in a "fat" loss of about 1 LB per week or about 12-17+ LBS over his 12 week contest prep period. If he had added a thermogenic product such as any ephedrine based product, the result would have been about an additional 1-1.5 LBS of "fat" loss per week.

Add some form of interval-anaerobic /aerobic activity 5 times per week for 20-30 minutes each session and Frank would be ready in 12 weeks easy.

I cannot stress enough the importance of **staggered calorie** counts. This method prevents the body from adapting too quickly to the diet/calorie deficit protocol. Normally a set calorie deficit will cause a decrease in endogenous thyroid hormone activity. This in turn quickly negatively alters the athlete's BMR.

Obviously, daily calorie counts would be adjusted if fat loss were not within diet protocol parameters. During the first 2 weeks actual weight loss would exceed the 2-3 LB parameter simply due to combined fat and water loss. The body stores 2.5-3 grams of water per gram of glycogen (carbs) stored. So for a natural athlete (which Frank certainly was not) more than 2-3 LBS of actual weight loss weekly is acceptable up to week #3.

***However, Frank was chemically enhanced.*** I will discuss macronutrient parameters later, which have applied to actual natural or chemically enhanced athletes. For now, let's admit that the chemically enhanced have a serious advantage over actual naturals (Yes, there really are some left out there).

By the way, you should realize by now, that basic fat loss occurs at under 10 calories per pound of body weight daily. I just thought you might want the actual math practice.

Many bodybuilders employed drugs that significantly effect diet results. Thermogenics increase the amount of fat calories used to produce heat, thyroid hormones increase BMR, and GH increases the percentage of fat calories utilized for total calorie expenditure.

Add to this the fact that some AAS aid in lean mass retention, or even increase lean mass during contest prep, and you will realize why there are so many pro's on freak status. (Natural my ass!)

How often have you the reader, read that a pro diets on 4500 calories daily? Do the math and you will realize most natural athletes would gain, not lose weight with this daily calorie count. Frank, of course, dieted nicely at higher calories also.

The first priority in a contest prep diet must be protein. After all, the lean mass Frank later exhibited was predominately protein, right? Researchers have proven fat loss will occur if there is an existing calorie deficit.



However, science has also proven that during diet restricted periods, the best lean muscle mass retention requires at least 1 gram of whole complete protein per pound of lean bodyweight daily. For a natural competitor, this would come to 271 grams of good protein daily, resulting in 1084 calories from protein intake alone. The macronutrient percentages would therefore break down like this:

Day #1: 30% protein, 60% carbs and 10% fats.

Day #2: 35% protein, 55 % carbs, and 10 % fats.

Day #3: 45 % protein, 45% carbs, and 10 % fats.

The daily protein intake would remain constant at 271 grams.

## **How Much Protein?**

However, chemically enhanced beasts that utilized drugs that influenced the PTOR made the best progress when they used a daily protein intake formula of lean mass weight in pounds multiplied by 1.818 as a minimum guide-line. In Frank's case, this amounted to 492.6 grams of protein daily (271 LBS x 1.818 =492.678 g), or about 2000 calories from complete protein alone daily.

## **Thyroid Hormones Alter Protein Intake**

Depending on natural or endogenous T-4/T-3 thyroid hormone production, an exogenous dosage of T-4/T-3 usually altered BMR and macronutrient turn-over significantly. This of course was difficult to account for mathematically without a hormone profile (which provided information about a beast's normal thyroid hormone production). But the average individuals daily endogenous T-3 production/release is around 26 mcg daily. So 50-100 mcg of Cytomel administered daily roughly resulted in 1 25-200% above normal BMR as well as a corresponding increase in the PTOR.

Obviously it would have been quite counter productive for Frank to ingest less daily protein than his daily PTOR required. (Gee, ya think?) This means that, in many cases, a beast needed to increase total protein intake 25-100% and reduce carbohydrate intake to maintain correct daily calorie intake as a means of keeping up with increased PTOR.

## Carbohydrates and Fats...The Easy Part

The necessary daily carb intake count equaled Frank's weight training calorie expenditure, or about 450 calories (112.5 g) from carbs. His fat intake daily remained at 10% of total daily calories.

This meant that his lowest calorie count day was about 2700 calories. Utilizing this base information, Frank's calorie stagger looked like this:

Day #1: 3245 calories  
Day #2: 2971 calories  
Day #3: 2700 calories

This calorie stagger was based upon Frank's lean mass weight. Day #1 allowed for 2 additional calories per pound of bodyweight, and Day #2 allowed for one additional calorie per pound daily. As I said earlier, this was a "base" calorie count. It sometimes needed to be adjusted when fat loss/weight loss parameters were not between 2-3 pounds weekly.

If the body is unable to utilize or convert enough fat into energy, once muscle and liver glycogen stores from carbs are depleted, the body will strip proteins/amino acids (like uh, from muscle) of their nitrogen components and use the calorie source for energy. This is called deamination.

It is also very bad when lean mass retention is the goal. When adequate protein intake is provided the body will use incoming amino acids from protein to create glucose for glycogen synthesis by a process called gluconeogenesis. So it was paramount that an all beasts adjusted carb calorie intake to maintain a weight loss of 2-3 pounds per week. Usually I adjusted calories by adding or subtracting 200 calories to each of the three days of the staggered count.

You probably would be surprised how many individuals there are that simplify this further by eating only tuna and skinless chicken breast with steamed brown rice and a few spoonfuls of extra virgin olive oil. Hey, it works pretty well if they add a good clean protein powder or Human Profile. They would do better however if they traded some of that rice for vegetables, MCT oil and CLA.

### **One Size Does Not Fit All (But it does work for most)**

For many, the sudden decrease in calories was just a little too difficult to endure. Personally, I dropped weight too quickly utilizing the prior diet outline. (Most people do well with it) If this was the situation with Frank, he would have began his pre-contest diet phase 16 or even 20 weeks out with an initial daily calorie count of 6000 calorie

Daily protein intake would have remained constant at 1.818 grams per pound of lean mass weight and fat would have remained at 10 % of total daily calories. However, carbohydrate calories would have been decreased about 250 calories per day each week still utilizing the necessary 3-day calorie stagger. So beginning 20 weeks out, the stagger would have looked like this:

Week #20 = Day #1 -6000c, Day #2-5750c, Day #3-5500c and repeat for Day #4-6. Day#7-5750c. Week #19= Day #1 -5750c, Day#2-5500c, Day #3-5250c, Day #3-5000c and repeat for Day #4-6. Day #7-5250c.

This 250 calorie decrease would have continued until a calorie stagger for Day #1-3245c, Day #2-2971 c, Day #3-2700c was accomplished. At that point the calorie count and stagger would remain constant.

\*C = Calories

Obviously, calorie intake would be adjusted to meet an "averaged" 2-3 pound weekly weight loss after that. For many athletes medium chain triglyceride (MCT) oil was an excellent calorie option when making calorie adjustments since it burns fairly clean. The body actually has difficulty storing it as fat. And it provides good energy as a result. If an athlete does not have enough energy to train intensely, the diet results will be poor.

Remember; calorie deficits are relative, not absolute!

How often did Frank eat? 6-12 times daily. Each meal, whether liquid or solid, provided no more than 500 calories. The body digests food better when ingested in smaller portions. Also each time calories are introduced, the body increases metabolism. I have had several clients who simply ate very small liquid or solid meals every hour through out the day. It took the edge off hunger.

Personally I ate every 2 hours once my daily calorie count fell to and below 4000 calories. It really was pretty simple to diet successfully without the stress and attitude problems.

## INSULIN AND CARBOHYDRATES

Many so-called experts believe that carbohydrates are the enemy of successful fat loss. They claim that eating carbs make you fat. This is based upon the theory that high carb intake diets cause high insulin release levels, and that high insulin release levels lead to high fat storage levels.

They further the claim of "evil insulin" by pointing out the fact that insulin is the body's main storage hormone. And after all, fat is the body's main energy storage place. For the most part, this is total bull-shit with the exception of *simple sugars*.

The fact is that during a calorie deficit period (any period that less calories are ingested than burned) the body has no inclination toward fat storage from any macronutrient.

Think about that for a second. How can the body "store" calories if it is currently depleting its reserves? When it is an issue of fat loss or gain, calorie intake count vs. calorie expenditure count is the most important determining factor.

But for those doubters (and nutritionist/dietitians) who scoff at this, read the Study "**Differences in insulin resistance do not predict weight loss in response to hypocaloric diets in healthy obese women**" McLaughlin, T., et al. (1999) **Journal of Clinical Endocrine Metabolism 84:578-581**. There are several other studies to support the facts also. But as most bodybuilders are aware, most nutritionist and dietitians still believe that the RDA for protein is all of the protein anyone will ever need for maximum athletic progress.

## PROTEIN SOURCES

Protein source is of unique importance to muscle-maniacs. The necessity for complete proteins/correct amino acid profile has been discussed prior, so let's move on to whole foods. Lean meats are the best whole food protein sources. What is lean?

Beef tender loin, London broil, beef filet, flank steak, veal and pork tender loin, skinless chicken or turkey breast, water packet tuna, and egg whites. Milk is bad during diet phases since it is the single most hyperallergenic food I know of.

Most athletes appear leaner after only 2 weeks without milk products. However, if an athlete has access to fresh human milk during a contest prep phase... (Yes, I do know of a few bizarre tales)

## CARBOHYDRATE SOURCES

All carbs are not created equal. ***Simple sugars and other high glycemic index carbohydrates will fail to provide a sustained energy level.*** The spiking effect from

frequent ingestion of simple carbohydrates results in an increase in the fat storage enzymes. For this reason there is a repartitioning effect for nutrients away from muscle and toward fat stores.

Fructose is bad. I could write a book on the negative effects caused by high fructose diets including an increase in the aging process and free radical problems. The better choices are slow burning complex carbohydrates such as brown rice, multi-grain breads, and yams (yes, they do contain some fructose, but it is mostly mediated by other complex carbohydrates contained as well).

Of course D.E. 20 would fall into this category, but during restricted diet phases liquid carbohydrates tend to leave an athlete feeling calorie deprived. Watch out for any carb source that has more than a 20% calorie content from fructose.

Lots of vegetables are a must. The lack of adequate roughage intake will result in a clogged GI track... which means poor nutrient absorption. (Most people do not like daily enemas to clear such problems. Go figure. Yikes!!)

### **FAT SOURCES**

Lean meats will usually come close to providing the 10% daily fat intake for the contest prep period. However, it just is not healthy to eliminate essential fats that provide EFA 's. Extra virgin olive oil, canola oil, evening primrose oil as well as fish or flax oil.

Like I said. Keep it simple.

# PHARMACOLOGICAL FAT BURNING

## (A BRIEF SCIENCE LESSON)

Adenosinetriphosphate (ATP) is more than the chemical our bodies utilizes to fuel muscular contractions and nerve impulses. Obviously we would die without it. However, our ATP level and function also dictates how muscular and fat we are to a great extent.

As we know, the body needs a constant supply of ATP. When ATP is utilized as energy, it loses a phosphate molecule which creates Adenosine Diphosphate (ADP). ADP turns to creatine phosphate (CP) stores for the missing phosphate molecule and ATP is again manufactured. This is of course why creatine products aid in strength and lean tissue gains.

**Note:** HM Gear has a great "sterile oral" super ATP product I will try to discuss later.

The body can produce ATP from proteins, glucose, or fats. This is possible due to cellular mitochondria. When cellular ATP stores are low, such as after a heavy set of squats, and cellular "ADP" levels rise, the cells must undergo oxidative phosphorylation and electron transfer to restore ATP.

Oxidative phosphorylation is the process of adding a phosphate group to ADP to make ATP. Electron transfer is the process or way in which this happens. Mitochondria pump hydrogen ions from the interior to the exterior of our cells. This creates an imbalance where there are a greater number of hydrogen ions on the outside of the mitochondria which results in the pairing of ADP with an extra phosphate group. Shazam!: We have ATP and cellular energy, which allows us to live and train another day.

The electrons for electron transfer come from NADH, which is made from macronutrients. As you know, macronutrients are protein, carbohydrates, and fats. Which obviously become circulatory cell food: amino acids, glucose/glycogen, and fatty acids. So the series goes like this:

1. NADH donates electrons to the mitochondria for transfer.
2. The electron transfer creates a hydrogen ion imbalance.
3. The imbalance powers the "coupling" of ADP and a phosphate group resulting in ATP. (Again; Shazam!)

No doubt you remember the term "coupling" during the ADP/ATP process of oxidative phosphorylation. Well, "uncoupling" is in simple terms a method of making this whole process harder. In the case of oxidative phosphorylation, it is a matter of making the mitochondria work very hard to create the hydrogen ion imbalance.

The little goodies responsible for this uncoupling are uncoupling proteins, which are found in both muscle cells and brown fat cells. When these uncoupling proteins make their way into the mitochondria, the resulting reaction is that the mitochondria must work overtime trying to pump hydrogen ions from the inside to the outside of the cell. This is due to the effect of uncoupling proteins allowing the hydrogen ions to migrate back in. Since the process wasted the electrons from NADH and no ATP was made, the process has to start all over again.

Uncoupling proteins in muscle cells act to regulate free fatty acids in the myocytes. And uncoupling proteins in brown fat has a regulatory effect upon body temperature/ thermogenesis. The result of this uncoupling is that the mitochondria uses up much more fatty acids, and to a lesser extent, more glucose to normalize and regenerate ATP levels. When all this takes place, obviously an athlete burns more calories (most of which come from fat stores) and becomes much leaner.

The chemically enhanced beasts had utilized many drugs that influence the uncoupling process: DNP, Thyroid hormones T-4/T-3, amphetamines, ephedrine, norephedrine (phenylpropanolamine) and caffeine to name a few. Before I go on, let me say that amphetamines are a really bad idea under any circumstances. They are very illegal, they sucked at burning fat, and they screwed up people's minds.

On top of this, they were dirty. Most black-market "meth" was and is manufactured using red phosphorus as part of the process. This is not properly removed or purified at any point in the process. Red phosphorous is cancer causing and destroys immune function factors! Users commonly get weird sores and seem to catch every cold and flu they come within 50 yards of. Picture that on contest day!

### **EPHEDRINE-NOREPHEDRINE-CLENBUTEROL**

All three of these compounds are called beta -agonists, which means that they stimulate beta receptor sub types 1,2, and 3. Clenbuterol is a beta-2 receptor specific agonist. This means that its fat burning effect is due to specific stimulation of beta-2 receptors. Ephedrine is a non-specific beta-receptor agonist. Meaning its effects are due to stimulation of beta 1,2,and 3 receptors. Norephedrine (phenylpropanolamine) is a non-specific beta-receptor agonist as well, but seems to effect beta 2 receptors in a slightly different way.

All beta agonist affect the fat burning process through mild thermogenesis, which of course involves the process of uncoupling.

Clenbuterol administration tended to cause down-regulation of beta-2 receptor activity. Since beta 2 receptors are a major regulator of the fat break down (lipolysis) process, this sucked.

Continuous administration of Clenbuterol allowed the drug to lose its CNS effectiveness after only a few weeks. Regardless of dosage. For this reason, a 2 day on-2 day off protocol was necessary during Clenbuterol use. This approach slowed the adaptive down-regulating response by beta-2 receptors.

Please be patient. I will explain all the "receptor" stuff in a few minutes.

Unlike Clenbuterol, Ephedrine and Norephedrine did not cause an excessive adaptive response and therefore continued to effect thermalgenic activity for months.

Both induce the release of "Noradrenaline", which in turn stimulates all three beta receptor sub types, and both can stimulate the same receptor-sites directly to some degree. Ephedrine products induce the thermalgenic effect by stimulation of all 3 receptor sub types, but 35-40 % of their stimulatory effects are upon type 3 beta receptors.

This is significant because the response governing activity down-regulation of beta 2 receptors is not possible with beta 3 receptors. So ephedrine products have potential long term effectiveness. Simple? Beta-3 receptor stimulation increases the break down of fats. This also results in an increase in uncoupling protein levels. It should be noted that Ephedrine products increase the conversion of T-4 to T-3 by the liver. (Yup!!)

### **CAFFEINE**

When beta agonist compounds are utilized, the body releases adrenaline and nor-adrenaline, which is secreted due to stimulation of the sympathetic nerves. Ultimately, the body has an adaptive response which regulates the long term activity of beta agonist and therefore their thermalgenic value (fat burning effect).

Methylxanthines such as caffeine, enprofylline, forskolin, and theophylline, block or inhibit the body's adaptive response to a respectable degree. Co-administration of methylxanthines with Clenbuterol, ephedrine or norephedrine resulted in a longer and more potent effect/result period. Normally the most effective ratio of ephedrine or norephedrine to caffeine was about 1 to 10. (20 mg of Ephedrine to 200 mg of caffeine.) The chemistry behind this is quite complex, but the basic action is this:



## More Science Geek Stuff

Beta agonists modulate their fat break-down signal through an energy precursor within the cell called cyclic adenosine monophosphate (cAMP).

Compounds called phosphodiesterases counter-act this by breaking down cAMP and the fat loss signal decreases or stops. Methylxanthines either inhibit or block phosphodiesterase which is the enzyme that degrades the phosphate in cAMP. Therefore the fat burning effect/thermogenic value of the chosen beta-agonist signal continues.... (Sorry, but you made me do the science geek thing.)

### RECEPTORS

Most cells and organs in the body possess a distribution of adrenergic receptors. These receptors have sub types: ALFA sub-1, 2, and beta sub-1, 2, 3 and 4. The receptors we are interested in initially for fat loss/thermogenesis are alfa-sub-2, and beta-sub-2 and 3. These are the adrenergic receptors having the greatest initial effect upon fat loss.

Stimulation of beta-sub-2 receptors creates an anti-catabolic response meaning protein sparing. Unfortunately beta-sub-2 receptors down-regulate after 2-3 weeks of continuous stimulation. Action/Reaction, remember?

Stimulation of Alfa-sub-2 receptors blocks the mobilization of fat stores. Blocking these receptors with Yohimbine increases fat expenditure.

**Interesting fact:** Women have greater numbers of Alfa-sub-2 receptor (and estrogen receptors) in their lower bodies when compared to males. Hey, they bit the apple, and I didn't design the receptor distribution ratio. This is why women have so much trouble losing fat in these areas. (Which really sucks)

If I had designed the receptor ratio and distribution, the women of this world would have significantly higher ratios of Alfa-sub-2 adrenergic and estrogen receptors in the breast with few anywhere else. I would also greatly reduce 16-alpha-hydroxyestrone production so women could have great racks, lower incidence of breast cancer, and remain lean while eating cookies and ice cream. But that is another story...and lots of hate mail, no doubt.

There are a few compounds that block Alfa-sub-2 receptors (antagonists) and therefore prevent the fat loss inhibiting effect. Pure Yohimbine is a good example. It actually inhibits the negative feed-back loop that inhibits continued fat loss. Unfortunately, the effects of Yohimbine are mitigated by insulin release (or administration). This means that yohimbine is only effective as an Alfa-sub-2 receptor antagonist if the athlete utilizes it in a fasted state (like before breakfast).

## Chiseled SGR (Site Reducing Gel)

As stated earlier in this book, AGR Nutrition ([www.agrnutrition.com](http://www.agrnutrition.com)) has a very effective fat-burning topical product called "Chiseled SGR (Site Reducing Gel)", which contains pure Yohimbine and other goodies that prevent insulin from getting in the way of Yohimbine doing its job.

It is in a gel form intended for topical site-specific application with an amazing absorption rate. Most note benefits in the first few days to a week of application. It is actually so effective that users are suggested to apply the product to only one site at a time (abs, lower back etc) to prevent an over accumulation of newly freed fatty acids from entering the circulatory system too quickly. (I truly enjoyed working on this one)

As stated prior, ephedrine products modulate 35-40% of their fat burning/thermalgenic effect through activation of beta-sub-3 receptors. However, to a lesser degree, they do stimulate thermalgenesis through activation of the beta-sub-1 receptors. Since beta-sub-2 receptor activity significantly down-regulates after only a few weeks of continuous stimulation, the question of "how long before beta-sub-land 3 receptors down-regulate activity" should come to mind.

After about four weeks of ephedrine/caffeine use, thyroid hormone levels increase. At about 12 weeks of use thyroid hormone levels decrease below normal levels. As stated prior, Ephedrine products increase T-4 conversion to the more potent T-3 thyroid hormone. Unfortunately, this too has a negative feed-back loop that kicks in at about 12 weeks of continuous ephedrine use.

Guess what? We have **beta-sub-4** Andrenergic receptors which **over-ride** the rest and continue the fat burning process even with lower circulatory thyroid hormone levels. In fact, the thermalgenic effects of an ephedrine /caffeine stack is better at 12 weeks of use and remains significantly effective for up to 50 weeks.

This is due to the body's ability to increase the number of beta-sub-3 and 4 receptors in response to down regulation of beta -sub-1 and 2 receptors. Cool! By the way, the enzyme that converts T-4 to T-3 thyroid hormone is 5-deiodinase. Low selenium intake greatly reduces this enzyme's effects.

**Note:** most athletes experienced a 3-10% gain in strength and anaerobic capacity when using beta agonist. This resulted in more lean mass gains due to greater muscle fiber recruitment... potentially.

There are two readily recognized thyroid hormones. T-4 (thyroxine) and T-3 (triiodothyronine). In response to TSH, (Thyroid Stimulating Hormone) the thyroid gland releases T-4 hormone, and to a lesser degree T-3 hormone.

Daily T-4 production is normally about 76 mcg, and T-3 production is about 26 mcg daily. The majority of T-3 hormone production is due to the conversion of T-4 to T-3 by the enzyme 5-deiodinase primarily in the liver and kidneys. T-3 is about 5-10 times more active or potent (depending upon which research paper the statistic originated from) than T-4.

Remember that only unbound or free testosterone can initiate a reaction? Well about 0.02 % of circulatory T-4 is unbound and about 0.30% of T-3 is unbound or free. Only free or unbound thyroid hormones can merge with and activate thyroid hormone receptor sites.

Thyroid hormones increase the levels of uncoupling proteins. They also increase ATP turn over by stimulating enzymes that effect nerve impulse conduction. A great deal of research supports the idea that thyroid hormone receptor-sites are directly linked to the genes that regulate the amount of uncoupling proteins produced. It should seem evident that thyroid hormones played a significant role in a chemically enhanced beast's contest prep in most cases.

My opinion was that a few weeks of exogenous thyroid hormone use was far healthier than long periods of calorie restriction. But that was only my opinion and not a recommendation. As stated prior, beta agonist aid in the conversion of T-4 to T-3 so there was an existing synergy when referring to fat loss or lean mass gains.

## CONTEST PREP Chemistry

The goals of contest prep obviously included lean mass retention or addition. How many top competitors have you seen doing 60-90 minutes of aerobics twice daily and train like animals during contest prep yet retain incredible lean mass? Well, for the average actual-natural, this would result in serious over training and lean mass tissue loss at a very high rate.

Anyone who had administered 2-4 grams of androgens weekly did quite well as long as their diet was somewhat appropriate. But there were protocols that provided Frank with far superior health and results. These protocols always assured necessary factors were met. All of these drugs were discussed thoroughly in Chemical Muscle Enhancement, however a brief recap maybe needed.

**Testosterone:** During contest prep, testosterone assured a high androgen level that resulted in significant training intensity and recovery. Testosterone also inhibits the enzyme that allows fat cell fatty acid deposits (like love-handles). As long as estrogen control was adequate, testosterone aided in providing a superior hardness to musculature while stimulating good strength gains for heavy training and necessary maximum muscle fiber stimulation.

Most AAS did increase red blood cell count and therefore vascularity. Reasonable increases in blood pressure from androgenic compounds helped to finish "the look".

**High Anabolic Steroids:** A highly anabolic steroid provided increased muscle protein synthesis while reducing the catabolic ratio. **Some** AAS worked well in a calorie deficit environment. This was paramount for lean mass retention or increase. In order, those that worked best to least were: Parabolan, Finabolan, Masteron, Primobolan Depot, Deca Durabolin, Winstrol Depot, and Equipoise.

However, some combinations created a synergy that resulted in improved muscle separation/striation and lean mass tissue growth or retention. As example, Winstrol Depot, utilized as a site-injection protocol, with Testosterone Propionate has been the cause of many deep pec and delt striations and separations.

**Growth Hormone:** When properly layered in during contest prep, GH brought a new level of quality to a physique. It increased the use of fat stores as energy, acted both anabolically and as an anti-catabolic. CH also aided in injury prevention.

Research has shown that as little as 2 i.u. daily of CH can reduce fat stores about 35% when utilized with a calorie restricted diet. The diet need only equal "normal" daily calorie expenditures. There is some research that suggests that GH administration increases endogenous testosterone synthesis. But to say that this alone would have HPTA regenerative qualities is likely a stretch.

There is a notable difference between the methodology of GH use in relation to mass gain and contest prep. This is due to Direct and Indirect affects.

GH refers to Growth Hormone, which is a sort of master substance produced by the pituitary gland. It has somewhat of a prohormone quality to it in that it triggers several other active hormones to be produced (indirect effect) while maintaining the ability to trigger metabolic activities (direct effect) itself.

### **Indirect**

GH initiates the process of IGF-1 synthesis in the liver and other tissues as well as having an effect upon the synthesis of the other 7 growth factors as well. IGF-1 is an amazingly powerful anabolic with the ability to increase satellite cell count and inclusion for an actual increased muscle cell/fiber count. Yup, brand new muscle cells and fibers to add to the muscle mass.

### **Direct**

For the most part, GH has a direct effect most notable upon adipose (fat) tissue. A fragment of the GH molecule stimulates the B-3 receptors on fat cells. This then triggers lipolysis (fat burning) while simultaneously blocking fat storage.

*\*Researchers are currently working on creating the unique GH fragment committed to stimulation of the B-3 receptors on adipose tissue for a fat loss drug. With some luck it will clear FDA approval by 2008.*

## **GH and Mass**

During any type of mass phase there is a large increase in calorie intake to facilitate new growth. When GH is administered during high calorie periods there is little in the way of increased fat accumulation as compared to protocols for mass that lack the drug.

This is due to the fact that, yes, GH helps to block fat accumulation in this environ, but it fails to significantly increase fat expenditure. This is due to the fact that food increases insulin release that in turn blocks fatty acid release from fat cell.

## **GH and Contest Prep**

During contest preparation total daily insulin release decreases due to a decrease in calorie intake. As a result there is a corresponding decrease in blood glucose levels that predicts these low insulin periods. As a rule CH administration during a period in which a glucometer reading of below 65 exists results in a significant increase in fat expenditure.

Many have followed this practice and followed with cardio sessions to heighten the effect. Interesting, huh? It has been a wise choice to keep glucose tabs nearby incase hypoglycemia sets in. (It would have been really embarrassing to pass-out on a tread mill and have it toss a beast to the carpet)

**PGF-2/IGF-1:** Obviously IGF-1 was both anabolic and anti-catabolic. However when paired or stacked with PGF-2, a synergistic response was realized. The combination was perfect for symmetry adjustments during pre-contest diets simply because both were some-what site-specific in action.

As Frank now realized, different muscles have a unique and different PTOR, which means such areas as his arms lose mass more quickly than others. The PGF-2/IGF-1 stack significantly increased anabolism in those areas either as he dieted down or during the last 3 weeks before his contest.

**Thyroid Hormone:** T-4 and T-3 thyroid hormones increased the BMR and the PTOR while significantly increasing nutrient absorption and utilization. They also had a very notable synergistic response with AAS and GH/IGF-1. Since T-4 and T-3 increased BMR and a high protein diet was utilized with anabolic chemistry, the result was faster fat loss (also better lean mass retention and growth).

**Thermalgenics:** Thermalgenics increased the number of calories burned as heat expenditure. Clenbuterol and Ephedrine also had anti-catabolic qualities that also acted synergistically with GH. Caffeine greatly extended the time period for fat burning effectiveness of most thermalgenics.

**Anti-Estrogens:** Estrogen control was absolutely necessary during contest prep. Any increase in estrogen activity could have lead to stubborn fat deposits (or even female pattern fat deposits), gyno, and serious water retention. The reader knows well the fact that estrogen leads to a negative feed-back loop that inhibits HPTA function.

However, since contest prep cycles lasted so long, HPTA regeneration post-cycle was quite necessary anyway. But why would we have added to the problem to begin with? No competitor came in truly hard without estrogen suppression. **FACT!!**

**Cortisol Suppression:** During calorie deficit periods, the body increases catabolic hormone production. Cortisol, the body's main catabolic hormone increases protein based tissue wasting by triggering the release of amino acids from muscle cells. The amino acids are stripped of their nitrogen components and then utilized as an energy source.

When cortisol suppression was utilized the body increased the use of fats for fuel. Total cortisol suppression would have been counter-productive as there existed an obvious synergy between cortisol and most contest or growth chemistries relating to growth, as well as immune function. Over dosing or prolonged use of cortisol suppressing drugs would have potentially resulted in serious negative feed-back loops.

**DNP:** DNP affected the Krebs cycle by making the mitochondria create heat instead of ATP (energy). It did so by introducing lots of hydrogen ions between the two cellular outer membranes. The Krebs cycle is a series of chemical reactions that occur within the mitochondria that are responsible for the breakdown of nutrient molecules to form water and carbon dioxide as well as ATP. In short, DNP uncoupled the process.

About a 50% + increase in metabolic rate occurred with the use of 5mg/kg of DNP daily. For most beasts this translated into a fat loss of up to 6-10 LBS in a 7-day span. Most of this heat was surface heat not internal heat so it was necessary for Frank to monitor his body temperature; 101-102 degrees was not uncommon. Also Frank slept in an air-conditioned room or had a fan on his face while he slept.

Though I would never claim DNP was safe, I will say a maximum dosage of 5 mg/kg daily appeared not to be dangerous for most individuals. At a dosage of as little as 8 mg /kg daily, DNP could have been DEADLY!!!

During periods of DNP use, Frank experienced major carb craving periods. Usually 25-50 grams of peptide or L-Glutamine resolved this. Arginine worked well also, but we had another use for this muscle mass amino acid we will discuss later.

Another factor that fascinated me about DNP was its ability to prevent fat gain during insulin use. It did so by screwing up the shape of the insulin molecule. This also meant it was far more difficult to go hypoglycemic when DNP and insulin were stacked. Obviously DNP use increased the PTOR which then allowed for increased calorie counts and therefore increased nutrient availability at the cell.

I personally believed DNP was very useful but had to be utilized with serious caution. Some users simply ingested the 5mg/kg dosage first thing in the morning. Others broke it up into 2 evenly divided dosages. I liked DNP's ability to clear receptor sites. This was especially effective for androgen receptor clearing and count up-regulation.

While utilizing DNP, Frank had to train with higher reps and lower weight loads. This was because DNP users have experienced serious muscle cramps and tears due to interference with ATP re-synthesis and other factors.

**Red Blood Cell Expanders:** AAS such as Anadrol-50 were sometimes utilized as a method of increasing red blood cells count. This allowed a beast to appear much fuller and vascular because there was more blood to expand vascular tissue (Like veins and muscle).

There is of course a correlation between red blood cell count and hemocrit. I have often seen athletes with a hemocrit of over 52 thus exceeding the safer reference range. Instead of using one of the many hemocrit control drugs it has always been so much healthier to simply donate blood. The reduction is immediate and effective...and far more healthy.

Eprex was a protein that aided in inducing an increase in red blood cell count. It too has been used pre-contest to create improved vascularity and a fuller musculature. It took about a month to become effective when utilizing daily dosages of 1000 iu/d subcutaneously (sc). SC injections were far more effective when Eprex was the drug. 60 days total use was a maximum use period. A blood viscosity increase too high could have resulted in heart, vascular, and brain damage.

Eprex was often utilized as a method of increasing vascular support for new growth potential. As I have explained prior, new tissue only grows if there is an existing supply of vascular and nerve tissue as well as enough blood/nutrients to adequately supply it. If there is an existing inability for nerves to communicate with the cells or a lack of vascular tissue to supply blood, no growth occurs. Eprex was employed by some beasts, but others wisely avoided it.

**PGE-1:** PGE-1 was a prostaglandin of the same group (but not series) as PGF-2. The difference was size created from deep site-injections was mostly cosmetic and resulted in a size increase lasting 4-6 hours. When 10-20 mg of PGE-1 was injected directly into a muscle, swelling occurred within about 10 minutes. Using arms as an example, 10-20mg was injected deeply into each bicep/tricep, resulting in a size increase of 1-2 " per arm.

This has been utilized as a "last minute fix" for obvious lagging body parts. PGE-1 came in 1cc vials, providing either 10 or 20 mg. Nollatil, Kaverject (5cc ampules) and generic PGE-1. They are both used in medicine to induce penile erections for those who have such problems. I find it interesting that research has found that regular use, for this purpose, results in permanent size gains.

Yes, there too. The issue of layers, not including AAS foundation and estrogen control, was a matter of synergistically improving results.



## Getting Started...

Beginning with the AAS foundation, Frank utilized 2-6 week periods or phases. I believe it was not necessary to administer mega dosages of AAS during calorie restricted periods. This was due to the fact that there simply was not enough calories to support much growth... usually. For this reason, Frank's foundation included approximately 2-3 mg/lb weekly of a fast acting Testosterone, 1-2mg/lb weekly of a high anabolic /moderate androgenic AAS, and 1 mg/lb weekly of a high anabolic/low androgenic AAS for the first 6 weeks.

The second 6 week period included a slightly higher level of testosterone, 1-2mg/lb weekly of a high anabolic/low androgenic AAS, and 1 -1.5 mg/lb weekly of a very high androgenic steroid that aided in lean mass retention and increased fat loss in a calorie restricted state.

For the very high androgenic AAS, Primobolan, Masteron, Finabolan, trenbolone acetate, or Primobolan Depot (Not in that order) were good choices. Often Winstrol Depot was added to beasts contest prep cycles for the same reason. And I have to admit that it increased muscle quality and separation when it was utilized site-specifically such as in pecs.

The results would not have been quite as impressive, but some beasts utilized a far more economical AAS foundation. This was a matter of: The first 6 week period provided 2-3mg/lb weekly of Testosterone Propionate, or in a pinch, Testosterone Enanthate. We added 2 mg/lb weekly of Deca Durabolin and the base was set. Some believed that nandrolones left an athlete looking flat. This was actually a factor of low DHT activity combined with improper glycogen loading.

The second 6 week period provided 2.5-3.5 mg/lb weekly of Testosterone Propionate or Enanthate, 1 mg/lb weekly of Deca or Equipoise, and 1 mg/lb weekly of Primobolan Depot. The Primo had also been replaced by Masteron or Finabolan, but that became another cost issue. Later, I will explain how the whole contest prep phase had been cut to 9 weeks or less for beasts that had not allowed themselves to become too sloppy. It was mostly a matter of muscle quality acquired during mass gain phases and how much loose skin there was to allow time for tightening.

As I wrote prior, it all came down to planning way ahead.

## FRANK'S AAS FOUNDATION

VIAL#1 (100ml Vial)	VIAL#2(100ml Vial)
Deca Durabolin 16.5 ml (100 mg/ml)	Parabolan 33 ml (22 76 mg Amps)
Equipoise 33 ml (50 mg/ml)	Equipoise 33 ml (50 mg/ml)
Testosterone Propionate 33 ml (100 mg/ml)	Testosterone Propionate 39 ml (100 mg/ml)
Protest or Other 1 ml	Benzyl Alcohol 0.5 ml
Benzyl Alcohol 0.5 ml	Total = 105.5 ml
Total = 84 ml	
<b>* 2 ml Per Day For 42 Days</b>	<b>* 2.5 ml Per Day For 42 Days</b>

**Vial #1** allowed for 2 ml of Max Mix daily. Frank split this 1 ml per side for each body part worked that day. Testosterone really ramped up training intensity while aiding in post-work out rejuvenation. Equipoise worked well as a lean mass retention drug while adding to superior protein synthesis /sparing effects of Deca. I think Deca drew too much water when used by some individuals, even with a good estrogen suppression layer. Oust my opinion) So it was not my personal "best choice" during the last few weeks before a show.

**Vial #2** allowed for 2.5 ml of Max Mix daily, obviously split 1.25 ml per side for each body part trained that day. Parabolan was an amazing AAS. It dramatically increased hardness, strength, and aided in fat burning. However, it was rough on the kidneys, so Frank needed to increase water intake until the last couple of days. (Masterone was successfully employed by some beasts to replace Parabolan as was Finabolan /trenbolone when timing factors are considered).

### **Example #1**

The following example of layers Frank utilized (in a progressive manner) is pretty straight-forward. The AAS Foundation was set and estrogen control was layered in. Prolonged periods of AAS use significantly increased aldosterone production due to estrogenic activity and kidneys/adrenals stimulation. Control was a must or Frank would have appeared smooth and water logged. Nolvadex alone did not do it.

GH was layered into the AAS foundation beginning 8-12 weeks before his contest date in a daily protocol that slowly increased or ramped up in dosages. This aided in maintaining activity or counter acted the resulting up-regulation of somatostatin. The amino acid arginine was included to aid in somatostatin control. GH was removed 6-7 days prior to contest day as many athletes hold extra subcutaneous water during use. It takes 5-6 days for this to clear.

Due to calorie restriction and GH use, thyroid hormone activity decreased as a result of endogenous secretion/conversion down-regulation. For some beasts triacana was successfully utilized to restore and slightly elevate T-3 levels up to the last few days before a show.

The most common effective dosage was 1 mg/ 50 lbs of bodyweight daily divided into 4 equal dosages. More will not increase thyroid hormone activity. Triacana was also utilized on non-Cytomel days to assure adequate T-3 levels. Remember: triacana had a half-life of 6 hours.

Cytomel (T-3) was layered in beginning 6-8 weeks before a show. This was plenty for most beasts, unless they had gotten too fat during mass phases. The goal was to be lean and ripped, not to be hypothyroid for life or counter act all that anabolic/anti-catabolic chemistry. (Odds of this are very low but gambling is not a wise choice)

Thyroid hormone dosages had to be adjusted in respect to anabolic/anti-catabolic chemistry dosages. Not the other way around. This was how effective dosage levels were exceeded and long-term growth potential destroyed for all to many would-have-been-beasts. One step at a time!!

Cytomel was utilized in a 2 day on/2 day off protocol until the last 20-30 days before a show. So beginning 6 weeks out during a 12 week contest prep, dosages were: Week #7-8 50-100 mcg/d, #9-10 75-150 mcg/d, #11-12 100-200 mcg/d. Triacana was effectively utilized on non-Cytomel days and 100-200 mcg/d of Cytomel was utilized daily the last 20 days continuously.

Obviously the synergy between AAS and GH increased lean mass retention and/or growth (when thyroid hormone use was not excessive) and fat metabolism was significant. During the last 3 weeks before a show, Frank commonly utilized PGF-2 and / or IGF-1 to bring up weak body parts.

As mentioned prior, some body parts lost mass more quickly than others during calorie restricted periods. This would obviously potentially destroy symmetry. PGF-2 also aided in an over all hardening and fat loss effect. This was due to a synergistic response with T-3 and GH, but also through a different metabolic pathway. I have witnessed athletes adding 2-3 inches to their arms and/or calves during the last 3 weeks before a show.

Other muscle groups also had this response to site-specific PGF-2/IGF-1 administration. The average effective minimum IGF-1 dosage was 0.10 mcg/kg 3-5xd. The dosage for PGF-2 was 2 mg 3-5xd.

Beginning 8 weeks before a show, Frank utilized Clenbuterol and an Ephedrine / Caffeine stack in a 2 day each rotation. This also aided in T-4/T-3 conversion while providing some anti-catabolic qualities and increased thermogenesis.

Beginning 8 weeks out, during a 12 week contest prep, an example of Frank's layer looked like this: Week #5-6 Clen. 80-100mcg/d-Eph 25 mg-Caff. 250 mg 3xd; #7-8 Clen. 100-120 mcg/d-Eph. 25 mg, Caff. 250 mg, #9-10 Clen. 120-140 mcg/d - Eph. 25 mg, Caff. 250 mg 3xd; #11-12 Clen. 140-160 mcg/d-Eph. 25 mg, Caff. 250 mg 3xd.

When Frank was not utilizing PGF-2, he increased Clenbuterol dosages an additional 20 mcg for each 2 week segment. Some beasts utilized 50-100 % higher dosages, but why would Frank have blown an effective threshold before necessary? Clenbuterol dosages were divided into 2-4 equal daily dosages due to that Frank and some other individuals experiencing some stomach discomfort with single dosage protocols.

Frank regularly monitored his body temperature to assure effective dosage and health. Over 101 degrees was too high, under 99.6 degrees was too low. (Elevated thyroid hormone levels also increased body temperature.)

*"Just because it is done, does not mean it is right or best!"*  
*Coach*

- Frank ceased CH on the last 6-7 days before a show. This aided in carb loading and water depletion both.
- During the last 1-2 days, Frank used 40 mg/d of Lasix to drop excess water weight.
- Frank had also successfully used 8-10 iu Humalog 2xd with 10 g creatine, 75 g dextrose, 25 g glutamine, 400 mg lipoic acid during the carb-loading period the last 2-3 days.
- Some beasts added PGF-2 week # 10 2 mg 3xd, #11 2 mg 4xd, #12-2 mg 5xd.

## Example #1

### Week#

Week 1	Vial # 1-2 ml/d	GH 2 iu/2xd	Arimidex 1.5mg/d	Nolvadex 20mg/d
Week 2	Vial # 1-2 ml/d	GH 2 iu /2xd	Arimidex 1.5mg/d	Nolvadex 20mg/d
Week 3	Vial # 1-2 ml/d	GH 2 iu/2xd	Arimidex 1.5mg/d	Nolvadex 20mg/d
Week 4	Vial # 1-2 ml/d	GH 2 iu/2xd	Arimidex 1.5mg/d	Nolvadex 20mg/d
Week 5	Vial # 1-2 ml/d	GH 2 iu/3xd	Arimidex 1.5mg/d	Nolvadex 30mg/d
Week 6	Vial # 1-2 ml/d	GH 2 iu/3xd	Arimidex 1.5mg/d	Nolvadex 30mg/d
Week 7	Vial # 1-2 ml/d	GH 2 iu/3xd	Arimidex 1.5mg/d	Nolvadex 30mg/d
Week 8	Vial #2-2.5 ml/d	GH 2 iu/3xd	Arimidex 1.5mg/d	Nolvadex 30mg/d
Week 9	Vial #2-2.5 ml/d	GH 2 iu/4xd	Arimidex 1.5mg/d	Nolvadex 40mg/d
Week 10	Vial #2-2.5 ml/d	GH 2 iu/4xd	Arimidex 1.5mg/d	Nolvadex 40mg/d IGF-1 3xd
Week 11	Vial #2-2.5 ml/d	GH 2 iu/4xd	Arimidex 1.5mg/d	Nolvadex 40mg/d IGF-1 4xd
Week 12	Vial #2-2.5 ml/d		Arimidex 1.5mg/d	Nolvadex 40mg/d IGF-1 5xd

- Some did not respond to GH until daily dosages were at least 9iu
- Arimidex did possess "some" anti-catabolic /cortisol control qualities. However, Frank did best when he layered in a cortisol inhibitor the last 4 weeks before a show. 2 days on/2 days off was still best. He had employed three choices:
  1. Cytadren-500 mg/d week #9, 750 mg/d week #10, 1000mg/d week #11-12.
  2. Metryapone-500 mg/d week #9-12.
  3. Trilostane -120 mg/d week #9, 180 mg/d week #10, 240 mg/d week #11-12.
- Remeron 1 5mg/d has been successfully used for this reason during the last 3 weeks.

It should be noted that prolonged periods of high dosage use of cortisol inhibitors (Except Remeron) has been reported to result in influencing ACTH production which in turn induces uncontrolled cortisol problems.

The use of different cortisol suppression drugs on alternating or consecutive schedules, would have been theoretically more beneficial over-all for delayed Action/Reaction Factors and negative feed-back loops.

An example utilized by a few beasts was that Metryapone inhibits 11-beta-hydroxylation in the adrenal cortex where as Trilostane inhibits the enzyme 3-beta-hydroxysteroid dehydrogenase delta 5,4 isomerase. This was two different metabolic pathways that accomplished the same goal of cortisol and/or estrogen inhibition.

Frank needed to regenerate proper HPTA function after the show since this protocol was lengthy. This meant HCG, Teslac, and Proviron or one of the methods explained else where in this book, or in "Chemical Muscle Enhancement".

Proviron has also been universally successfully utilized the last 3-8 week before a show to enhance over all hardness. This was also true of the liver hater, Halotestin, too. Male Mix also worked pretty well for post-cycle HPTA regeneration.

Frank layered in 6-10 iu 2xd of Humalog every other day during the last 4 weeks before a show only when he lacked over all size. Thankfully it was only once that this was deemed necessary. When a beast's size **really sucked**, he did the same during the first 4 weeks of a 1 2-week diet contest prep period as well. This allowed a 4 week off period between Insulin use.

Post competition, Frank was in a perfect state to begin an Absolute Anabolic Phase. (Gee, Ya Think?)

**Note:** Viagra and 3g of L-Arginine ingested an hour before each show resulted in a far better pump (not there) due to increased total body NO. This means improved vascularity and muscular harness with a fuller look.

Another approach to pre-contest fat loss utilized DNP (Dinitrophenol) in two different protocols. The first was simply a matter of ingesting 5 mg of DNP per KG of bodyweight daily for 7 days. Then begin an 8 week layered cycle. This cleared receptor-sites and made the body's musculature more sensitive to AAS and other protocols.

It also caused a 4-8 LB fat loss while preventing the need for an entire 12-16 week period of AAS use. This was possible only when a beast did not have too much skin to tighten up from getting sloppy during mass gaining phases...

The second method involved the use of DNP on one or two weekly non-training days (with aerobics) only. This DNP/non-training day(s) was best at the beginning of each week for 8 consecutive weeks. T-3 such as Cytomel was usually layered in as well and the total cycle length ran 60 days. This allowed for greater receptor sensitivity each week. The AAS Foundation was either a Max Mix or as listed, assuming a lean mass weight of 271 lbs.

**GH** use was 2 iu 2xd day #1-20, 2 iu 3xd day #21-40, 2 iu 4xd day # 41-57 and 2 iu 2xd on day #58-60.

**Cytomel (T-3)** use was 50-100 mcg on days #4-30, 75-150 mcg on days #31-45, 100-200 mcg on days #46-60 (on listed days) and Triacana on non-T-3 days.

**Estrogen Control** began on day # 1 and continued for atleast 1 0 days after day # 60 to limit negative post-cycle feed-back loops.

**Cortisol Suppression** was sometimes layered in beginning day # 30 and ran a total of 30 days. 2 on/2 off. This was long enough to significantly reduce an overwhelming cortisol level post-cycle as well.

**IGF-I/PGF-2** was sometimes layered in during the last 21 days on non-DNP days.

**Insulin** (Fast acting) was an additional layer sometimes employed on DNP days only equal to carb intake post work-out and during the 2-3 days of carb-loading.

**Thermalgenics** like E/C/A helped some beasts reduce carb craving on DNP days. Glutamine was excellent for this purpose also.

## FRANK'S CONTEST PREP Chemistry -Example #2 (Chart)

DAY	DRUGS	DAY	DRUGS
1.	DNP/Sus-2 50/Deca 250 mg/GH	31.	GH/T-3
2.	GH	32.	GH/T-3
3.	GH	33.	Sus-250/Primo D. 200 mg/GH/T-3
4.	GH/T-3	34.	T-3/Win. D. 50mg/GH
5.	GH	35.	T-3/Win. D. 50mg/GH
6.	GH	36.	DNP/Win. D.50 mg/GH
7.	GH	37.	Sus-250/Primo D. 200 mg/GH/T-3
8.	DNP/GH/T-3	38.	T-3/Win.D. 50 mg/GH
9.	Sus.-250/Deca 250 mg/GH/T-3	39.	Win. D 50mg/GH/T-3
10.	GH	40.	Win. D 50mg/GH/T-3
11.	GH	41.	Sus-250/Primo D. 200 mg/GH/T-3
12.	GH/T-3	42.	T-3/Win. D. 50mg/GH
13.	Sus.-250/Deca 250 mg/GH/T-3	43.	DNP/Win. D 50 mg/CH
14.	GH	44.	T-3/Win. D. 50mg/GH
15.	DNP/GH	45.	T-3/Test. P. 150 mg Primo D 200 mg/GH
16.	GH/T-3	46.	Win. D 50mg/CH/T-3
17.	Sus.-250/Deca 250 mg/GH/T-3	47.	Test. P. 1 50mg Win. D. 50 mg/GH/T-3
18.	GH	48.	T-3/Win. D 50 mg/GH
19.	CH	49.	T-3/Test. P. 150 mg Primo D 200 mg/GH
20.	GH/T-3	50.	DNP/Win. D 50 mg/GH
21.	Sus.-250/Deca 250 mg/GH/T-3	51.	T-3/Test. P. 1 50 mg Win. D. 50mg/GH
22.	DNP/GH	52.	Win. D 50 mg/GH/T-3
23.	GH/T-3	53.	T-3/Test. P. 1 50 mg Win. D. 50mg/GH
24.	GH/T-3	54.	Win. D 50 mg/GH/T-3
25.	Sus.-250/Deca 250 mg/GH	55.	T-3/Test. P. 200mg Win. D. 50 mg/GH
26.	GH	56.	T-3/Win. D 50 mg/GH
27.	GH/T-3	57.	Test. P. 200 mg Primo D. 200mg/GH/T-3
28.	GH/T-3	58.	Win. D. 50 mg/GH/T-3
29.	DNP/Sus-2 50/Deca 250 mg/GH	59.	T-3/Test. P. 200 mg Win. D. 50 mg/GH
30.	GH/T-3	60.	T-3/Win. D 50 mg/GH

- E/C/A stack had run 60 days
- Triacana was sometimes added on non-T-3 days

I realize some may feel that the dosages listed in any example thus far were low. There were individuals who I knew that introduced as much as 3000 mg (even 6000 mg) of AAS alone weekly. Just because it was done, did not make it right... or necessary.

Personally, I did not believe that a weekly AAS actual "plasma threshold" should have ever exceeded 1500 mg. (Remember: This issue relates to "plasma threshold" and not to the ester weight adjoined to the active AAS itself.) If it had become necessary to do so to make gains, then it would have been time to focus upon clearing/up-regulating androgen receptor-sites and re-evaluate Action/Reaction Factors.

In most cases, those individuals who failed to realize results at 1 500 mg weekly plasma levels were those whose phases were fighting the body's adaptive response mechanisms. Not properly utilizing these adaptive responses for maximal progress always resulted in failure eventually.

Another example of a plan to fail was the abuse of cortisol inhibitors such as Cytadren. 2500 mg/d had become common during contest prep for less knowledgeable individuals. What on earth made someone believe they needed a dosage above that of a Cushings Syndrome patient?

2000 mg/d totally inhibits hormone biosynthesis for Cushings victims and they are producing many times more cortisol than the hardest training mega dosed bodybuilder ever was. To add to this I lost a dear and close friend not long ago. He had been in a minor car crash. The poor doctors could not stop the bleeding due to my friends secretive over-use of Cytadren.

Take a good look at *some* of the Masters Olympia lads. Vince Taylor was well past 40 years old and he looked better than ever. Vince had been a top competitor for 20 some odd years and the man was healthy. Do you honestly believe he utilized life long mega dose protocols? He would have been dead or totally burned out if so. I hope he, and several other greats, continue as they have. They will actually get better still. It's a fact!



## Of Course There Were Other Chemistry Contests Preps...

Following is an 86-day chemistry count-down that was used for a rather large and experienced beast last show.

- 1.75ml is administered to each target muscle (i.e. left side and right side) for a total of 3.5ml administered daily.
- Day 86-40 8iu Humalog + 10g glutamine + 10g BCAA's + 50g whey protein upon waking daily.
- 12iu Humalog Post work-out
- 10g Arginine before bed on non-GH days

*"Create the following Max Mix vial using a 7 00ml vial:*

### Vial A Contents:

28ml Reforvit-B (Injectable methandrostenolone 50mg/ml)

28ml Trenbolone Acetate (75mg per ml)

28ml Testosterone Propionate 50mg/ml

86.	Vial A 1.75ml outer bicep
85.	Vial A 1.75ml medial tricep/GH 2iu 3xd
84.	Vial A 1.75ml lower lat/GH 2iu 3xd
83.	Vial A 1.75ml upper trap/GH 2iu 3xd
82.	Vial A 1.75ml outer calve
81.	Vial A 1,75ml outer bicep
80.	Vial A 1.75ml medial tricep
79.	Vial A 1.75ml lower lat
78.	Vial A 1.75ml upper trap + stanozolol 25mg/GH 2iu 3xd
77.	Vial A 1.75ml outer calve + stanozolol 25mg/GH 2iu 3xd
76.	Vial A 1.75ml outer bicep + stanozolol 25mg/GH 2iu 3xd
75.	Vial A 1.75ml medial tricep + stanozolol 25mg
74.	Vial A 1 75ml lower lat + stanozolol 25mg
73.	Vial A 1.75ml upper trap + stanozolol 25mg
72.	Vial A 1.75ml outer calve + stanozolol 25mg
71.	Vial A 1.75ml outer bicep + stanozolol 25mg/GH 2iu 3xd
70.	Glucophage 425mg 2xd/ Vial A 1.75ml medial tricep + stanozolol 25mg/GH 2iu 3xd
69.	Glucophage 425mg 2xd/ Vial A 1.75ml lower lat + stanozolol 25mg/GH 2iu 3xd
68.	Glucophage 425mg 2xd/ Vial A 1.75ml upper trap + stanozolol 25mg
67.	Glucophage 425mg 2xd/ Vial A 1.75ml outer calve + stanozolol 25mg
66.	Glucophage 425mg 2xd/ Vial A 1.75ml outer bicep + stanozolol 25mg
65.	Glucophage 425mg 2xd/ Vial A 1.75ml medial tricep + stanozolol 25mg
64.	Glucophage 425mg 2xd/ Vial A 1.75ml lower lat + stanozolol 25mg/GH 2iu 3xd
63.	Glucophage 425mg 2xd/ Vial A 1.75ml upper trap + stanozolol 25mg/GH 2iu 3xd
62.	Glucophage 425mg 2xd/ Vial A 1.75ml outer calve + stanozolol 25mg/GH 2iu 3xd
61.	Glucophage 425mg 2xd/ Vial A 1.75ml outer bicep + stanozolol 25mg
60.	Glucophage 425mg 2xd/ Vial A 1.75ml medial tricep + stanozolol 25mg
59.	Glucophage 425mg 2xd/ Vial A 1.75ml lower lat + stanozolol 25mg
58.	Glucophage 425mg 2xd/ Vial A 1 75ml upper trap + stanozolol 25mg
57.	Glucophage 425mg 2xd/Sustanon-250mg

56.	Glucophage	425mg	2xd/Sustanon-250mg	
55.	Glucophage	425mg	2xd/Sustanon-250mg	
54.	Glucophage	425mg	2xd/Sustanon-250mg	
53.	Glucophage	425mg	2xd/Sustanon-250mg	
52.	Glucophage	425mg	2xd/Sustanon-250mg	
51.	Glucophage	425mg	2xd/Nandrolone Decanoate 50mg	
50.	Glucophage	425mg	2xd/Nandrolone Decanoate 50mg	
49.	Glucophage	425mg	2xd/Nandrolone Decanoate 100mg	
48.	Glucophage	425mg	2xd/Nandrolone Decanoate 100mg	
47.	Glucophage	425mg	2xd/Nandrolone Decanoate 1 50mg	
46.	Glucophage	425mg	2xd/Nandrolone Decanoate 1 50mg	
45.	Glucophage	425mg	2xd/Nandrolone Decanoate 200mg	
44.	Glucophage	425mg	2xd/Nandrolone Decanoate 200mg	
43.	Glucophage	425mg	2xd	
42.	Glucophage	425mg	2xd/GH 2.5iu 3xd	
41.	Glucophage	425mg	2xd/R-3 IGF-1 60mcg 3xd	
40.	Glucophage	425mg	2xd/GH 2.5iu 3xd	
39.	Glucophage	425mg	2xd/R-3 IGF-1 60mcg 3xd	
38.	Glucophage	425mg	2xd/GH 2.5iu 3xd	
37.	Glucophage	425mg	2xd/Oxandrolone 50mg/R-3 IGF-1 60mcg 3xd	
36.	Glucophage	425mg	2xd/Primo Mix 100mg/Oxandrolone 50mg/GH 2.5iu 3xd	
35.	Glucophage	425mg	2xd/Oxandrolone 50mg/Testosterone Enanthate 600mg/EQ 300mg/R-3 IGF-1 60mcg 3xd	
34.	Glucophage	425mg	2xd/Oxandrolone 50mg/GH 2.5iu 3xd	
33.	Glucophage	425mg	2xd/Primo Mix 100mg/Oxandrolone 50mg/R-3 IGF-1 60mcg 3xd	
32.	Glucophage	425mg	2xd/Oxandrolone 50mg/GH 2.5iu 3xd	
31.	Glucophage	425mg	2xd/Oxandrolone 50mg/Testosterone Enanthate 600mg/EQ 300mg/R-3 IGF-1 60mcc 3xd	
30.	Glucophage	425mg	2xd/Primo Mix 100mg/Oxandrolone 50mg/GH 2.5iu 3xd	
29.	Glucophage	425mg	2xd/Oxandrolone 50mg/R-3 IGF-1 60mcg 3xd	
28.	Glucophage	425mg	2xd/Oxandrolone 50mg/GH 2.5iu 3xd	
27.	Glucophage	425mg	2xd/Primo Mix 100mg/Oxandrolone 50mg/Testosterone Enanthate 600mg/EQ : i00mg/R-3 IGF-1 60mcg 3xd "	
26.	Glucophage	425mg	2xd/Oxandrolone 50mg/GH 2.5iu 3xd/GH 2.5iu 3xd	
25.	Glucophage	425mg	2xd/Oxandrolone 50mg/R-3 IGF-1 60mcg 3xd	
24.	Glucophage	425mg	2xd/Primo Mix 100mg/Oxandrolone 50mg/GH 2.5iu 3xd/GH 2.5iu 3xd	
23.	Glucophage	425m	2xd/Oxandrolone 50mg/Testosterone Enanthate 600mg/EQ 300mg/R-3 IGF-1 60mcc g j 3xd	
22.	Glucophage	425mg	2xd/Oxandrolone 50mg/GH 2.5iu 3xd	
21.	Glucophage	425mg	2xd/Primo Mix 100mg/Oxandrolone 50mg/R-3 IGF-1 60mcg 3xd	
20.	Glucophage	425mg	2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/ Testosterone Enanthate 600mg/EQ 300mg/GH 2.5iu 3xd	
19.	Glucophage	425mg	2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/GH 2.5iu 3xd	
18.	Glucophage	425mg	2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/Primo Mix 100mg/CH 2.5iu 3xd	
17.	Glucophage	425mg	2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/GH 2.5iu 3xd	
16.	Glucophage	425mg	2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/ Testosterone Enanthate 600mg/EQ 300mg/GH 2.5iu 3xd	
15.	Glucophage	425mg	2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/Primo Mix 100mg/GH 2.5iu 3xd	
14.	Glucophage	425mg	2xd Turniabol 50mg/Oxandrolone 50mq/Stanozolol Oral 50mg/GH 2.5iu	

	3xd
13.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/GH 2.5iu 3xd
12.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/Primo Mix 100mg/Testosterone Enanthate 600mg/EQ 300/GH 2.5iu 3xd
11.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/GH 2.5iu 3xd
10.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/GH 2.5iu 3xd
9.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/Primo Mix 100mg/GH 2.5iu 3xd
8.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg Testosterone Enanthate 600mg/EQ 300mg/GH 2.5iu 3xd
7.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/GH 2.5iu 3xd
6.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/Primo Mix 100mg/GH 2.5iu 3xd
5.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg
4.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/ Testosterone Enanthate 600mg/EQ 300mg
3.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg/Primo Mix 100mg
2.	Glucophage 425mg 2xd/Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg
1.	Glucophage 425mg 2xd Turniabol 50mg/Oxandrolone 50mg/Stanozolol Oral 50mg

- Liquidex day 86-42 1 mg/1 ml, day 41-1 1.5mg/1.5ml (Check label for mg per ml)
- We may need to add Masteron beginning day 42 if holding fat

## MORE THINGS TO THINK ABOUT

We have discussed many things that Frank and other beasts did with great success in the area of muscular augmentation. We have discussed total body mass and site-injection protocols. Hell, we have even discussed Frank's aerobic sex Olympics. So lets take another look at drug synergy and restructuring the PTOR of lagging body parts employed.

We have discussed in depth, both in the first Chemical Muscle and the present text, the fact that testosterone induced muscular hypertrophy and, to a lesser degree, hyperplasia. The latter of course meant muscle cell growth by increasing both contractile and structural proteins, as well as an increase in satellite-cell production.

These satellite-cells were incorporated into existing muscle fibers and created new fibers. We have also learned that testosterone transformed type-I muscle fibers into size /strength type-II muscle fibers. We even discussed the cool fact that these newly formed and incorporated satellite-cells possessed a greater than normal number of nuclei (brains) and androgen receptor-sites. So more nuclei, receptors, cells and fibers meant more growth potential for beasts.

GH/IGF-1 induced muscle cell hyperplasia and increased structural proteins at an impressive rate. (They also positively affected contractile proteins to a lesser degree.) Which in itself increased muscle mass and growth potential. IGF-1 was very effective for site-specific growth, though some IGF-1 did migrate into the vascular system.

This meant some IGF-1 contacted and stimulated IGF-1 receptor-sites everywhere in the body. So more site-specific cellular growth potential, and some total musculature growth stimulation. This also meant more site-specific increases in cell and fiber counts for AAS to stimulate hypertrophy and hyperplasia type growth.

This also added up to a greater support network over time to feed and communicate with the new tissue and it's "altered" potential.

PGF-2 stimulated serious site-specific protein synthesis. Obviously some PGF-2 migrated to the vascular system and created some total musculature growth. But due to its brief active-life, direct muscle site localization responded with direct muscle site growth to a far greater degree. PGF-2 utilized site-specifically also increased androgen receptor-site number, and sensitivity. The synergistic result was more muscular tissue and higher numbers of more sensitive receptor-sites to interact with AAS site-specifically.

Insulin stimulated nutrient absorption and subsequent cellular recovery and growth. It also induced secondary cellular size increases through osmotic over compensation. Remember? Think all those newly formed satellite-cells, muscle cells, and fibers with more receptor-sites and above normal sensitivity may have benefited from Insulin?

Site-injection protocols stretched fascia which allowed space for vascular tissue growth. GH increased vascular / capillary growth through other pathways we have discussed prior. Training and weight gain stimulated neurological tissue and vascular tissue growth.

Frank was an example of '***only just beginning in possibilities***'. Any question about how we built the perfect beast? There is still more to come, of course. Much More!

## POST AAS CYCLE (Lean Mass Retention)

I often read or hear individuals claim that lean muscle mass accumulated during AAS protocols is soon lost upon discontinuance. Based upon my experience I will say that this was often unnecessarily true for the less knowledgeable. Obviously water retention from androgen aromatization was lost, but lean muscle mass retention percentage was purely dependant upon how an athlete handled Action/Reaction Factors.

Often referred to as rebound or negative feed-back loops, these were factors which usually negatively affected the HPTA function. Factors such as long AAS cycles, high aromatizing-androgen cycles lacking an anabolic transition phase, diet, elevated post-cycle cortisol/estrogen levels, rest, recovery, and post-cycle training protocols were mostly to blame for the ridiculous percentage of post-cycle lean tissue loss some would-have-been's reported.

When AAS cycles ran longer than 30 days, the HPTA received mega negative feed-back loop signals from elevated androgen aromatization induced estrogen levels. The body attempted to regain homeostasis by increasing catabolic activity chemistry such as cortisol. After discontinuance of exogenous AAS, estrogen and cortisol dominated the declining AAS plasma level which in turn sent a negative endogenous androgen production signal (telling "the boys" to go sissy) to the hypothalamus. This lead to further HPTA suppression and a bad case of shrunken nuts syndrome.

This is exactly why brief hard-hitting Max Androgen Phases were more productive than longer protocols. Cycles terminating high activity in 30 days or less did not create serious HPTA dysfunction nor did the body have time to fully adapt with excessively high cortisol levels.

In short, brief intense alterations in the anabolic/catabolic ratio did not allow the body as much reaction time as long alterations do. Obviously another factor was cycles utilizing a transition period from high androgen to high anabolic periods like the examples found under Max Androgen Phases. High anabolic AAS tended to suppress HPTA function less, create less estrogen to control, and had a better cortisol inhibiting effect.

However, *some* HPTA and cortisol/estrogen problems still occurred post-cycle. Just as any other protocol had to embrace certain goals, post AAS cycle periods had to follow goals leading to the best lean mass retention possible. In some cases, there was an improved **quality** to musculature during an estrogen control/HPTA regeneration period.

## LEAN MASS RETENTION

### THE GOALS WERE TO:

- Quickly establish superior HPTA function.
- Control and suppress estrogen activity and production to prevent fat accumulation and estrogenic negative feed-back loops.
- Suppress cortisol production and inhibit catabolic activity.
- Maintain a high CP/ATP level.
- Maintain a positive nitrogen and glycogen balance.
- Retain as much strength as possible.

Obviously estrogen control was not as difficult when Frank utilized an estrogen inhibitor during his AAS cycle. This also was not necessary when a Cortisol/Estrogen Suppression Phase followed his Max Androgen Phase.

### Estrogen Control/HPTA Regeneration Example 1

DAY	DRUGS	DAY	DRUGS
1.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg	12.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
2.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	13.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg
3.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	14.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
4.	HCG 500iu 4xd/Clomid 50mg , 2xd/Nolvadex 30mg	15.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
5.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	16.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg
6.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	17.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
7.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg	18.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
8.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	19.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg
9.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	20.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
10.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg	21.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd
11.	Clomid 50mg 2xd/Arimidex 0.5mg 2xd	22.	HCG 500iu 4xd/Clomid 50mg 2xd/Nolvadex 30mg

Cyclofenil 400-600 mg/d was utilized by some beasts to replace Clomid in this example.

**Example 1** was a fairly common HPTA stimulation/estrogen control protocol. The reader may note the varying periods of HCG use. This was not an accident, it merely produced better results. HCG was an LH imitator, meaning it only replaced LH/FSH levels, not initiate their production. This meant the Leydig Cells in the testes received an immediate signal to produce androgen endogenously.

Clomid aided in kick-starting the Hypothalamus/Pituitary production of endogenous LH but took a few days to become effective. It did so, in part, due to reducing the effects of HPTA suppression via inhibition of estrogenic activity upon the hypothalamus and pituitary.

Arimidex inhibits estrogen production while Nolvadex blocks estrogen receptor-sites. This of course blocks or prevents negative feed-back loops (from estrogenic activity) as well as prevent female pattern fat deposits.

Note the 500iu 4x daily of HCG schedule. I have noted for better LH and endogenous androgen profiles with this method and quicker HPTA function rebound. Athletes often forget that HCG is actually a female hormone that only mimics LH in males. Too much too fast and too long can be a bitch, (all puns intended) Cyclofenil can replace Clomid in many cases and Teslac can replace Arimidex.

Also Menotropins (75 iu twice daily with HCG injections) is another replacement for LH or can be utilized with HCG for a double LH kick. Normal adult male testosterone levels are 300-1000 ng/dl and are evaluated by a blood test. Remember; different countries labs use more or less conservative reference ranges.

**Note:** Zinc, magnesium, and vitamin B-6 are necessary co-factors for endogenous androgen production and significantly elevate free or unbound testosterone levels from any source. There are several nighttime specialty products available at health food stores of this nature.

If Frank did not have legal access to the items listed in example #1, he could accomplish the HPTA rejuvenation portion by taking "Male Mix" from Hazardous Materials daily for 21 days and eat broccoli 4 times daily (about a pound total) for some estrogen control. It actually does a fair job. Adding 15,000 mg of Tribulus Terrestris daily (45% samponin content) can also ramp up LH levels some.

The next goal, cortisol inhibition, was fairly easy. And in some beasts cases it was done without prescription drugs (and about 50-80% as effective as low dosages of Cytadren when evaluating value based solely on actual results). Again, this was only when a Cortisol/Estrogen Suppression Phase did not follow a Max Androgen Phase.

Several OTC products contain phosphatidserine. At a dosage of 800 mg daily phosphatidserine is said to inhibit about 30 % of cortisol production/activity, which is a start.



7-isopropoxyisoflavone (ipriflavone) is a weak partitioning agent that inhibits cortisol's effects at the cellular level. About 1000 mg daily is necessary. 5-Methyl-7-Methoxyisoflavone is more potent than 7-isopropoxyisoflavone. It inhibits cortisol activity, and to some extent, its accumulation to a greater degree. Again about 1000 mg daily is necessary and works best when stacked with 7-iso. Interesting fact is that due to its molecule structure, 5-Methyl does have some aromatase inhibitive value as well.

Vitamin-C, 1000mg 3 times daily also inhibits cortisol formation. Ephedrine at a dosage of 25mg-50mg three times daily significantly inhibited catabolic activity when it was stacked with any other cortisol inhibiting goodie.

Clenbuterol too, except we are not talking about prescription drugs this time.

The issue of Action/Reaction Factors applied to OTC products as well. Of course the effectiveness of any OTC product was highly dependant upon dosage and quality.

## MAINTAINING HIGH ATP LEVELS

During AAS phases intense strength gains were realized and protein synthesis at the cellular level increased dramatically. This was in part due to, and dependent upon, an up-regulation in internal production of CP (Creatine Phosphate) and ATP (Adenosine Tri-phosphate) from AAS use. As readers are aware from reading Chemical Muscle Enhancement or elsewhere, ATP is the body's primary high energy source for anaerobic activities such as lifting weights.

And CP is the resource chemistry the body uses to regenerate ATP. So, more CP meant more ATP, which meant more strength and more anaerobic capacity. ATP is also necessary part of the equation for protein synthesis and growing requires a great deal of both.

Supplemental creatine products were an excellent source of CP, and they aided in maintaining much of the AAS induced ATP/CP elevation post-cycle. I have already written extensively about creatine monohydrate and how beasts utilized its maximum potentials. So let's look at the king of creatines: Effervescent Creatine.

### Coach's EFFERVESCENT CREATINE FORMULA

*"The main problem with powdered creatine monohydrate is water solubility. It takes 100 milliliters of pure water to dissolve 750 milligrams of creatine monohydrate. So 5 grams would require about 670 ml or roughly 24 ounces of room temperature water to dissolve. Warm water does a somewhat better job. Remember, if creatine does not dissolve, it can not be absorbed. Some bright individuals actually just toss creatine powder into their mouths and chase it with water. That is like not chewing your food. Most of the creatine granules will not go anywhere useful. The second problem is the monohydrate electrical charge. Intestinal mucosa cells, which absorb nutrients, resist to some extent anything containing a net electrical charge of other than zero. (Which creatine monohydrate does not have) By the way, a molecule with a net electrical charge of zero is called a zwitterion and is readily absorbed by intestinal mucosa."*

*"Next is the issue of PH. The PH that allows creatine to have a net electrical charge of zero is 4, which is less acidic than normal stomach PH, but more acidic than water. Pure water is 7. A point of interest is the fact that a PH value of 4 is also the physiological signal to transfer goodies from the stomach to the small intestines where most absorption occurs. So now we realize that creatine is best absorbed at a PH value of 4, and that the creatine must be separated from the monohydrate. This allows 2 choices for athletes:*

*Purchase a good quality effervescent creatine, or you can be a mad chemist and make it yourself for under half the price."*

## **WHAT YOU WILL NEED:**

1000 grams SKW creatine (Prolab/Kaizen/Muscle Tech)  
400 grams Potassium Bicarbonate.  
450 grams Citric Acid.  
3600 grams (7.9 LBS) Dextrose.  
Flavor with sugar free drink mix (no artificial sugar/sweetener or Vitamin-C)

"Potassium Bicarbonate and citric acid powders can be bought at any science or chemistry shop quite cheap. Pick up some PH test strips allowing for a 3.0-5.0 PH test range while you are there. Dextrose runs about \$1.00 per pound at health food stores. Throw all the stuff in a DRY 5 gallon bucket with a secure tight fitting lid and roll it around while you watch T.V. for about 30 minutes."

"Using a 50 cc (ml) protein powder scooper (ya, we all have several from tons of protein powder) toss one level scoop into 8-10 oz of water. Test PH level. If it is above 4.2 PH, add more citric acid. If it is below 4.0 PH, add more Potassium Bicarbonate, to the mixture. Load with 4-6 servings daily on an empty stomach (it takes only 20 minutes for the mixture to clear the stomach) for 4-7 days and maintain elevated CP/ATP levels with 1-3 servings daily thereafter."

"By the way, it should fizz and completely dissolve in water. How much more effective is effervescent creatine than regular creatine monohydrate? How about 84% better than most (if not all) high dextrose content creatine transport mixtures. Creatine induced muscle cell volumization? 10-25%. Oh ya, it's about 194.9% more effective than regular creatine monohydrate powder. The PH does not have to be perfect but it should be close to 4.0. If the solution has no grit in the bottom after the addition of water, you have successfully created a Zwitterion. The best times to take creatine is upon waking, 20 minutes before a pre-workout meal, right after a workout, and atleast 90 minutes after any meal. Pretty simple, huh?"

## MAINTAINING STRENGTH LEVELS

Maintaining as much AAS induced strength post-cycle as possible was paramount to long-term results. I cannot tell you how many wanna-be hard-core bodybuilders I had witness training like sissies just because they were not juiced. They shrank, whined, and did anything but train smart and force themselves to focus on actual training.

Post-cycle (during "off periods" from AAS), Frank's strength, recovery, and training energy was less than while he was artificially chemically enhanced. His body simply could not handle the volume or intensity and expect to recover between workouts. During "off-periods" Frank maintained his normal set-load but simply eliminated high intensity techniques such as rest/pause, drop sets, and low/high sets.

This allowed for greater ATP regeneration between sets and less cortisol production due to training stimuli. He reduced his weight-loads by "about" 10% and did straight sets to failure only. He trained 2 days on/1 day off focusing on utilizing 5-7 reps to positive failure for each work-set with 2.5 minutes rest between sets. When he totally focuses on each set this load was not that difficult and post AAS cycle lean mass retention was far greater.

The vast majority of chemically enhanced athletes either over trained or sissy trained post-cycle. This of course was why so many never seemed to come off the gear. Sad, the "off periods" were the perfect time to recuperate and prepare for the next stage of growth with total focus.

I am not talking about Pro Bodybuilders that had guest appearances and shows near year round. Those poor bastards were under contract and felt that they had to do so. The competitive beasts that planned their schedules better realized that this was not necessary however. But in most other cases they were not in control, someone else was.

## Glutamine

Glutamine is a non-essential amino acid that plays an important role in both anabolism and anti-catabolism. It is the most abundant free amino acid in muscle cells. In fact glutamine makes up about 2/3 of total cellular amino acid content. That is significant. Simply stated, under the right conditions high plasma glutamine levels trigger cellular hyper-hydration which in turn triggers primary hypertrophy (anabolism due to a different mechanism. And if circulatory or cellular glutamine levels drop below normal, catabolism occurs due to amino acids exiting muscle cells.

Glutamine also plays a role in growth hormone release. Research has shown significant GH pulses in response to as little as 4 grams of supplemental oral glutamine. And we know GH has both anabolic and anti-catabolic effects.

Glutamine aids in immune system stabilization and function. It does so by contributing to the production of glutathione which is the body's most abundant water soluble anti-oxidant. High intensity training allows post-work out free radical levels to increase which in turn prolongs recovery time. Glutamine along with Methionine, Cysteine, and selenium aid in preventing this.

I have noted beasts who had utilized 50-100 grams of oral glutamine daily were leaner, bigger, healthier, and maintained post-cycle mass better than those who did not. There is **some** research that supports the belief that Peptide Glutamine is absorbed about 10 times better than free or L-Glutamine.

Personally I do not support the claimed statistic, but the value of certain peptide structures adjoined to glutamine can provide realistic synergistic value. And when glutamine peptide was available at an affordable price... So how did some poor beasts afford 50-100 g of peptide glutamine daily?

Whole wheat flour contains about 15-50 % peptide glutamine by volume, depending on the wheat type. Most health food stores sell a whole wheat flour containing about 25% peptide glutamine and it is usually under one dollar per pound. Each ounce provides about 125 calories and about 7 grams of peptide glutamine.

This makes whole wheat flour an excellent carb and glutamine source which is easy to absorb and afford. I mixed 2 ounces of whole wheat flour with either 2 oz of Human Profile or whey protein 4 or more times daily.

**WARNING:** It did jack up energy levels and had to be consumed immediately after mixing. If whole wheat flour sits in water (or any liquid) for any period of time, it tries to turn into bread. So for about \$.50 a day, a beast was able to get about 56g of peptide glutamine and an extra 1000 easily absorbed carb calories. Whole wheat flour may have tasted bad, but most of us once ate paper construction paste during our earlier grade school days anyway.

## HORMONES AND BALDING

During AAS use, hair loss from the scalp and swelling of the prostate gland (androgenic-alopecia and benign prostate hypertrophy) were a concern. If an individual was genetically predisposed to balding, AAS did obviously speed things along in many cases.

Let's look at Action/Reaction Factors leading to these conditions. Testosterones and many other AAS either convert to DHT to some extent or they themselves are DHT derivatives. The enzyme responsible for the conversion is type II-5-alfa-reductase.

DHT is about 5 times as androgenic as testosterone and receptor-sites in the scalp and prostate gland have a high affinity to DHT. Normally the body produces DHT, in part, as a hormone involved in sex drive and it aids in achieving and maintaining an erection. In bodybuilding DHT and its derivatives were used for their distinct hardening effect upon the musculature.

This should not be a surprise to readers since DHT and its derivatives do not aromatize to estrogens. The total inhibition of DHT activity would have lead to a distinct lack of wood in woody.

Finasteride is a prescription drug that acts as both a specific and competitive DHT inhibitor. It inhibits testosterone's conversion to DHT by binding the 5-alfa-reductase enzyme and preventing the activity necessary for the chemical reaction. It also acts as a competitive inhibitor by blocking the keyhole to the DHT metabolic pathway.

The latter is similar to the way Nolvadex inhibits estrogenic activity. Finasteride causes somewhere around a 70% reduction in the levels of both circulating DHT and DHT activity at specific sites in the body such as the scalp and prostate gland.

There are two prescriptions drug trade names for finasteride: Proscar which is 5 mg per tab, and Propecia which is provided as 1 mg tabs of finasteride. Proscar 5 mg tabs/30 count cost about \$85.00, where as Propecia 1 mg tabs/28 count cost about \$65.00. Normally about 1 mg daily of Finasteride is perfect for hair loss prevention, so a Proscar tab cut into quarters is obviously more reasonably priced.

Studies have shown that 88% of test subjects kept their hair after beginning use of 1 mg daily. A quarter tab of Proscar yields about 1.25 mg.

I am not suggesting or endorsing the idea of anyone doing this (weasel statement) but a friend of mine who lived in a country that lacked over the counter (OTC) access to such things obtained Proscar this way. Proscar was prescribed as

treatment for benign prostate hypertrophy (enlarged prostate gland) which is usually "said" to be caused by excess DHT activity.

He went to his doctor and complained of the symptoms for enlarged prostate. These are decreased flow of urine, frequent need to urinate and a sensation of the need to go with little volume resulting from the attempt. Then he informed the doctor that every male from his grandfather to his nephew has suffered from an enlarged prostate.

Next he endured a rectal exam to be certain his prostate was not actually dangerously swollen, and he received his prescription. The good news was that he had stopped losing his hair. The bad news is that, like about 2% of the test subjects, he initially suffered a reduction in both sex drive and a decrease in semen during ejaculation. Some (among the 2%) reported difficulty in achieving full erections. Which sucks, or doesn't ... whatever. You get the point.

Before I babble on, I would like to say that saw palmetto really is not a positive approach to DHT control for most serious athletes. There is a great deal of research that shows that saw palmetto actually inhibits testosterone activity by acting as a receptor-site blocker. But some do not care, so it's their choice.

Another prescription (in the USA, it is over the counter in Canada and Mexico) option was Nizoral shampoo. I am not referring to Nizoral orals, as they are quite liver toxic. Nizoral shampoo kills dandruff, fungus, and seborrhea also, but since it is a shampoo, it is unlikely much of the active chemistry will enter the vascular system unless someone drank it.

The label states Nizoral should be used atleast twice a week and left in the hair for 3-5 minutes. Some individuals suggested just leaving the stuff in and not rinsing. I did not agree. This lead to other problems such as a very dry scalp in most cases. Good results were obtained as directed, though I personally used it 4-6 times weekly.

It seems Nizoral inhibited the conversion of AAS into DHT in hair follicle receptors by binding to these receptors. The product also did a good job of getting rid of sebum, which is the fat like substance secreted by the scalp.

Another reason hair loss occurs is due to cytokines and superoxide, according to the late and great Dan Duchaine. Cytokines are substances produced in the body as are superoxides. Together they team up and burn hair follicles to death.

The superoxides also have been sited to cause many bad things including auto immune diseases. The best way to prevent superoxide activity is to take anti-oxidents (Duh!) such as glutamine, vitamin A, E and C, zinc, co-enzyme Q-10, selenium, and alfa lipoic acid.

Another concern for chrome dome syndrome was IGF-1. A recent study compared men with higher IGF-1 levels (and lower IGF-BP-3 levels obviously) to those with lower IGF-1 levels. The results showed that those with higher IGF-1 levels went bald sooner.

This is fairly easy to explain. IGF-1 inhibits hair follicles from entering their resting or catagen phase of hair growth. No rest period? Yikes. Anyway, any androgen promotes the local release of IGF-1 in androgen sensitive tissue (such as hair follicles) which then in turn mediates the initiation of our old pal 5-alfa-reductase. (Which of course mediates the conversion of testosterone and other AAS into DHT.) DHT then shrinks the hair follicle itself and hair falls out.

Now for the bad news: Finasteride (Proscar/Propecia) down-regulates gene expression of IGF-1 while increasing IGF-BP-3 levels, which I think sucks. IGFBP-3 is a binding protein of IGF-1. IGFBP-3 inactivates IGF-1 similarly to the way SHBG (sex hormone binding globulin) binds and deactivates androgens. Only the free or unbound hormone can activate a cell receptor-site. For this reason, I preferred Nizoral shampoo over Finasteride except for extreme cases. But that is only a point of view.

In truth there will always be those who will lose hair on their heads and grow it on their backs as a result of simply looking at an androgen funny. It should also be noted that GH converts into IGF-1 and therefore can cause gene expression.

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***Editor's Note:** Okay, here 's where I step in since hair is my sworn enemy! As stated earlier in this book, I haven't taken any steroids for years due to the whole hairloss/balding issue. That's why I love many of the products I mentioned earlier at both [www.hmgear.com](http://www.hmgear.com) and [www.AGRNutrition.com](http://www.AGRNutrition.com). The Nandrolone-OH and Testosterone-OH products are VERY safe for the hair if you are prone to hairloss. Anyway ...*

*I personally do NOT think that 1 mg of finasteride is enough to prevent or stop hairloss. Heck, I think if you are balding already, you should be taking 5 mgs of finasteride daily and if you take steroids, especially higher-dosed testosterones, you should up your dose to 10 mgs of finasteride daily! Yup ... no misprint. I've done it, my friends do it and we ALL get much better hair growth up on our scalps at this higher dose.*

*Always keep in mind that this is a dose dependent drug (like most drugs) ... the more balding hormones your body naturally makes (high DHT levels) or with the addition of steroids, the MORE finasteride you will need to fight this off.*

*Now, since we are on the subject of finasteride, there is a BETTER DHT inhibitor drug called dutasteride.*

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*Here in America, it's called Avodart and they come in 0.5 mg caplets and run about \$2.50 per caplet. It's expensive, especially since the really cool benefits happen at a higher dose of 2.5 mgs daily (hence, 5 caplets daily ... about \$12.50 daily in this drug!).*

*Where at a dose of 5 mgs daily finasteride blocks 70% of DHT conversion mainly through the Type II 5AR, 2.5 mgs of dutasteride blocks 90%-100% of BOTH Type II and I enzymes.*

*For those that don't know, the Type II is predominantly in the prostate, where Type I is mainly in the skin/scalp. Hence, dutasteride is a MUCH better drug - not only for hairloss, but also prostate problems.*

*All around, dutasteride is better... with the exception of the its 5-week half-life. If you start to get side effects such as lowered sex drive ... it's going to take a couple of weeks after getting off the drug before things return back to normal. While finasteride you'll be fine within 24-48 hours.*

*My personal anti-balding/hair-keeping stack is as follows:*

- *2.5 mgs of finasteride, twice daily - 5 mgs total daily dose*
- *0.5 mgs of dutasteride daily (more if you can afford it)*
- *An aromatase inhibitor such as Arimidex (0.5-1 mg daily) or Femara (1.25-2.5 mg daily). You can use both, on alternate days actually for best results.*
- *5% minoxidil, used topically twice daily*
- *2% topical spironolactone solution used twice daily (It blocks androgens at the scalp ... best not used it at the same time of the minoxidil application or it will give off a funky odor)*
- *Wash scalp with Nizoral shampoo 3-5 times weekly*

*I know that's a lot of work, but if keeping your hair is important, it's well worth it. It's just getting into a habit of doing the above on a daily basis. By the way, some of the side effects of finasteride or dutasteride such as lowered sex drive, less muscle hardness, etc. can be vastly reduced by taking an aromatase inhibitor such as Arimidex or Femara.*

*The reason is when taking finasteride/dutasteride by themselves, you lower DHT levels, this increases testosterone levels (good so far!) but if you tend to have a lot of aromatase enzymes in your body (as most balding guys do), this new higher testosterone will now convert to ESTROGEN which in turn makes you soft, lowers sex drive, gyno, etc., etc., etc.*

*Got it! Make sense? As ALR has said through out this book, Action/Reaction and synergy is what it's all about. Hairloss, hair growth, fat loss, muscle growth is no different!*

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## WHAT THE F@#K IS SYNERGISTIC CHAOS TRAINING?

I seem to constantly answer the question: "What is the best training method? " Wow is that answer a bitch to fit into a few lines of e-mail.

First, there is no one exact single training system that is best. Training is both individualistic and intent specific. I train clients utilizing a system called "Synergistic Chaos". It is not a simple to understand idea, but the goal of training is to realize the greatest results for a specific intent, in the shortest period of time. Forget the idea of "one best method" and much of what is written about a pro's training.

"Synergistic Chaos Training" was dubbed so by a client who was so impressed with his own results that he asked if I would co-write a book on it with him. I told him if he wrote it I would answer his questions as it progressed. 3 months later he gave up and named the method "Synergistic Chaos Training".

To be honest, I can not take all of the credit for the phenomenal results clients achieved. The system is simply applied science mostly researched by others. The available research has been utilized in separate "parts" by several writers and trainers as a one-size-fits-all system to make a name for themselves. Some are very good in fact, but the so-called systems often result in some kind of one-size-fits all strategy.

Personally I could not care less about a name (for the system or myself). I just liked building the perfect beasts. And after all, the system had to be called something. (*Building The Perfect Beast...Naturally* out-lines this method quite well and will be done sometime in 2004-2005)

I trained each client as an individual, and Frank was no exception. First we evaluated each muscle groups PTOR and fiber type ratios. Then discussed training, diet, and medical history. (Duh!) Sounds boring but I enjoyed structuring each athlete's Action/Reaction Factors into specific-intent plans that produced the fastest possible results, logically. So let's look at what basic facts about training we considered.

In most cases, muscle does not grow in a linear manner. When the issue is training stimuli and growth, we are actually talking about action/reaction and an adaptive process. Muscle mass accumulation is mostly dependant upon "weight-load range" and "work-load capacity". Let me explain.

An athlete may be able to bench press 300 lbs for 8 reps (1.5 second positive phase/2 second negative phase) before failure during the last rep's positive phase. And maybe able to follow this set with 300 x 7 and 300 x 6 with 2.5 minutes rest between each set. His "work-load-capacity" is the total number of sets and reps multiplied by the "weight-load" utilized.

So  $8+7+6=21$  reps total x 300 lbs =6300 lbs moved in a time period of 373.5 seconds. (Huh?) Assuming a rep speed average of a 2 second negative and 1.5 second positive, each rep would be about 3.5 seconds. So 21 reps x 3.5 seconds =73.5 seconds under load. The rest periods between set #1 & #2 and #2 & #3 total 5 minutes or 300 seconds. So his work-load capacity for 373.5 seconds is 6300 lbs.

This athlete may not realize significant muscular growth as the weight load goes from 300 lbs to 310, 320, 330, or even 340 lbs with the same rep speed and same total number of reps for 3 sets. However, the athlete may seem to grow almost over night, significantly, when the weight load reaches 350 lbs. This is because the existing muscle mass already possessed the **capacity** to move a weight-load **range** of 300-340 lbs for 21 reps total in 373.5 seconds. The muscle simply did not need to increase in mass as an adaptive response until its current load range and work capacity was exceeded.

Most strength gains are initially due to improved neuro-muscular efficiency of existing muscle mass. This in itself should explain one reason why testosterone was so effective at increasing net muscle mass. The administration of the drug all but guaranteed an increase in weight-load range and work-load capacity at an amazing rate while increasing post-training regeneration and recovery.

This was due to androgenic activity which rapidly increased strength, and anabolic activity that resulted in an adaptive response of an increase in protein synthesis. Action/Reaction, remember? Together these qualities aided in inducing a stronger stimulus and the resulting adaptation, or Action/Reaction. (Bigger and stronger muscles. "Duh" again)

Stimulation of muscle growth is actually all about muscle fiber recruitment. This means that a target muscle must be stimulated significantly by activating as many nerve motor units as possible in a given period of time. Sets that do not achieve positive failure also fail to induce significant motor unit activation/fiber recruitment.

To expand upon this, we should understand a basic training reality. If an athlete performs a set of 6 reps on a bicep curl using a weight load he could perform 10 or 12 reps with, the body restricts the number of motor units it will activate. If he follows this first set with another 6 rep set using the same weight load the body will incorporate more or additional fibers only once the previously activated fibers exhaust. This is due to the fact that the body is lazy and favors the easiest means of performing a task.

Since muscle tissue hypertrophy (growth) is an adaptive response to stimuli which exceeds current load range and work load capacity, it would require a very high number of sets to activate a significant number of motor units (fiber recruitment) and induce growth this way. The lesson here is that any set terminated prior to positive

failure will also fail to induce a maximal stimulus. An athlete must learn to train to positive muscular failure, not failure of spirit.

Earlier we discussed muscle fiber types. We know there are two fiber types, Type-I and Type-II with each consisting of sub-types. Basically Type I are endurance fibers with a low growth (size) potential. They respond to low weight loads, and sets that achieve positive failure above 15-20 reps. Type II fibers are strength orientated fibers with a high growth potential. They respond best to higher weight loads and sets that achieve positive failure below 15 reps.

Like I said, this is basic. Type II fibers are the primary focal point in bodybuilding since the goal is maximum size. This means utilizing a weight that achieves maximum fiber recruitment and achieves positive failure at 15 reps or below. However, there are three Type II fiber sub-types; a, b, and c. Each responds to different and somewhat specific rep/load protocols. (1) Type Ma are mostly effected by sets of 11-15. (2) Type Mb are mostly affected by sets of 6-10. (3) And Type IIc are mostly affected by sets of 1-5 reps.

Remember, these rep ranges refer to sets taken to positive failure. This is why most training systems call for sets that result in positive failure in the 9-12 rep range. Unfortunately 8-12 reps would mostly focus upon Type IIb fibers with some carry over effects upon Type Ma and Type IIc muscle fibers.

**"Lesson number two; To achieve maximum growth, maximum specific muscle fiber recruitment must be considered with specific intent."**

*Coach*

So what is "Synergistic Chaos Training?" It is the utilization of multiple training protocols with specific intent upon maximum fiber recruitment. These protocols must take into consideration chemistry synergy and induce a stimulus conducive to utilization of their potential. This means training in a rep range applicable to specific chemistry and intent while striving to increase load range and work load capacity beyond a muscle group's existing capability. The result is increased muscle mass and a new load range/work load capacity, or threshold.

The method in which the body gets stronger is mostly due to improved neuro-muscular efficiency. This is basic biological adaptations at it's best. To execute a set, the body recruits only as many fibers as is necessary to perform the task. When the set results in failure and the ensuing cellular damage occurs, the body reacts by increasing the stimulation of additional motor units to make the task easier or less traumatic next time. This in turn leads to an adaptation or improved neuro-muscular efficiency and a greater number of motor units/muscle fibers doing the task together.

More motor unit activation/fiber recruitment means a greater strength potential. This adaptation occurs over time and eventually leads to a target muscle exceeding its load range and work capacity resulting in muscular hypertrophy and hyperplasia.

**"Lesson number three; Don't be a sissy."**

***Coach***

Often we see a workout in a magazine or book calling for specific numbers of reps or rep ranges per set. The usual result is some would-be-bodybuilder bouncing a weight around to achieve the given number of reps. More recently we have seen articles listing rep speeds such as a 1.5 second positive phase and 2 seconds negative phase. Or the ridiculous of up to 30 seconds of each.

Let me simplify this by changing "reps" into "time under load". Type Ia fibers respond best to sets of 11-15 reps or a time under load of 40-60 seconds. Type IIb fibers respond best to sets of 6-10 reps or a time under load of 20-40 seconds. And Type IIc fibers respond best to sets of 1-5 reps or a time under load of 1-20 seconds. The positive phase should be explosive but controlled. The negative phase should be about 2 seconds.

**"Lesson number four, train the target muscle not the ego."**

***Coach***

It should also be obvious now that any training protocol can be altered or utilized for specific intent or even multiple goals. And so can any one set.

It should be apparent that magazines do not contain work-outs intended specifically for chemically enhanced athletes too often (Though few naturals could survive the total work-loads suggested). And if so, they are usually contest prep work-outs.

### **A POINT OF INTEREST**

A quick thought concerning low rep training protocols. When a set reaches positive failure in the 4-6 rep range and negative phases of each rep average 2 seconds, how explosive is the positive phase? Think about that while I babble and bitch a bit.

The other day my training partner and I are doing some heavy squats. My partner hits 5 reps at 605 lbs and I spot him for his 6<sup>th</sup> rep. The 5<sup>th</sup> was all he had and the 6<sup>th</sup> rep was obviously the actual failure rep. I spotted just enough to help him complete the 6<sup>th</sup>. He did the 5<sup>th</sup> rep, so that was not actual positive failure.

My turn! I un-rack and hit the first 4 reps solid. During rep #5, I'm digging hard but it's mine. I take a couple of breaths and start my 6<sup>th</sup> rep when an individual says

"hey, you already hit failure". Of course, any hard-core bodybuilder knows it is *very bad* to blow someone's concentration and that you have not "failed" if the rep was completed. By the way, an individual who hangs their towel on the bar end during your set is not only rude, but should be considered a food source.

Anyway, I rack and wait until the white buffalo clears from my head and ask the slender lad "What?"... (semi-politely) He explained that he is a personal trainer and that...

- (1) All reps should be a 2 second positive and negative each.
- (2) Failure is when you think you cannot do another perfect rep unassisted.
- (3) Positive acceleration removes stress from target muscles."

I only replied "force = mass x acceleration". Do the math. He was right to a point. Only when weight loads negate the ability to exceed 6 reps before positive failure, total explosion during a positive rep phase will always be a 1-2 second positive. The formula  $F = M \times E$  dictates the load target muscles move is greater than the load on the bar. But...thanks. Really nice kid!

## TRAINING DURING MAX ANDROGEN PHASES

It is important to realize that supraphysiological androgen (AAS) levels had three distinct effects upon muscle fibers. First, hypertrophy of Type II fibers was greatly elevated. Second, androgens, such as testosterone, increased the percentage/count of Type II fibers at the expense of Type I fibers.

This means testosterone altered the distribution of muscle fiber types in favor of size and strength fibers. It also is a fact that testosterone increased the amount of contractile proteins in muscle fibers (which was a function of the first effect of testosterone). These two combined effects added up to significant size and strength increases.

Third was the effect androgens had upon satellite cells. Satellite cells are muscle cells that are metabolically active yet merely sit on the outside of muscle fibers and therefore are not able to contribute to fiber contractile function. They sit there just waiting to join a fiber type so they too can grow and move freaky weight-loads.

Androgens increased the number of satellite cells. These newly formed cells, due to the effects of testosterone and other androgens, had more androgen receptor-sites when compared to other or prior existing muscle cells. This meant that the newly formed cells were even more receptive to the influence of androgens.

Once joined to a fiber, these satellite cells contributed to strength and size while having an even greater growth potential. To some extent androgens stimulated this fiber/satellite cell joining process as well. (But not as well as some other muscle chemistry did)

So the training stimuli during Max Androgen Phases had to be geared toward maximum use of these effects. This means maximum stimulation of Type II a, b and c fibers in an over-load format. This was required to accomplish Frank's goal of forcing the muscles to react to the training action by adapting through an increase in Type II fibers while also increasing total contractile proteins.

Over-load training protocols focused upon Type II a, b, and c fibers were not difficult to construct. Remembering the fiber type response/activation factor discussed earlier it seems obvious a few rules had to apply.

1. Work-sets achieved positive failure in a rep range of 4-15 reps. This meant that higher reps, or training techniques which extend total rep count above this range were most likely to be less productive. Remember: Frank's goal was long-term results, not merely momentary gains.
2. Work-outs had to end in an hour or less so as to avoid a fiber recruitment transition toward endurance or Type I fibers.
3. Aerobics were best terminated during Max Androgen Phase high activity periods. This was similar to the prior rule in that again an adaptive response to aerobics is an increase in Type I fiber activity.
4. Rest between work-sets was best set at about 2 minutes to 3.0 minutes. This length of rest period was necessary to allow cellular regeneration of ATP from CP to reach near highest cellular levels. Note I said "near". Total ATP recuperation/regeneration allowed Type IIc fibers to fully regenerate. This would have drawn stimuli intensity away from the real growth fibers, Type IIa and b. Type IIc muscle fibers are mostly strength oriented fibers with less growth potential. It is important that the reader realizes that some stimulation of Type IIc muscle fibers was needed for maximum growth. Remember the discussion on prior pages number concerning work-load capacity and weight-load range? (If not, go re-read it)
5. Total work-sets per muscle group were kept at 10-12 sets for larger muscle groups, and 6-10 for smaller muscles. Work-sets (not including warm-up sets) had to achieve positive failure, or beyond. "Beyond Failure" techniques such as forced-reps, drop-sets, rest/pause, or low-rep-same-muscle-super-sets are examples of beyond failure traumatic sets. Compound exercises worked best.
6. Individual muscle group PTOR was accounted for. Lagging body parts had to be trained more often but with less traumatic sets such as straight set techniques: 10 x 10 (10 sets-10 reps each set) and isolation type exercises. (This was excluded if a Max Androgen Phase was utilized as a site-injection protocol since circulatory androgen supply would not be an issue.) So each body part was trained once per week with traumatic sets. A lagging body part was trained twice per week with low traumatic sets.



## **TRAINING DURING CORTISOL/ESTROGEN SUPPRESSION PHASES**

The problem some beasts experienced during Cortisol/Estrogen Suppression Phases was joint pain. This was due to inhibition of the body's natural inflammatory response system that normally would aid in joint function. So it seems obvious that heavy training could have been counter productive during these phases, for some.

But remember, since catabolic responses or the catabolic side of the anabolic/catabolic ratio was suppressed, pretty much any challenging training stimuli resulted in elevated site-specific anabolism, or depending upon diet and Phase Cycle, maximum lean tissue retention.

The body maintains homeostasis through action/reaction factors to any given stimuli. Continuous over-load or high intensity training will eventually result in over training, injury, or growth plateaus with subsequent strength and lean mass tissue loss. However it is possible to beat homeostasis, as we all know by now, through working with instead of against the body's natural Action/Reaction Factors. This is based upon the ideal of adaptation. Initially the body adapts to continuously increasing weight-loads and training intensity by up-regulating anabolic chemistries in excess of catabolic chemistry.

The prior point referred to both chemically enhanced and natural athletes alike since the chemistry up-regulation I refer to is predominantly endogenous co-factors such as enzymes. Within 2-3 weeks however, the body is able to begin over coming this training induced action by reacting with excess catabolic responses.

By cycling Max Androgen Phases, Absolute Anabolic Phases, and Cortisol / Estrogen Suppression Phases, Frank was able to either super charge each muscle growth action (while either suppressing or defeating negative feed-back loops) or react "with" the body to utilize the feed-back loop to his advantage.

Training during Cortisol / Estrogen Suppression Phases was best utilized as an adaptive period by unloading the training stimuli about 10-15%, depending on how joints responded. This was a matter of simply reducing work-set weight loads and/or intensity about 10-15 %.

This allowed for physiological and psychological rest and adaptations. This in turn prepared the body and mind for the next high growth period. This was also a great time to take a look at lean mass results acquired during prior high growth periods (such as symmetry and muscle shape)

Additionally it was realized that an over-all improvement in musculature quality was the result from the current Cortisol/Estrogen Suppression Phase. Of course the added plus of less bodyfat to diet off later due to estrogen inhibition was pretty conducive to long term progress too. (Less fat to lose meant less chance of lean mass tissue loss. Gee, ya think?)

**The goals during Cortisol/Estrogen Suppression Phases were pretty simple:**

- (1) Work-sets reached positive failure at rep 10-15.
- (2) Work-outs periods were terminated before 90 minutes.
- (3) Total work-sets per body part was best set at 1 2-1 5 for large body parts and 10-1 2 for smaller ones.
- (4) Few, if any, high intensity/traumatic techniques were utilized.
- (5) At least 25 grams of Glutamine was ingested daily.
- (6) 5 grams of Vitamin-C was ingested daily.
- (7) MSM, chondroitin sulfate and Glucosamine Sulfate was ingested daily at a rate of about 2-3 grams each daily.
- (8) Each body part was trained every 3-5 days.

## **TRAINING DURING ABSOLUTE ANABOLIC PHASES**

Just as androgens had distinct effects upon muscle fiber type, so did Absolute Anabolic Phases. GH and IGF-1 increased structural proteins therefore adding mass or size with little results in the way of strength gains...initially. GH also provided a strengthening and repair quality for soft tissues such as tendons, ligaments, and cartilage.

Another quality or effect of GH and IGF-1 protocols was hyperplasia of muscle fibers, predominantly type I and transitional fibers. Hyperplasia is a condition where the number of muscle cells and fibers increase. (Remember satellite-cells and androgens? Well, this is the "other chemistry" I mentioned) This was mostly due to incorporation of satellite cells into existing fibers.

More cells more fibers, meant more growth potential. These newly formed fibers were transformed into Type II fibers during periods of Max Androgen or other AAS phases and protocols.

Specific training stimuli has been noted to increase the hyperplasia phenomenon as well as aid in some Type I muscle fiber transition into Type II fibers. Insulin significantly aided in this Action/Reaction Factor by assuring a more than adequate nutrient supply for growth. So did thyroid hormones such as T-3 and T-4, but only when the calorie intake exceeded metabolic needs and there was an excess of amino acids (from protein) constantly available.

Theoretically, during diet or calorie restricted periods, the inclusion of GH alone (without androgens) would have caused an increase or sustaining effect upon Type I fibers at the expense of Type II fibers. But the theory is based upon total macronutrient intake down-regulation. Obviously inadequate amino acid levels would have allowed endurance oriented Type I fibers a survival advantage.

So training for best long-term results while utilizing GH, Insulin and / or Thyroid Hormones in the absence of androgens, PGF-2, PGE-2, Interleukin-15, or MGF had to meet the following requirements:

1. A work-set rep range that stimulates Type II fibers for growth and sustaining effects, and sets which stimulate Type I fibers. This was easy to accomplish. Frank could have trained a body part with work- sets that reached failure at 4-10 reps for 2 exercises and then done work-sets for 2 exercises utilizing a higher rep range of 12-20 reps. But there were more effective methods: Triple-drop-sets where each weight can be utilized for 4-7 reps to achieve failure before each drop. This allowed a total rep range of 12-21 (or more) reps per set.

These were very traumatic sets. Low/High sets. These are sets that an initial weight was utilized that resulted in failure at 6-10 reps and then the weight was reduced quickly 50 % and failure was achieved again. The rep total per set sometimes reached as high as 30 with some individuals. Same-Muscle-Super-Sets were used as a Low/High set or as a straight failure at 6-10 reps per exercise set. Examples of same-muscle-super-sets are heavy flat bench (failure at 6-10 reps) and incline flys (failure at rep 6-10, or heavy squats (failure at rep 6-10) and sissy squats (failure at rep 6-10). Personally I liked a mixture.

2. Stretch position exercises had to be utilized for each body part to assure the highest IGF-1 and MGF production. Stretch position exercises are those that result in an extreme stretch of the target muscle at the end of the negative phase of each rep. Example: Quads-sissy squats. Pec any fly, cable-cross-over, or pec-deck exercise. Lats-lying DB pull-over, straight-arm-pull-downs. Biceps-incline DB curls or incline-low-pulley-cable-curls. Triceps- long-pulls, standing DB or EZ bar tricep extensions. Hams-straight leg dead lifts, or good mornings. Calves-donkey calve raises. Lateral Delts-incline cross-body DB side laterals.

An easy way to incorporate stretch exercises is super-sets such as those listed earlier. Simply stated, it would be a matter of super- setting a mid-range compound exercise with a stretch position type exercise. It should be obvious that this will also aid in fascia stretching to some extent.

3. Most best Incorporated anaerobic / aerobic interval training 3 times per week for 20-40 minutes per session. Studies show that the body adapts to aerobic training within 6-8 weeks. This is a matter of capillary and cardio pulmonary adaptations, which of course aid in increases in vascular tissue growth and nutrient supply. It also aids in both type I & II fiber recruitment. Frank could have done 20-40 minutes of kick boxing heavy bag work, play hand-ball, or alternating intensity levels on a stair climber for 20-40 minutes. It was a simple matter of high intensity for 40-60 seconds coupled with easier semi-resting intervals of 1-4 minutes. This was the most effective fat burning/lean mass retention method I know of, by the way. Body fat levels above 12 % just are not healthy or acceptable.

4. Phases that had PGF-2, PGE-2, androgens, or Interleukin-1 layered into them incorporated these goals also. However since these drugs significantly affected increases in contractile proteins and Type I to Type II muscle fiber transition, the first exercise for each body part was a lower rep rest/pause compound exercise technique. It is important to realize Absolute Anabolic Phases had a main goal of increases in total muscle cell and fiber count while increasing vascular tissue. This added significantly to any Max Androgen Phase effectiveness as well as aiding in vascular tissue growth into new areas created by site-injection protocols. If these goals were not accomplished, long term results were nowhere near as amazing as they could have been. Also I should add, I have witnessed several hard-core types adding over 2" to their arms on cycles containing PGF-2, Insulin and GH when properly structured. This was in addition to serious growth throughout their musculature and freaky hardness even on very high calories.

### **ANY ABSOLUTE ANABOLIC PHASE PROVIDED EXCELLENT RESULTS WHEN THESE GOALS WERE MET AS WELL:**

- > Total work-sets for large body parts had to be set at 8-10 sets if traumatic sets such as triple drops, low/high, or rest/pause were the main exercise choices with 6-8 sets for smaller body parts.
- > If lower traumatic straight sets were the main exercise choice, large body parts had to receive 12-15 sets and small parts received 10-12 sets.
- > Beasts trained each body part every 5-7 days depending on recovery /diet.
- > Kept work-outs under 60-75 minutes.
- > All beasts took a complete rest day at least every 4<sup>th</sup> day.
- > Rested 90-120 seconds between work sets.
- > Go for the burn Baby! That burning sensation triggered localized IGF-1, and MGF production as well as an increase in GH conversion to other growth factors.
- > Work-sets and exercise choices allowed for failure at multiple rep ranges. (4-12 and 12-20 reps).

## Things To Think About:

### ZINC: The Anabolic Activator

The mineral zinc is at last receiving the respect it deserves from the athletic community. A now famous study conducted at Western Washington University evaluated the hormonal effects 8 weeks of nightly supplementation provided. A group of football players were divided into two test groups that either received the supplement or a placebo.

The testing procedures included a double blind and randomized protocol. Since most athletes are zinc deficient, the results were not all that surprising. The supplemented group, receiving 30 mg Zinc Monomethionine, 450 mg Magnesium Aspartate, and 10.5 mg Pyridoxine (Vitamin B-6) nightly, showed a blood plasma increase in both total and free (active) IGF-1.

The placebo group showed an effective decrease in both hormone forms. This study triggered the mass marketing of high absorption zinc supplements like Z-MASS and ZMA. Strangely enough, the supplements actually work in an accumulative manner for so-called naturals and chemically enhanced alike. The strange part is that the supplement industry is right in a percentage of these product claims. Imagine that!

### THE MANLY MINERAL

Zinc should be called the **MANS MINERAL**. Its presence in the body literally makes the difference between hormone profiles that put you in either high heels and sweater puppets, or baritone bad boy biker status. Zinc is a key factor in enzyme activation's relating to the anabolic potential of testosterone, Insulin, IGF-1, GH, Prostaglandins, and about every other hormone or hormone-like substance relating to muscle growth.

If you have not as of yet realized how manly zinc is, try this. Without adequate zinc supplies, you would not have a penis (if you are male) or enough muscle to power the hand to scratch it with. "Optimal " levels of Zinc are absolutely vital to manhood and bodybuilding progress. .. even for women. (The bodybuilding part)

### ZINC FACTS

Zinc is an enzyme activator intricate to somewhere between 200-300 enzyme/ zinc dependent reactions in the body.

- > Zinc deficiency can and will result in a reduction in an androgen receptor count of 20-40 % and an increase in estrogenic receptor count of 40-60 %
- > Zinc deficiency can cause up to a 63% reduction in androgen binding sites which can cause gyno by altering the androgen-to-estrogen activity ratio, (sweater-puppets?) Zinc deficiency increases the aromatization of androgens to estrogens.
- > Some AAS reduce the level of Zinc in seminal fluid after 14 days of administration. This is an additional inhibitory factor upon HPTA function.
- > Prohormones such as 19-Norandrostenedione, 19-Norandrostenediol, 4-androstenedione, and 4-Androstenediol (and most other variants) are dependent upon the enzymes 17-BHSD and 3-BHSD for activity and conversion to more powerful androgens. Both 17-BHSD and 3-BHSD depend upon zinc.
- > Zinc aids in immune response.
- > Zinc acts as an anti-oxidant by way of the "zinc activated" superoxide-dismutase system. The system helps protect cells from oxidation.
- > Zinc deficiency prevents pancreatic beta-cells from storing or secreting Insulin.
- > Zinc is necessary for protein metabolism.
- > Zinc is necessary for DNA transcription and cell division.
- > The prostate has the highest level of zinc concentration of all body organs, and it does not play nice if zinc deficiency is excessive and prolonged.
- > Lactate Dehydrogenase and Carbonic Anhydrase are enzymes that allow the body to process lactic acid and carbon dioxide. Both are zinc dependent.
- > Blood Cortisol levels elevate significantly during times of zinc deficiency. This hinders GH release and IGF-1 formation. And this negatively alters the testosterone-to-cortisol ratio.
- > Zinc is needed for vitamin A metabolism.
- > Zinc is intricate to the production of sperm and testosterone.
- > The thymus gland shrinks if circulatory zinc levels and reserves are low. The thymus gland produces immune T-cells that protect against tumors and disease.
- > The body contains about 2000 mg of zinc. Most is stored in the bones and muscles. Zinc is transported in blood as a component of red blood cells. Of course

some other blood proteins carry zinc to other body tissues as well. About 55% of the body's zinc is stored in slow twist muscle fibers.

- > Plant phytates and fiber block zinc absorption because the alkaline PH environment of the small intestines (where most zinc is absorbed) allows zinc and the phytates to complex, or become joined as an insoluble compound. Phytates are found in both grains and vegetables. In the presence of calcium, zinc will pass through totally unabsorbed. (Vegetarian Diets suck!)
- > As a rule, only about 20 % of a single dosage of zinc is absorbed and the larger the dosage the lower the percentage of single dose absorption. Citrate, Vitamin B-6, picolinic acid (a metabolite of Tryptophane) all increase absorption. Zinc gluconate is highly absorbed.
- > The toxic level/dosage of zinc sulfate for a 200 lb male is almost 20 grams (19,890 mg).
- > The liver enzyme 5-deiodinase, that converts the thyroid hormone T-4 into the five time more active thyroid hormone T-3, is zinc and selenium dependent. During low carbohydrate diets or calorie restricted periods (or supplemental T-4 use) adequate zinc intake prevents 67 % of the normal decrease in 5-deiodinase activity. Protein synthesis is dependent upon thyroid hormone activity.
- > Adequate zinc levels increase free (active) AAS plasma levels during cycles and decrease HPTA inhibitor.

Personal experience and blood test results had led me to strongly support the use of am and pm dosages of zinc supplements both during and after AAS protocols as well as the co-factors magnesium, selenium, and vitamin B-6. Care should be taken to assure adequate copper intake with zinc (1 mg copper per 15 mg zinc). ZMA products too. But, of course, few will even read this section anyways. SISSIES! As to daily dosages, the Western Washington University study was an apt guide-line for beasts.



## **INTERVAL AEROBIC/ANAEROBIC TRAINING BURNS MORE FAT AND SPARES MUSCLES.**

Readers of previous CME books have commented upon my dislike for aerobics as a means of oxidizing (burning) fat stores. Normally I simply defend my views with "boring and ineffectual". Why?

The place where fat is burned or oxidized is the cellular mitochondria. There are two primary types of mitochondria; S-mitochondria that is found in the connective tissue of muscle (sarcolemma), and M-mitochondria found in the muscle fibers themselves (myofibrilla).

Standard aerobic training mostly favors beta-oxidation (fat burning) in S-mitochondria, where as near-anaerobic-threshold- training favors beta-oxidation in M-mitochondria. Near-anaerobic-threshold-training is defined as the point in which muscle fatigue is induced by the build-up of muscle by-products such as lactic acid (feel the burn baby!) caused by increased exercise intensity. About 10 % of total muscle mitochondria exists as S-mitochondria, and about 90 % exists as M-mitochondria.

This in itself should prove near- anaerobic-threshold-training is 9 times (900%) more effective as a means of causing beta-oxidation (fat burning) when compared to standard aerobic training. The mitochondria ratio/stimulation factor is cool, but there is more.

When near-anaerobic-threshold-training is utilized, a build-up of citrate and lactate in muscle tissue results. Both of these by-products inhibit glycogenolysis, which is the break-down of muscle glycogen and glucose. When glycogenolysis is inhibited, an elevation in plasma GH levels is realized thus forcing the muscles to rely more on fat (from beta-oxidation)as an energy source.

These fats for energy come from circulatory free-fatty acids and stores of muscle triglycerides. How? (Be patient) When we exercise intensely, insulin secretion is suppressed by a group of chemicals from the adrenal gland called catecholamines. You probably know them as either epinephrine and norepinephrine, or adrenaline and noradrenaline. These catecholamines in turn trigger the release of free fatty acids (from bodyfat stores) into the circulatory system by stimulation of hormone-sensitive-lipase-enzyme found in lipocytes (fat cells).

The greater the mitochondria stimulation (or number of mitochondria stimulated), the greater the amount of fatty acids /triglycerides burned as fuel. Since about 90% of mitochondria exist as M-mitochondria, (stimulated by near-anaerobic-threshold-training) and the level of intensity increases, the level of catecholamines released ...duh, you do the math.

Additionally realize that by structuring near-anaerobic-threshold-training into interval aerobic/anaerobic training, your body becomes much more efficient at breaking down all the nasty fatigue produced metabolic by-products.

Why interval aerobic/anaerobic training? Who can handle 20-30 minutes of consecutive near-anaerobic-threshold-training?

So how did we structure this into a realistic training protocol for Frank and the rest of the beasts? (Sex is still best!!)

- Find a high intensity aerobic activity you enjoy, or hate less than others.
- Find your maximum heart rate ( $220 - \text{your age}$ ) and subtract 10%. Example:  $220 - 30 = 190 - 10\% = 171$  heart rate.
- Do 2-3 minutes of near-anaerobic-threshold-activity at a heart rate of maximum minus 10%.
- Lower the intensity to 50-60 % of maximum heart rate until you feel sufficient recovery.
- Repeat for a total of 20-30 minutes 2-3 times per week.

Interval aerobic/anaerobic training burns 100-120 % more fat in 30 minutes than traditional aerobic training burns in one hour. And Frank's lean muscle mass was preserved or increased depending upon diet/calorie structure and co-administered chemical muscle enhancement.

**"IMPORTANT: Never attempt this or any other training technique without a doctor's approval (Weasel Statement)**

## MORALS OR DOGMA/TRUTH = POLITICAL AGENDA?

When I first agreed to write about the strategies and protocols that had been employed to induce chemical muscle enhancements, the political and media dictated morals were of issue. To say the least, any dogma that can subjugate the AMA is persuasive. And to be blunt, such tactics as those integrated into law to punish medical professionals for remaining true to their oath, are insane.

The obvious conclusion being that anabolic / androgenic steroids (AAS) such as testosterone are deadly and evil. Evidently all things that produce, convert to, or increase, testosterone levels within ones body must also be satanic to some extent. Why else would such practices as male bashing (they produce testosterone) be legal? (Watch any day-time-whine-and-talk-show)

For the above reason I strongly suggest readers should review the following reference sites and sources so I can get on with this book. My morals rest in truth. (Lots more at the end of course)

- 1. Does testosterone cause "roid rage"?** No, just an improved mood, self- confidence and feeling of well being. Oh, and heightened libido similar to an average 14 year old boy with a Playboy Magazine.  
\*\*Rabijewski, M., et al (1998) Pol Arch Med Wew N100 (3):21 2-21  
\*\*Almeida, O.P. (1999) Arg Neuropsiquiatr. 57 (3a): 701-6  
\*\* Tricker, R., et al (1996) J. Clin. Endocrinol Metab. 81 (10): 3754-8
- 2. Does testosterone increase the risk of heart disease?** Difficult question, but several studies show that testosterone actually reduces heart disease risks and complications.  
"Shapiro, J. et al. (1999) Amer. J. Ther. 6(3): 167-74  
\*\*Zmuda, J.M., et al. (1997) 130(1-2): 199-202 (Atherosclerosis)  
\*\*Zmuda, J.M., et al. (1997) AM. J. Epidemiol. 146 (8): 609-617  
\*\*Salke, R.C., et al (1985) Med Sci. Sports Exer. 17 (6):701-4
3. Actually, testosterone has been shown to have a positive effect upon high-density-lipoprotein (HDL) cholesterol levels.  
\*\*Zhao, S; L; X; and Wang, Z., (1998) Hunan I Ko Ta Hseah Pao. 23(3): 299  
\*\*Barrett-Connor, E. I. (1995) Diabete. Metab. 21 (3) : 156-61
4. Testosterone has been shown to significantly improve chronic angina as well as myocardio ischemia from exercise.  
\*\*English, K.M. et al. (2000) Circulation. 102(16):1906-11
- 5. Speaking of heart risk factors, doesn't testosterone and AAS raise low density-lipoprotein (the bad cholesterol-LDL) and Lipoprotein (a)?** Actually the opposite is still the truth. Testosterone has been shown to lower LDL and Lipoprotein (a)  
\*\*Zmuda, J.M. et al. (1996) AM. J. Cardiol 77(14): 1244-7  
\*\*Rabijewski, M., et al. (1998) Pol. Arch. Med. Weun. 100 (3): 212-21  
\*\*Uyanik, B.S., et al. (1997) Jpn. Heart J. 38 (1): 73-82  
\*\*Zgliczynski, S., et al. (1996) Atherosclerosis. 121 (1):35-43.

6. **Testosterone reduces to DHT (dihydrotestosterone) so testosterone will increase the risk of prostate cancer, right?** It should be of interest to hear that DHT has been patented as a treatment for BPH (Benign Prostate Hyperplasia). However several studies have shown that estrogen elevation and subsequent interaction with factors of DHT activity cause the negative effects in prostate size and PSA levels. (Prostate-Specific-antigen) (Is estrogen evil?) "Kenny, A.M., et al. (2000) Endocr. Res. 26 (z): 1 53-68 "Cooper, C.S., et al. (1 998) J. Urol 1 59 (2): 441 -3

An interesting note concerning prostate cancer is that 2 out of 3 individuals with PSA levels high enough to indicate cancer of the prostate, test negative in pathology. Scary huh? Oh, this is after prostate removal surgery. That could be a pain in the ass!

Additionally, a study by **J. Ghosh et al (1998) Proc. Nati. Acad. Sci. USA 27: 1382-7** showed another interesting aspect of prostate cancer. It seems that inhibition of the enzyme arachidonate-5-lipoxygenase (5-LO) triggers massive apoptosis (suicide) in human prostate cancer cells. 5-LO is an enzyme that acts upon a fatty acid called arachidonic acid that is found in all animal fats and induces proliferation of prostate cancer cells. It is also a substrate for some types of prostaglandin production.

## **AAS AND THEIR MULTI-MECHANISMS OF GROWTH**

There seems to exist an over simplification concerning the mechanisms by which AAS trigger muscular hypertrophy or growth, common to most literature on the subject. In most cases, it should be assumed that the authors of these simplifications did so as a means of generalizations for a purpose referred to as "dumbing it down".

As a whole, it is believed that most readers are idiots waiting to be told what to think and do, rather than educated through truth and "necessary" information. Some authors lack personal experience or take research as conclusive rather than realizing that calling a cat a dog won't make it bark. Please realize that many writers are just boring or scared to say "fuck you" when they mean it. Some are **great**, and their simplifications are for the generalization purpose.

BUT the statement "AAS induce growth solely due to molecule mergence with an androgenic receptor-site and subsequent message transcription" is totally unacceptable. If we were to leave it at that, my books would have the value of toilet paper. So "fuck them", and lets discuss the primary and secondary mechanisms and factors relating to maximizing AAS potential. (This info applies to natural hormone profiles also.)

### **PRIMARY MECHANISMS AND FACTORS**

The primary mechanism by which AAS trigger growth or muscular hypertrophy begins when an AAS molecule merges with an androgen receptor-site on the exterior of a cell. If the cell contains adequate ATP reserves, the receptor-site/molecule complex travels to the interior of the cell where it delivers a "make new proteins" message via DNA/mRNA transcription. Basic, but it barks because it "is" a dog. This is the primary anabolism mechanism since it directly stimulated protein synthesis.

The other primary mechanism by which AAS increase net cellular proteins concerns cortisol. When AAS molecules merge with a cortisol receptor-site, nothing happens. (Huh?) That is the point. Since the AAS molecule acts as a cortisol receptor-site antagonist, it triggers no response while also locking cortisol molecules out of the receptors it occupies. The result is an increase in net proteins due to retention. And that is an anti-catabolic effect, so it is called the primary anti-catabolic mechanism.

However, there are several factors that determine the potential magnitude of total net anabolism achieved by any AAS. These factors correlate with a drug's total potency.

## FACTORS OF POTENCY

**Binding Affinity:** Different AAS chemical structures or deviations either increase or decrease the molecule's attraction to its respective receptor-sites. The greater the affinity the specific AAS possess the greater the chance of a higher rate of molecule / receptor-site mergence.

**Binding Time:** Each variant of AAS has a common binding time unique to its structure. This means that the molecule binds to its respective receptor-sties, both androgenic and corticoid, for a given period of time. The longer the binding time, the greater the period of activity. This also affects a drugs anabolic/catabolic ratio.

**Percentage Bound/Unbound:** All sex steroids and hormones exist in the body in either a bound or unbound state. Kind of like "The Taming of O", but on a much larger scale. Bound refers to the inactive portion of a given level (amount) of an AAS or natural hormone. Unbound refers to the free or active portion. In both cases, we are referring to the circulatory or blood plasma levels.

Only the free unbound AAS molecules can merge with a receptor-site and therefore be termed as active. The bondage team that binds hormones are blood proteins called Sex-Hormone-Binding-Globulin (SHGB) and Albumin. Different alterations in chemical structures, either man-made or naturally occurring, are either more or less resistant to binding. This means alterations in an AAS chemical structure could allow it to exist in a greater percentage of unbound/active state. So less could do more.

Testosterone circulates in a bound state at a rate (or percentage) of 98-99% and an unbound state at a rate of 1 -2%. For the curious, 65% is bound by SHBG, and 33% is bound by Albumin, plus or minus 1%.

So the higher the percentage of an AAS circulating in the blood stream in the unbound/active state, the greater the number of potential bindings /mergence with receptor-sites.

**Active-Life/Half-Life:** All drugs have an active-life and a half-life. Knowing a drug's average *effective* active-life and half-life is important information. An active-life is the entire period of time a parent drug remains active once administered.

A drug's half-life is one half of its active-life and the period of time required for half of its administered dosage to disperse and metabolize. Simple huh? It should be but it actually is not. Stay with me here.

Using testosterone enanthate as an example should explain this more effectively. Testosterone Enanthate has an "effective" active-life of about 8 days and a half-life of about 4 days. Once a 200mg dosage of testosterone enanthate has been administered, it will take about 4 days for 100 mg of the administered dosage to disperse from the injection site and reach peak activity. This is the half-life point.

After the peak activity point/half-life, the second 100 mg disperses over the following 4 days. The two 4 day periods make up the 8-day "effective" active-life. That explanation would seem idiotic if equal daily dispersal rates actually existed, and it would be nice.

Actually, from the point of administration of the 200 mg dosage, the amount of testosterone that is released from the injection site gradually increases until it peaks at the half-life. After the peak or half-life, the drug is less and less active (less anabolic/less androgenic/less anti-catabolic) until it is completely dispensed and metabolized.

If the goal is a more even dispersal rate, a drug must be administered on a repeat basis at its average half-life point. This is true regardless of method for drug administration. It also allowed for pre-chosen peaks and dips in a drug's activity for specific-intent, and the ability to structure protocols for specific interests or Action/Reaction Factors.

**Specific Activity of Parent Drug and Metabolites:** A parent drug is the actual drug or chemical structure initially administered. A metabolite is any one of possible several various chemical structures created when a parent drug is metabolized. Many metabolites are active and can possess a higher or lower potency than the original parent drug.

Additionally each metabolite may provide either an additive or mitigating effect upon its parent drug. An example of this is what occurs when ethlyestrenol (Orgabolin/Orabolin) is ingested. Due to metabolic factors, it is partially converted into a more powerful metabolite called northrandrolone (Nilevar).

The activity of the two chemicals together is greater than the parent drug alone. It is important to realize that different metabolic environments can be created, and each environment can have a positive or negative effect upon over-all drug metabolism.

An example of the metabolite mitigating effect can be seen with the aromatization of testosterone into estrogens. Testosterone itself is highly androgenic. But due to a high rate of metabolization to estrogens, it also induces estrogenic activity that mitigates some of the androgenic activity. This example applies only if the intended action factor is the androgenic factor of testosterone alone. As we know by now, estrogen can have significant benefits when utilized properly.

And that too is an issue of the specific activity of a parent drug and its metabolites. But in this case we are dealing with a secondary mechanism. Remember "specific-intent"?

## **SECONDARY MECHANISMS AND FACTORS**

The secondary mechanisms by which AAS induce growth or muscular hypertrophy deal with a given drug's capacity for conversion or reduction into other hormones. This also applies to the cascading effect resulting from the parent drug and conversion/reduction product. Some of these mechanisms are very synergistic to the growth process and had to be taken into consideration when constructing cycles and protocols for beasts. Others may have inhibited a chosen outcome or goal by rendering one or more drugs in a stack useless.

**ESTROGENIC CONVERSION RATE:** Most AAS are susceptible to conversion into an estrogen. Like different AAS possess different levels of activity, so do different estrogenic conversion products. AAS can be converted into estrogens by two main pathways. The most commonly discussed pathway occurs when a susceptible AAS molecule encounters the aromatase enzyme. The conversion process is called aromatization, of course.

In most cases, individuals with higher levels of body fat will experience greater levels of AAS aromatization due to the fact that lipocytes (fat cells) are primary producers of the aromatase enzyme. This makes sense because estrogens are anabolic to lipocytes and all cells possess certain survival mechanisms.

The second conversion pathway is activated during liver/intestinal deactivation of susceptible AAS. Various P-450 enzymes induce a conversion process that results in a percentage of the AAS being metabolized into estrogens. Some AAS are more or less susceptible to estrogenic conversion and therefore convert at a higher or lower rate or percentage. And the method of administration can effect this as well.

As example, testosterone in its unaltered form converts to estrogens at a much higher rate if ingested than if it is injected. The same is true of some pro-hormones. The estrogenic conversion rate of a drug or its metabolites maybe either beneficial or counter-productive, depending on the intended outcome or goal of the protocol.

Estrogens increase GH and IGF-1 production by initiating a cascading effect beginning with the hypothalamus. The synergy between GH, IGF-1, and AAS is well known. The issue of estrogenic value was always a matter of ratio between androgens and estrogens.



Estrogens act to trigger another cascading effect by up-regulating production of an enzyme necessary for glycogen synthesis and intracellular storage in muscle tissue. The resulting increase in cellular glycogen content allows for prolonged ATP production during anaerobic activities such as weight training and subsequent recovery process. Both require a great deal of ATP. (We will discuss that issue in depth later.)

Additionally, each gram of glucose stored as glycogen in muscle tissue brings with it 2.7-3.0 grams of water. This triggers osmotic anabolism similar to "one" positive factor of creatine supplement use. This simply means cells are super-saturated with growth nutrients intracellularly. The osmotic effect also increases localized IGF-1 production due to the increase in pump and stretch realized during conditions of elevated glycogen storage. (Guess that explains the cookies-n-cream ice cream carb-loading during women's periods)

Estrogens increase anaerobic weight-load capacity. This is due to an increase in the whole body water table that results in an increase in muscle integrity and joint stability. Simply said, it allows for a greater and sounder foundation from which to lift weight from. Greater weight-load capacity means more weight used and a greater stimulus for anabolic over-compensation during recovery. GROWTH!

The mitigating effects of estrogens should seem evident. Gyno, increased fat synthesis, and water retention are not pre-contest goals. The issue during mass gaining periods therefore became estrogen control, not elimination.

**Reduction to DHT or Derivative:** DHT (Dihydrotestosterone) is 3-7 times more androgenic than testosterone. High androgenic AAS tend to promote an increase in total muscle glycogen synthesis. The result is similar to the prior mentioned osmotic effect.

It differs however due to the lack of estrogenic activity which would normally draws water into the entire body water table. In this case, due to no aromatization, the high androgenic value draws water from under the skin and forces it "into" muscle cells. This is why drugs that are structurally similar to DHT such as Parabolan, Halotestin, and Masteron, were used pre-contest to create hardness of the complete musculature and increased viewable vascularity.

Many AAS are susceptible to a percentage of enzymic reduction to either DHT or a metabolite/derivative of DHT. This is the result of a susceptible AAS molecule encountering the enzyme 5-alfa-reductase. The majority of 5-alfa-reductase is found in sex-specific tissues such as hair follicles, and prostate cells. Some does exist in muscle and the vascular system of course.

Once an AAS molecule encounters 5- $\alpha$ -reductase, whether in the vascular system or in sex-specific tissues, it is reduced to a derivative of DHT corresponding with its unique chemical structure. As example, Nandrolone reduces to NOR-DHT. Therefore the resulting reduction products may be more or less androgenic than actual DHT. This means the new product can have a more or less potent androgenic effect upon tissues it merges with.

Since different AAS reduce to DHT at different rates or percentages, each can trigger a growth or androgenic mechanism to a greater or lesser degree of a secondary nature. This of course, also is dependent upon the potency of the specific reduction product as well. It should be noted that some DHT reduction products can possess an inhibitory effect upon estrogens.

**Of course there are other pathways and mechanisms by which AAS induce the growth process.**

- When 17- $\alpha$ -alkylated steroids are detoxified/deactivated by the liver there is a significant increase in hepatic IGF-1 production and secretion.
- AAS increase CP (Creatine Phosphate) synthesis.
- AAS increase circulatory amino acid (s) levels by redirecting amino acids away from liver oxidation / destruction.
- AAS susceptible to estrogenic conversion suppress cortisol levels and activity by stimulating GH/IGF-1 production.
- AAS inhibit the synthesis of fat by blocking the enzyme lipoprotein lipase which in turn spares calories that can be utilized for the growth process of muscle tissue.
- Some AAS increase muscle cell PGE-1 and PGF-2 receptor-site count/concentrations.
- By stimulating an increase in nitric oxide synthesis, AAS increase vascular endothelial growth factor (VEGF) which is responsible for capillary formation. Nitric oxide also positively effects erectile function. (Think about it)

There are several other mechanisms by which AAS induce or mediate the growth process but for now we will simply accept that a foundation for greater knowledge has been laid down. And it barks like a dog because...

## **CYCLES AND CYCLES**

We have discussed the benefits and methodology of Max Androgen Phases that had employed different variants of high anabolic/high androgenic testosterone up in the front of the cycle, and a high anabolic/moderate-to-low androgenic at the cycle exit. Growth potential during these brief protocols was nothing short of miraculous in most cases. Normally Max Androgen Phases were at a point of high activity for 30 days or less, thus functioning within the basic Action/Reaction "initiation" time period of 2 weeks.

Of course, we are speaking of the time frame in which the body normally begins significant response or counter measure to a given stimulus. As example, HPTA inhibition became a concern at the 2 week point when using AAS that aromatize at a higher rate. This is the body's attempt at down-regulating the level of androgens/estrogens circulating in the vascular or lymphatic system. It does so by decreasing natural (endogenous) androgen (testosterone) production.

The main stimulus that acts as the "action" is estrogens produced by way of the activity of the aromatase enzyme upon testosterone. The "reaction" is the down-regulation of HPTA function that results in major post- cycle lean mass tissue loss (total in most cases) and "raisin nuts syndrome". By using the get in, hit hard, and get out structures of Max Androgen Phases, beasts like Frank retained a far greater level of their lean mass gains post-cycle. And their nuts played nice too. But what about longer AAS cycles?

AAS cycles that used testosterone always induced the greatest strength, weight, and lean mass gains for the least financial cost. But prolonged use of testosterone would have resulted in HPTA shut down and reduced (like serious) post-cycle lean mass retention. Since long term continuous progress was the goal of any muscular augmentation protocol, this situation was simply unacceptable.

### **WHY WAS TESTOSTERONE KING?**

Testosterone and its endless esters is equally anabolic and androgenic. This meant the hormone triggered a very high rate of muscle protein synthesis and growth while improving beast nitrogen retention resulting in an anti-catabolic effect. Additionally, the high androgenic quality of testosterone provided the increase in weight-load/work-load-capacity necessary to provide the stimulus for significant protein synthesis.

Action/Reaction, remember? For this reason, testosterone, like all other AAS, tipped the scale of the anabolic/catabolic ratio in favor of anabolism or growth. The high androgenic environment created by elevated testosterone levels also aided in increased glycogen synthesis and storage in muscle tissue, and improved recovery.

When speaking of growth potential environments this was far superior to almost any single AAS. But testosterone aromatizes to estrogens and reduces to DHT (Dihydrotestosterone), which is bad, right?

What many would-have-been-beasts failed to understand was that it was the "ratio" of testosterone-to-estrogen/DHT that triggered negative side effects, and the length or period of time elevation lasted that mattered. Not merely the existence of estrogen or DHT.

To begin with, women would not have great racks without estrogen, but let's look at the benefits the beasts realized from a bodybuilders prospective.

### **ESTROGENIC BENEFITS REALIZED**

- Triggers GH release by promoting the release of GHRH in the hypothalamus.
- Increased androgen receptor-site concentration and sensitivity.
- Increased muscle glycogen synthesis due to activation of the enzyme Glucose-6-Phosphate Dehydrogenase and the now famous GLUT-4 receptor.
- Elevation of total body water content table resulting in an anabolic osmotic effect, increased muscle fiber tension, and joint stability for increased work load-capacity.

*"These benefits attributed to estrogen were the exact goals of a muscle mass gaining protocol. Estrogenic negative side effects occurred mostly due to inadequate zinc absorption or from a hormone profile that allowed estrogenic activity to mitigate androgen activity. The cause of gynecomastia, as the reader knows by now, is estrogen and prolactin. More specifically this is mostly due to a non-favorable ratio between the two estrogens 2 $\alpha$ -hydroxyestrone and 16 $\alpha$ -hydroxyestrone. Just as different androgens (such as DHT) can possess site-specific triggering effects upon sex-specific-tissues, so can different estrogens. You should note, Frank, that in most cases it is prolonged exposure to elevations of certain hormones that allows triggering of most of the negative side effects an athlete can experience. Remember our 2 week Action/Reaction time frame?"*  
Coach

(Think about these points of interest as you read on ...)

## DHT (dihydrotestosterone)

DHT elevation from AAS use has been blamed for everything from balding to prostate cancer. Just to throw a wrench in the media and AMA dogma machine... consider this: A so-called "normal" blood testosterone level/reference range for a male is 300-1000ng/dl, and 14-76ng/dl for "normal" females. DHT is a metabolite of testosterone due to reduction by the 5 $\alpha$ -reductase enzyme.

Now, look at the above male and female reference ranges and answer a few questions:

1. Why are 40% of the world's **females** facing noticeable thinning hair by age 40?
2. Why has DHT been approved and patented as a treatment for BPT and prostate enlargement?
3. If male testosterone levels decrease as they age, and DHT is a product of testosterone, then why does the chance of experiencing BPT, prostate cancer, and balding increase with age? (Females do not have prostate glands)

There is a great deal of evidence to support the belief that estrogenic activity in the presence of DHT and an environment of low testosterone production may be the main cause of BPT and attributory toward prostate cancer. However, too much of anything can eventually be the cause or mediator of a negative reaction.

Note: Beasts used DHT and its derivatives to increase vascularity and hardness.

## TIDE CYCLES

Athletes who experienced negative side effects from AAS use did so due to either ignorance or lack of financial means. HPTA dysfunction, gynecomastia, and kidney / liver stress are examples of avoidable situations if an athlete had no pre-existing condition(s).

As we have discussed prior, estrogen did provide several benefits favorable to muscular growth when the period of elevated circulatory levels and tissue exposure was not prolonged or allowed ratio dominance over androgens. For most beasts, this was simply a matter of adding an anti-estrogen/aromatase drug to their cycle or the proper utilization of a Cortisol/Estrogen Suppression Phase.

But some either could not afford or find legal means to acquire the drugs. For the prior situations many beasts used AAS protocols that simply did not require estrogen control.

### **A Tide Cycle employed the same basic principles as a Max Androgen Phase:**

1. A maximum androgenic environment to stimulate rapid weight and strength gains for a brief period.
2. A transitional period between high androgenic and moderate-low androgenic with a maintained high anabolic atmosphere.
3. A period of maximum anabolic environment and moderate-low androgenic atmosphere to solidify to newly acquired muscle mass into high quality lean mass tissue.

The result was improved estrogen cleaning prior to cycle-exit. This allowed for minimum HPTA suppression and maximum post-cycle lean mass tissue retention. For beasts who were attempting to quickly re-establish near hormonal homeostasis ASAP post-cycle, Tide Cycles were a simplified approach.

It should be noted that liver and kidney stress was seldom an issue with non-17-alfa-alkylated AAS. This too was considered as a factor of post-cycle homeostasis.

**During Frank's creation many stages or dosage thresholds were employed to assure that no growth threshold was lost. The following examples were those utilized for his various Tide-Cycles.**

**Example #1 (Novice / Intermediate)**

Day #1,15,28,43, Testosterone Cypionate 400 mg (each day) Day #8,22,36,50 Deca Durabolin 400 mg (each day)

**Example #1 (Advanced)**

Day # 1,2,15,16,29,30,43,44 Testosterone Cypionate 400 mg (each day) Day #8,9,22,23,36,37,50,51 Deca Durabolin 400 mg (each day)

Both Testosterone Cypionate and Deca Durabolin had an active-life of about 16 days. This of course meant each drug had a half-life of about 8 days, so peak activity occurred at about 8 days after each administration before gradually declining over the following 8 days.

The result was a tide-like effect where each drugs qualities ebbed and flowed thus allowing the benefits of estrogenic activity without accumulation. The 16 day active-life of testosterone cypionate was well within our 2 week Action/Reaction time frame. This example terminated high activity about 8 days after the final administration of Deca Durabolin.

Nandrolone, such as Deca Durabolin, converted to estrogens or DHT at about 1/5<sup>th</sup> the rate of testosterone. The resulting hormones were nor-estrogens and Nor-DHT's with far less estrogenic activity or potency than their non-nor cousins. For this reason, Nors were noted as slightly anti-estrogenic /anti-DHT.

**Example #2 (Novice /Intermediate)**

Day#1, 8, 15, 22, 29, 36 Testosterone Enanthate 400 mg (each day) Day #4, 5,11, 12, 18, 19, 25, 26, 32, 33, 39, 40 Equipoise 200 mg (each day)

Both Testosterone Enanthate and Equipoise had about an 8-day active-life. So the ebb and flow effect between high androgenic and high anabolic environments in this example occurred more quickly than example #1. For this reason a few highly estrogen sensitive beasts, such as most endomorphs, experienced some of the possible estrogenic negative side effects after 6 weeks.

This was mostly due to Equipoise aromatizing at about half the rate of testosterone rather than 1/5<sup>th</sup> the rate as Nandrolone does. Additionally, the mild anti-estrogenic effect realized with nandrolone did not occur with Equipoise administration.

### Example #3 (Novice /Intermediate)

Day #1-2	Dianabol (oral)	40 mg/d	Deca Durabolin 400 mg (Day #1)
Day #3-4	Dianabol	30 mg/d	
Day #5-6	Dianabol	20 mg/d	
Day #11-12	Dianabol	20 mg/d	
Day #13-14	Dianabol	30 mg/d	
Day #15-16	Dianabol	40 mg/d	Deca Durabolin 400 mg (Day #15)
Day #17-18	Dianabol	30 mg/d	
Day #19-20	Dianabol	20 mg/d	
Day #25-26	Dianabol	20 mg/d	
Day #27-28	Dianabol	30 mg/d	
Day #29-30	Dianabol	40 mg/d	Deca Durabolin 400 mg (Day #29)
Day #31-32	Dianabol	30 mg/d	
Day #33-34	Dianabol	20 mg/d	
Day #39-40	Dianabol	20 mg/d	
Day #41-42	Dianabol	30 mg/d	
Day #43-44	Dianabol	40 mg/d	Deca Durabolin 400 mg (Day #43)
Day #45-46	Dianabol	30 mg/d	
Day #47-48	Dianabol	20 mg/d	

The D-Bol / Deca stack has probably been the most commonly used cycle in the world of chemical enhancement. The reason is simple; it worked and it was fairly economical.

The actions of c17-alfa-alkylated steroids and liver deactivation lead to a notable increase in hepatic IGF-1 release, which explains one mechanism of D-Bol's multiple anabolic pathways. Sadly enough there was the severe HPTA suppression and aromatization products realized with the normal use of D-Bol.

In a continuous time frame manner at a single common daily dosage, Dianabol severely suppressed the HPTA causing metabolic/anabolic lag post-cycle. Since the aromatization product of the drug was a sort of **super-girl** estrogen called 17-methylestradiol, gyno was common as was an environment of high estrogenic activity both during and post-cycle. Deca Durabolin, like all nandrolone, had been employed to mediate these negative side effects to some degree. But obviously this was not effective post-cycle!

By structuring the D-Bol/Deca stack into a Tide Cycle, some beasts had reaped the benefits of this combined synergy effect while greatly limiting the negative. Liver enzyme value changes from c17-alkylated oral administration was also greatly reduced. Oddly enough, the values were usually comparable to a few weeks of daily Tylenol or some deviations of oral birth control use.



Advanced beasts had altered this example to fit their level by adding 400 mg of Deca Durabolin on days #2, 16, 30, 44 and adding 10-20 mg to each listed daily Dianabol dosage. Simple, but it was quite effective.

Also, it was paramount to health and long-term progress that beasts acquired regular blood work and monitored any negative progressive changes through qualified professionals.

## **AAS - CORTISOL/SHBG KILLER PHASE CYCLE**

The era of long continuous AAS cycles was finally coming to its end once we were well entrenched in the new millennium and more productive protocols. Athletes of all types and levels had concluded that "net gain" was for more goal progressive than "gross gain" and subsequent "gross loss".

The idea of a few 12-16 week cycles of continuous AAS use yearly was not only health threatening, it simply resulted in futile attempts at maintaining any respectable amount of mass or quality post-cycle. Lets face it: Gaining 20-40 pounds of lean tissue mass total per year only to lose it and gain 5-10 pounds of fat was not progress. At best, it was unhealthy maintenance.

The most progressive AAS protocols were those that either limited HPTA inhibition, or those that actually resulted in HPTA function up-regulation. We have discussed several examples including Max Androgen Phase, Tide-Cycles, and different types of Androgen Spike Cycles. Most functioned upon a protocol of 4 weeks on (or less) and 1 -3 weeks off with proper manipulation of specific drug profiles to reduce or totally avoid HPTA suppression.

No doubt that most realize that post-cycle lean mass retention is highly dependent upon normal or above HPTA activity and the resulting endogenous androgen production. For the most part, we knew that estrogen level elevation from androgen aromatization and metabolism was the cause of HPTA suppression. High estrogen levels post-cycle would have resulted in a very sluggish re-bounce of natural androgen production.

Most protocols discussed so far dealt with the Action/Reaction problems by nature of structure quite well. But what about other factors that robbed my boy Frank of AAS gains?

We know that cortisol and SHBG levels elevated during AAS cycles due to the body attempting to regain control of the PTOR (Protein Turn Over Rate) and re-introduce homeostasis or the balance between gain/loss. As post-cycle circulatory androgen levels from AAS use decreased, the elevated cortisol levels attempted to gain dominance and induce a very catabolic environment.

Since SHBG binds to and deactivates existing circulatory androgens, the elevation of this binding protein results in even less free or active androgens and therefore in Frank's case attempted to aid cortisol in robbing him of AAS induced gains. If that was not bad enough, having enough estrogen to grow boobs with a low active androgen level would have resulted in serious fat gain and lean tissue loss.

Unfortunately, SHBG has a greater affinity for testosterone than estrogens for the most part. As with most problems, the situation was resolved and used as an advantage once the cause and effects (action/reaction) had been evaluated. Piece of cake!

*"Remember, only unbound or free androgens (AAS) are able to bind with and activate muscle cell androgen receptors. The higher the level of unbound AAS, the greater the activity. Growth is eminent."*  
Coach

Structuring a protocol with the intent of gaining significant mass was not real difficult. When structuring a protocol that allowed the greatest progress however, demands of a plan to enable the beast to retain or even add to the progress made had to be met with an answer.

Progress without a plan is merely accidental and usually short lasting. Dealing with the problems caused by excessive cortisol, estrogens, and SHBG was not difficult. The most economical and effective method was an "AAS - Cortisol / SHBG Killer Phase Cycle." A "Phase-Cycle" was a series of phases strung together to create a cycle with a **series** of Action/Reaction Factors accounted and planned for to create a synergistic effect.

### **The Goal of this Phase Cycle Should Be Obvious:**

- Build significant mass quickly
- Correct, regenerate, and/or protect HPTA function
- Solidify mass gains into quality retainable tissue
- Retain quality tissue gained, destroy excess cortisol and SHBG levels while clearing estrogen levels.

Pretty much the common goals we required for all muscle protocols... but with a plan.

PIECE OF CAKE!

## AAS - CORTISOL/SHBG KILLER PHASE CYCLE

### Phase 1 Example - Chart

DAY	DRUGS	DAY	DRUGS
1.	Test. E / D-Bol 60 mg	12.	Test. E. / D-Bol 30 mg
2.	Test. E / D-Bol 60 mg	13.	Test. E. / D-Bol 30 mg
3.	Test. E. / D-Bol 50 mg	14.	Test. E. / D-Bol 30 mg
4.	Test. E. / D-Bol 50 mg	15.	D-Bol 30mg/HCG 500 iu/Prov. 50mg 2xd
5.	Test. E. / D-Bol 40 mg	16.	D-Bol 40 mg/HCG 500iu/Prov. 50mg 2xd
6.	Test. E. / D-Bol 40 mg	17.	D-Bol 40 mg/HCG 500iu/Prov. 50mg 2xd
7.	Test. E. / D-Bol 30 mg	18.	D-Bol 50 mg/HCG 500 iu/Prov. 50mg 2xd
8.	Test. E. / D-Bol 30 mg	19.	D-Bol 50 mg/HCG 500 iu/Prov. 50mg 2xd
9.	Test. E. / D-Bol 30 mg	20.	D-Bol 60 mg/HCG 500 iu/Prov. 50 mg 2xd
10.	Test. E. / D-Bol 30 mg	21.	D-Bol 60 mg/HCG 500 iu/Prov. 50 mg 2xd
11.	Test. E. / D-Bol 30 mg		

- The example dosage for Testosterone Enanthate was 100 mg/d for intermediate level and 150 mg/d for advanced level athletes on listed days.
- Dianabol was divided into 2-3 even daily dosages
- Proviron was divided into 2 daily dosages. (200 mg/d MAX)

### Drug Synergy

The descending/ascending dosage schedule for Dianabol initiates and maintained a more constant blood plasma androgen level as Testosterone Enanthate built up and peaked at day #8 then slowly decreased between day #15 to 22. This allowed a mostly clear system by day #22.

HCG established a superior endogenous androgen production level before cycle exit. Proviron was a respectable androgenic, and an aromatase inhibitor which maintained muscle hardness and some strength benefits while aiding estrogen clearing. However, the benefit of Proviron in this protocol was its very high binding affinity for SHBG. This approach made all other androgens more bio-active since less SHBG was available for binding.

Some experts believe that fast acting testosterone, such as suspension or propionate, suppresses HPTA function more quickly than slower acting testosterone such as enanthate or cypionate. This is true for a very simple reason.

Faster acting testosterone flood the system with the active drug much more quickly; whereas slower drugs require a protracted time frame to build-up to maximum circulatory levels. The result is that fast acting drugs mean faster

aromatization and lever metabolism to estrogens. Since estrogens are the greatest (or worst) cause of HPTA suppression and the resulting negative feed-back loop, it should be evident why this is so.

The injectable long acting anti-aromatase drug formestane (21-28 day active life) was noted as mildly anabolic as was the injection administered form of Teslac. Both drugs worked well to prevent excessive estrogen build-up when taken a few days before beginning administration of aromatizing androgens.

**The Phase 1 example** on the prior page resulted in serious androgenic / anabolic activity augmented by the synergistic effects of elevated IGF-1 production from the liver during oral alkylated AAS deactivation (Dianabol). Most Testosterones have been utilized or substituted for enanthate when the administration protocol was adjusted to facilitate system clearing by day #22.

After all, off meant off, or it was not a Phase Cycle. When Sustanon-250 was the substitution drug it had to be administered in its total dosage entirety within the first two days to clear the system adequately by day #22... by the way. That was 1000-1500 mg of mixed testosterone esters per day for the first 2 days. Interesting. Unfortunately the over abundance of the propionate ester in Sustanon-250 had decreasing value here.

## **AAS - CORTISOL/SHBG KILLER PHASE CYCLE** **Phase 2 Example - Chart** (continued)

DAY	DRUGS
22-28	HCG 500iu/Proviron 50mg 3xd/Humalog 4-6iu 2xd

- Proviron was divided into 3 daily dosages (200 mg/d max)
- Humalog was administered first thing in the morning and immediately after training (on training days).
- Humalog was very fast acting and required the ingestion of carbohydrates within 15 minutes after administration. (See "***The key to freak status is sensitivity***" for supplemental /dietary insights and action/ reaction factors of insulin)

## **Drug Synergy**

HCG provided above normal endogenous androgen production while Proviron augmented total androgen levels and cleared the system of estrogens. This improved HPTA function significantly. Insulin was a powerful cortisol inhibitor and was utilized at the two highest cortisol activity/secretion periods of the day to act both anti-catabolically and as an anabolism promoter.

The goal was to maintain an anabolic / protein synthesis environment and signal between AAS phases thus preventing tissue loss of a quality nature. Insulin also inhibited SHBG synthesis in the liver resulting in significant free/active androgen levels.

By utilizing the drug synergy of this Phase 2 example, beasts were able to realize much greater benefits and fewer inhibiting factors during Phase 3. This was due to the beast's insight to pre-load the system for minimum estrogenic activity and SHBG activity.

This resulted in a much higher active-free-androgen/anabolic level realized from AAS administration. This meant more results from lower dosages and fewer negative side effects. A lower Cortisol/Catabolic level at the Phase 3 entry point meant less anabolic /catabolic competition and a lower Phase 3 exit level for cortisol. This ensured superior post-cycle lean mass retention in itself.

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***Editor's Note:** Many have successfully used the OTC products **Testosterone OH** by HMGear ([www.HMgear.com](http://www.HMgear.com)) as a means of increasing anabolism while supporting and increasing HPTA function during this period.*

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## **AAS - CORTISOL/SHBG KILLER PHASE CYCLE**

### **Phase 3 Example - Chart** (continued)

DAY	DRUGS
29-42	Winstrol D. 50 mg + Finabolan 30 mg

- This example cycle/phase was highly effective for this purpose at 50 % of listed dosages for novice users.
- Finabolan is a black market drug containing Trenbolone Acetate.
- 50-100mg/d Proviron was sometimes layered to increase AAS activity/potency.

### **Drug Synergy**

Winstrol Depot was a very high anabolic drug that did not suffer aromatization. A high rate of protein synthesis was paramount at this point in the protocol (Phase Cycle) if mass weight gains from Phase #1 were to be solidified into quality lean tissue. Finabolan, like all Trenbolones, was a very high androgenic drug that was non-aromatizing. Drugs of this nature significantly increased glycogen synthesis in muscle tissue resulting in improved training recovery and adequate fuel reserves to hinder catabolism and improve protein synthesis rates. Trenbolones also tended to fit into cortisol receptors and misshape them much like Teslac does with estrogenic type receptors. The result was far less cortisol activity post-cycle and superior lean mass retention. Trenbolones were notably highly resistant to SHBG binding.

The goal during Phase #3 was to induce another high growth environment focused primarily on additional quality tissue and solidification of mass gains realized during Phase #1. At this point, the system was perfect for high anabolics such as Winstrol or Primobolan Depots and high androgens such as Masteron, Trenbolones, or Halotestin. None of these drugs were an aromatization concern and all worked well in a low cortisol/estrogen/SHBG environment. It was important to adjust administration time sequences to assure a mostly clear system by day #44 of this Phase Cycle.

The next phase had to assure an anabolic environment clear of AAS and substances that inhibited HPTA function. Off meant off.... So no Equipoise, Deca, Primobolan.

## AAS - CORTISOL/SHBG KILLER PHASE CYCLE

### Phase 4 Example - Chart (continued)

DAY	DRUGS		
43-46	Clomid	100mg/Humalog/Proviron	150 mg
47-56	Clomid	50mg/Humalog/Proviron	150 mg

- Clomid was divided into 2 daily dosages on day #1 -5.
- Humalog insulin was administered at a dosage of 4-6 iu only directly after training by beasts prone to easy fat accumulation.
- HCG 500 iu daily was sometimes substituted for Clomid with a lower overall drug synergy effect but it did up-regulate endogenous testosterone production.

### DRUG SYNERGY

Clomid was used in place of HCG to regenerate the entire HPTA rather than only testes function. Additionally, Clomid acted as an estrogen-site (receptor) antagonist while Proviron inhibited estrogen formation and allowed systematic estrogen clearing. As in Phase 2, Insulin /Proviron inhibited SHBG synthesis and binding resulting in a greater free testosterone level realized from elevated HPTA function while maintaining an anabolic environment and inhibiting cortisol.

This entire strategy allowed Frank to exit the completed protocol with above normal natural androgen levels and below normal estrogen, SHBG, and cortisol levels.

Some beasts extend the 4<sup>th</sup> phase an additional week. It was important to realize that the use of insulin in this protocol/Phase Cycle was intended to act as a means of reducing cortisol and SHBG levels. Athletes got fat when erroneously "over dosing" for this specific purpose.

When this phase was extended the additional week, low dosages of GH (1 iu 2-4 times daily) was often beneficial to add additional anabolism and to hinder fat accumulating from prolonged periods of exogenous insulin use. Zinc intake had to be augmented at this point in the protocol.... Well, you read "Zinc: The Anabolic Activator", Right? Injectable ATP, creatine products, D-ribose, and L-Arginine were quite effective in a synergistic structure during Phase #4.



## ATP: THE BASIS OF GROWTH

With all of the chemical muscle enhancement protocols, we sometimes forget the foundation or fundamental factors that allowed them to actuate a response. We often discuss Action/Reaction sequences as a means of creating a chosen outcome to a given stimulus. So a bit of bio-chemistry should not bring the reading process to a halt simply because it is basic in appearance or a little technical by necessity. In short, don't be a sissy just because you do not like the occasional science-geek type of information. With greater understanding comes greater growth.

### WHAT IS ATP?

ATP stands for Adenosine Tri-phosphate. (Remember that "tri" stands for three (3) phosphate molecules in the ATP structure.) It is the only energy or fuel supply the muscles can use. Without ATP, we would be unable to train, grow, eat, or have sex. In fact, the amount of and the ability to regenerate ATP dictates how well we do at any of these activities. Certain amino acids (from protein), carbohydrates, and fats can only be utilized as muscle fuel after they have been metabolically converted into substrates that are used to synthesize ATP. So ATP is life and **"the"** energy source for all cellular processes.

There are two energy systems used by the body. The anaerobic system supplies energy/ATP in the absence of oxygen. Weight lifting, sprinting, and other high intensity all out activities are anaerobic by nature. The other energy system is the aerobic system. It is used for low- moderate intensity activities such as walking, reading, and watching the hard-body on the Stairmaster. The aerobic energy system produces ATP too, but does so through the use of oxidation and macronutrients. Of course this also means that the aerobic energy system is dependent upon blood flow for oxygen.

### ANAEROBIC ENERGY PATHWAYS

Each muscle contains enough ATP to fuel the first few reps of a work-set. After stored ATP is spent, the working muscle fibers must produce more ATP in order to continue the set. After the first few seconds, the "CP/ATP- Pathway" kicks in and supplies fresh ATP for about 20-30 seconds. "CP" stands for "Creatine Phosphate" (sometime referred to as phosphocreatine and the ATP-PC system). After existing working muscle stores of CP are spent, the next anaerobic energy pathway kicks in and can fuel ATP production for an additional 60-150 seconds. This is the "Glycogen / ATP-Pathway". Of course 3 minute work-sets are unlikely.

**WARNING:** Serious Science Geek Info Follows. Personally I think the entire CP production process and "CP/ATP-Path way" mechanism is amazing. I also do believe understanding both are major mandatory factors in both growth and fat loss protocols.

How does that creatine stuff get created? Most athletes who live on this planet have heard of creatine monohydrate supplement products. Some do not realize that the body produces creatine (CP) endogenously everyday. Where did you think CP came from?

Natural or endogenous creatine is produced from amino acids. The two dominant amino acids in the process are Arginine and Glycine. Basically these two amino acids under the influence of "Glycine -amidino-transferase" form guanido-acetic-acid (GAA). GAA is then methylated in a reaction induced by "guanido -acetate-methyltransferase" forming "methylgluanido-acetic-acid, "or better know as Creatine. (Ya, both of the science geek readers enjoyed that one.)

Creatine is manufactured in the liver, and various other tissues, and then transported by the vascular system into target tissue such as muscle. The storage form is called CP or Creatine Phosphate. Once stored in target tissues, CP is specific to that tissue. As example, CP stored in a tricep cannot be used to fuel the "CP/ATP-Pathway" in other muscles such as a bicep.

A note of interest is that supplemental creatine is made from sarcosine and cyanamide usually. When an athlete utilizes supplemental creatine, the enzyme "Glycine-amidino-transferase" is inhibited and the body stops producing endogenous creatine. Within 7-14 days of supplemental creatine discontinuance, production returns to normal, if extracellular stores dwindle.

The interesting point to make here is that the oral AAS methyltestosterone increases the production of both guanidino-acetate and creatine. This is due to methyltest aiding in methylation which turns guanidino-acetate into creatine and it supports the production of rate limiting enzymes. The result was significantly greater cellular CP/ATP stores when methyltest and supplemental creatine are cycled together. As you will see later, adding insulin to this protocol had great potential applications. But we have more science geek stuff to learn first. **(QUIT WHINING)** So bare with me, please.

How does the CP/ATP-Path way Function? Now that we know how that creatine stuff CP is made, we now need to get a brief understanding of how the CP/ATP - Pathway functions.

Remember adenosine "tri" phosphate (ATP)? When cellular ATP is utilized, it gives up one of its high-energy phosphate molecules to fuel activities such as muscular contractions. Since ATP lost one (1) phosphate molecule it becomes "ADP" which is Adenosine Di- Phosphate". Note the "DI" refers to there being only two (2) remaining phosphate molecules in its structure.

Since ADP can not be used as an energy source, the cells turn to the "CP/ATP-Pathway". CP donates its phosphate molecule to ADP to create fresh ATP. (CP + ADP =ATP). This process occurs at nearly the rate of ATP expenditure during a set. Obviously each muscle contains enough CP to supply the "CP/ATP-Pathway" for about 30 seconds before the next anaerobic energy pathway must kick in.

\* It should be noted that the CP/ATP-Pathway is also referred to as the "Phosphagen (energy) system".

## **The Glycogen/ATP Pathway**

As a work-set continues beyond the "CP/ATP- pathway" capacity and cellular CP runs out, the working muscle must turn to the "Glycogen /ATP - Pathway" for energy. Glycogen is the storage form of glucose derived mostly from carbohydrates. Each working muscle contains enough glycogen to further fuel ATP production and subsequent muscular contractions for an additional 1.5-2.5 minutes. But of course, 3 minute work-sets at anaerobic levels are unlikely.

When cells use glycogen to produce ATP during a set, the by-product is predominantly lactic acid. Since the working muscle is contracting intensely, blood flow is restricted which traps lactic acid build-up in, and keeps additional glucose/glycogen out. This continues until the set is forced to terminate due to inadequate ATP/energy supply.

With the exception of weenie type athletes, the burning effect of lactic acid build-up is not the cause of muscular failure at this point. It is actually because the "Glycogen/ATP-Pathway" can only supply energy/ATP about 75-85 % as quickly as the "CP/ATP- Pathway". Energy requirements simply exceeded supply. However, if a work-set actually did continue for 2-3 minutes, lactic acid build-up would reach high enough concentrations to totally inhibit muscular contractions. (Burn Baby Burn)

\* It should be noted that the "Glycogen/ATP - Pathway" is also referred to as the "Lactic Acid (energy) System".

## **AEROBIC ENERGY PATHWAY**

As stated earlier, the aerobic-energy-system utilizes a different method to create energy/ATP called an "Oxidative Pathway". Those aware of the process of oxidative phosphorylation can skip ahead, but the explanation is brief anyway. The "Aerobic Energy Oxidative Pathway" simply uses macronutrients that have been converted into amino acids (protein), fatty acids (fat), and glucose/glycogen (carbohydrates) to manufacture ATP.

Since the pathway is used for low-moderate intensity activities, such as walking, blood flow is not restricted due to a lack of continuous muscular contraction. Therefore a significant build-up of lactic acid does not occur and the activity can go on for hours... in most cases. Because there is not significant lactic acid build-up in the working muscles, and due to continuous blood flow during this type of activity, ATP supply exceeds demand. Simple!

The method the body uses to select the proper energy system and pathway is simplistic in nature, actually. When you under take a given activity such as a bar squat at say 80% of your single rep maximum weight, the first energy pathway to kick in is the "Aerobic Energy Oxidative Pathway". This energy system is far better suited for hard-body watching and can supply energy at only 50-60 % of the rate of the "CP/ATP - Pathway".

So the body turns to the working muscle group for stores of ATP and the "CP/ATP-Pathway" for energy. Once local CP stores run out, the set reaches "positive rep phase" failure. However, if the work-load is immediately reduced 15-25%, and the set is continued as a drop-set, the body can (and will) kick in the "Glycogen /ATP-Pathway" which allows the set to continue at a reduced energy/ATP requirement level.

Why? Because the "Glycogen/ATP -Pathway" can only produce energy/ATP at about 75-85 % of the rate of the "CP/ATP -Pathway". Of course if the drop-set was super-setted with hard-body watching the "Aerobic Energy Oxidative Pathway" would kick in again at the watching point. Got it?

## **ATP/CP REGENERATION**

After a work-set is terminated and blood flow is returned to the target muscle, spent ATP and CP must be replaced. For each 30 seconds of rest, 50% of the remaining spent ATP and CP, is replaced. This means that it takes 30 seconds to replace 50%, 60 seconds to replace 75%, 90 seconds to replace 87%, and 120 seconds to replace 94% of the ATP/CP used during a work-set or anaerobic activity. For these percentages to apply, a total rest period for the target muscle must be utilized. This is because even low-moderate intensity activity requires ATP and will slow the process of ATP/CP replacement. Unfortunately, lactic acid build up can hinder ATP formation, too.

## LACTIC ACID CLEARING

Excessive lactic acid build-up can cripple ATP/CP regeneration. For each 15 minutes of time that passes, that does not employ the "Glycogen /ATP -Pathway, the target muscle can remove 50% of the lactic acid build-up that accumulated during a work-set. This means that it takes 15 minutes to clear 50%, 30 minutes to clear 75%, 45 minutes to clear 87%, and an hour to clear 94% of the lactic acid accumulated. Lactic acid is a major player in the anabolic / catabolic ratio relating to muscle growth and loss.

**A side note of interest.** Aerobic activities twice weekly for 20-60 minutes each will increase the body's ability to clear lactic acid build-up. This results in more rapid ATP/CP regeneration, and quicker recovery. (This means growth of course)

### DID YOU KNOW.....

Most bodybuilders are not aware of the fact that the ATP and CP levels in cells dictate how effective any chemical muscle enhancement protocol is. Since all cellular processes are energy / ATP dependent, this really should not be a big surprise though. All anabolic chemistry, endogenous or exogenous, requires receptor-site activity to actuate a response. And that too, requires adequate ATP levels intracellularly. An example is what happens when AAS meet target muscle cells after an intense work out.

## ATP AND ANABOLIC/ANDROGENIC STEROIDS (AAS)

Every muscle cell has about 50 different types of receptor-sites. Most receptor-sites can only be activated by their specific chemical counter part. This is like a key that fits and actuates only one type of lock. The receptor-site specific to testosterone and other AAS (Anabolic/Androgenic Steroids) is called a muscle cell androgen receptor-site.

When an AAS molecule merges with an androgen receptor-site, interesting things happen...or not. The androgen receptor / AAS molecule complex travels inside the cell and delivers a "make new proteins" (protein synthesis/ anabolism) message by way of DNA/mRNA interaction.

The result is repair of damaged proteins and subsequent growth due to over compensation if the number of anabolic messages exceeds the number of catabolic (tissue wasting) messages occurring. In short, hypertrophy. Cool huh?

Low cellular ATP/CP levels seriously screw up the whole program. When cells contain adequate, or about, amounts of ATP, androgen receptors are continuously moving in and out of them. They must be outside of the cell to merge with AAS molecules and then be capable of moving inside of the cell to trigger the

whole "Make new proteins" sequence. This is great until cellular ATP/CP reserves suck. The entry of androgen receptors into cells is ATP/energy dependent, but the exit is not. This means low ATP levels trap androgen receptors outside of the cell nucleus. When this happens, AAS molecules merge with androgen receptors and no signal or message is transmitted regardless of the level of androgens circulating in the body.

So when is cellular ATP/CP low? A single set of curls reduces the level of bicep muscle cell ATP/CP significantly as you may realize since positive failure occurs. Imagine the reduction that results from all of the sets that follow!

Another reality also reduces cellular ATP/CP. When muscle fibers are damaged from training, inflammation results. The inflammation is due to an increase in water being pumped into the muscle fibers (we call this swelling). The damage triggers the pumps located on the surface of each cell which then works feverishly to pump out the incoming water. Their goal is to rebalance the intracellular and extracellular (inside and outside) water tables by forcing out salt.

Unfortunately, the extracellular water rushes into damaged cells at a high rate forcing the pumps to work very hard. This would appear to be a good thing, since increased cellular volume triggers osmotic induced anabolism (growth caused by an increase in total intracellular nutrients and cell stretching). But it's not.

As a result of these cellular pumps requiring ATP/energy to actuate, energy stores are further depleted. So the water pumping process further decreases cellular ATP/CP stores and inhibits receptor activity even more. To make things worse, damage causes the intracellular release of calcium and other factors which destroy androgen receptors thus reducing the possible number of AAS molecule / androgen receptor-site mergence or paring.

Diet has a profound effect upon cellular ATP/CP stores and formation. When an athlete decreases available energy /ATP substrates (Nutrients from which ATP/energy can be manufactured by bodily processes), cellular loss of CP occurs and the entry of any creatine source is inhibited. This is simply due to a decrease in cellular energy /ATP available for cellular processes as a result of reduced calorie /substrate intake.

In short, you feel lazy and so do your cells. This is a major reason why AAS provided very poor muscle building qualities during calorie restricted periods. It was very important to use a carbohydrate "spike" day every 3<sup>rd</sup> or 4<sup>th</sup> day during diet phases and AAS use. This simply meant eating more carbs on a "spike" day to refill glycogen stores so creatine and androgens in the blood stream can enter muscle cells.

All of these ATP/CP wasting situations really decrease possible muscular growth. And it can take days to fully restore cellular glycogen, CP, and ATP after an intense training session. Oh, but it gets worse....

## **ATP SHRINKAGE AND BCAA'S**

During a work-set and the subsequent employment of the Glycogen/ATP - Pathway, a build-up of lactic acid occurs. Lactic Acid is a by-product of glycogen use as an energy/ATP source. It is made up of lactate and hydrogen, the latter being an acid. The build-up triggers acidosis in the working muscles cells which in turn activates a very muscle unfriendly enzyme called "Branch-Chain-Alpha-Keto-Acid-Dehydrogenase", (I really don't make these names up) or BCAA'S for short (Okay?).

BCAA'S are evil stuff that degrades Branch Chain Amino Acids (BCAA'S) in the working muscles. The BCAA's are leucine, Valine, and Isoleucine. These are essential amino acids, meaning that the body can not manufacture them from other amino acids, and they make up about 33% of total muscle proteins.

So lactic acid build-up results in acidosis, this in turn activates evil BCAA'S. BCAA'S degrades BCAA'S which means muscle protein catabolism or wasting. Catabolism means the dreaded state of shrinkage.(huh?)

When training or diet factors induce catabolism by way of activation of the BCAA'S enzyme, the muscle digests itself to yield BCAA's and glutamine for use as a source of substrate for energy /ATP production. To do this, the ammonia group is removed from the BCAA's structures to allow their carbon group to be used as a fuel or substrate source.

The remaining ammonia is obviously toxic to tissues, so the glutamine resulting from the muscle catabolism also acts like an ammonia conveyor. Meaning that the glutamine helps carry the toxic ammonia through the circulatory system to the liver and kidneys for excretion. It should be noted that glutamine can be converted to glucose, as can BCAA's, by the liver. So when an athlete smells ammonia in their sweat during or after training, this is why. The result is shrinkage... which of course all males know about and all females should.

## **IRS AT THE CELLULAR LEVEL**

There appears to be some shrinkage cosmic joke about genetics and size potential. Our bodies and muscles have a protein co-factor called "Ubiquitin" that binds to, and marks for destruction, muscle proteins. This ubiquitin marker is like the appraiser for an IRS-like protease enzyme called "Proteasome". Proteasome digests the ubiquitin marked proteins into simple amino acids.

The sole purpose of ubiquitin and proteasome seems to be to prevent excessive muscle mass gains. The result being significant attempts at placing more limitations upon those who work very hard toward progress. (Told you, just like the IRS). Since this activity "can" result in eventual energy production or possible repair, we could call it the "Ubiquitin-Proteasome-Pathway".

But it certainly is not effective or beneficial to those doing the work. Did I mention the IRS? The moral to this story is that when dealing with either the IRS or the "Ubiquitin-Proteasome-Pathway", every effort must be made to play nice.

The "Ubiquitin-Proteasome-Pathway" is the most catabolic chemical mechanism the body has. The cosmic joke is that we train hard to trigger an adaptive response resulting in anabolism and muscular growth. But training increases acidosis and cortisol production emphasizing the activities of Ubiquitin-Proteasome even further.

The punch line to this bad joke is that the activity of the "Ubiquitin - Proteasome-Pathway is dependent upon ATP for energy also. But even when cellular ATP levels are very low, it works just fine.

The reason we strive to understand these cellular events is to learn how to use them to our advantage or negate their negative effects. It is all action/reaction. This is how ordinary individuals become extraordinary bodybuilders.

## START AT THE SOURCE

Almost anytime low cellular ATP regeneration is an issue, supplemental creatine use is the first line of defense. Did you know that a creatine monohydrate loading phase of only five grams, 4 times daily for five consecutive days results in an 11-14% increase in CP and a 9% increase in ATP intracellularly? (YUP) A 5-8 gram daily maintenance phase will usually maintain this increase. Big deal! An 11-14% increase in CP and 9% increase in ATP also means a corresponding increase in androgen receptor activity and decrease in catabolism. Of course those individuals who utilize AAS, Insulin, IGF-1, Clenbuterol, Ephedrine, thyroid hormones, and/ or other specific drugs realize far higher cellular CP/ATP level increases. Yes, all these drugs require ATP to trigger maximum responses also.

The best loading phase dosage is 0.3 g/kg daily when using creating monohydrate, and a daily maintenance dosage of 5-10 grams. So a 200 LB athlete would consume 30 grams of creatine monohydrate in 3-6 divided doses for 5-7 days, then use 5-10 grams daily there after. (Only high quality creatine please)

2 grams daily of creatine monohydrate will **not** maintain elevated intracellular CP/ATP levels. Several so-called experts (the kind whom think "if Johnny jumps off the barn, so will I") actually write that it will. Okay, to maintain normal CP stores the body naturally produces about 2000 mg/2g of free creatine daily but stops when



supplemental creatine is utilized. 1000 mg of creatine "monohydrate" only contains 850-880 mg of free creatine. Do the math, there would be a daily deficit at a supplemental intake of 2000mg/2g daily. Creatine may buffer lactic acid and slow acidosis, by the way. (Think about that as you read on)

## GLUTAMINE

The amino acid glutamine is the most abundant amino acid in our bodies muscle cells. In fact, the "average" (this "average" stuff obviously does not apply to you or I) individual must consume or manufacture 50-120 grams daily of this NON-(?)-essential amino acid.

As you will recall, the body uses glutamine to escort toxic ammonia out of tissues and the body. But that is only a small part of this cool amino acid's abilities. (Some others relate to our main issue) 25g glutamine ingested orally reduces cortisol 26.9 %, increases IGF-1 101.32%, and L.H. 15 % at the plasma level. Glutamine replenishes muscle and liver glycogen post training as effectively as carbohydrates while decreasing the need for Insulin to trigger muscle cell volumization without increasing fat stores.

This obviously means more nutrients are pulled into the cell as well. 0.2-0.6 g/kg daily of glutamine supplied supplementally will provide a positive nitrogen balance (growth environment). That is 20-30 grams of glutamine daily for a 220 lb (100 kg) athlete. Oh, and Glutamine decreases insulin resistance/insensitivity.

It seems that some feel that glutamine is a worthless supplement... and there is even some viable research that suggests that the GI track may not even absorb Glutamine. Interesting to say the least. First consider that even if this was so, the stomach lining is predominantly made up of glutamine and that most research agrees that ingested glutamine is often robbed by the stomach lining for regeneration.

If this is so, then it simply means that there is a significant reduction in full body glutamine requirement due to the fact that the amount absorbed by the stomach now does not need to come from somewhere else (like, uh, muscle?). So worst case scenario is that the ingested glutamine spurs glutamine stores elsewhere in the body.

My personal experience is that supplemental glutamine is likely absorbed rather well. I base this upon glucose monitoring before and after ingestion. If 20g of glutamine is ingested during a period where blood glucose is near 65 the spike within 20 minutes is quite significant. Do you think maybe that this is only magic? (Geez!)

## **BCAA'S (Branch Chain Amino Acids)**

Since BCAA's are essential amino acids it should be obvious that we need to consume adequate daily supplies. However, we also know that BCAA's make up about 33% of our muscle proteins. This strongly suggests that as muscle-mass orientated athletes we must strive to super- compensate, not merely replace what is lost.

Unlike other amino acids, BCAA's are processed in the liver and metabolized in the muscles. Other amino acids are metabolized in the liver. BCAA's and insulin work together to transport other amino acids into cells for repair and growth also. This synergy effect between insulin, BCAA's, and other amino acids requires that complete proteins be provided as the predominant BCAA source.

This assures no weak link will exist in the repair and growth process. BCAA powders and capsules are also an option. But these should be taken with food for the prior explained reason. Ingesting 6-8 grams with a protein/carb drink before a workout can decrease the catabolic effects of evil BCAA's.

When shopping for a supplemental BCAA source look for a ratio of 76% leucine, 12% Valine, and 12% iso-leucine. The higher level of leucine is necessary because during high intensity activities, leucine is depleted quickly. Its by-products are used to create another amino acid called Alanine which in turn is metabolized into glucose in the liver for energy needs... ATP. (Oh ya, that stuff)

**An interesting side bar** about this 76-12-12% ratio is that athletes experience significant loss of visceral fat when using it. Visceral fat is the fat located in deeper layers of the body under subcutaneous fat stores. (Wow.)

Women especially experience difficulty when attempting to lose fat in these areas. As to daily dosage, 1 gram per 20 lbs of body weight of the 76-12-12% ratio in supplemental form is effective.

## INSULIN IS A POWERFUL PROTEASOME INHIBITOR

Insulin's anti-catabolic effects are well known. In most cases it is assumed that this quality can be attributed to insulin's cortisol suppressing capabilities. This is true, but insulin's greatest anti-catabolic effect is inhibition of the proteasome-pathway. Oh ya, but insulin molecules experience difficulty when attempting to bind to their receptor-sites on cells that are experiencing low ATP levels. (That blows)

Training decreases circulating insulin levels while increasing the activity of the ubiquitin-proteasome-pathway and BCAAAD. Sipping a carbohydrate drink during training mediates this to some extent due to elevated insulin secretion.

Obviously a carbohydrate / BCAA drink with peptide glutamine and creatine monohydrate would be more effective. (Peptides tend to resist rapid break-down in solutions better than free form amino acids)

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***Editor's Note:** Unfortunately, peptide glutamine also turns into cement if you don't drink it quickly. And it's very hard to mix. So, if you want to be able to enjoy your carb drink through out your workout, bring some regular L-glutamine powder and BCAA powder to the gym, pour it into your carb drink and shake it up and sip on it through out the workout. Just make sure you give it a good shake before every sip since the BCAA tend to settle to the bottom.*

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Some chemically enhanced beasts administered 4-10 iu of a fast acting insulin, such as Humalog, immediately after a work-out and downed a drink providing the following:

- 2-4 g L-Arginine
- 50 g Whey Protein
- 25 g L-Glutamine (or) Peptide Glutamine
- 10 g Creatine Monohydrate
- 25-75g Dextrose
- 16 oz. Water

The dry ingredients were pre-mixed and water was added only immediately before consuming to prevent degradation of L-Glutamine and creatine.

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***Editor's Note:** For some or many, taking that much L-glutamine (25 grams) is a lot for the stomach to handle and most want to throw up. Mainly because there is a massive insulin surge with 10 or more grams of glutamine and it makes most people nauseous. Add this with the insulin injection and the other insulin-spiking ingredients and you may black-out! Hehe ...my suggestion is to test this out at half the dose of glutamine and see how you do the first time.*

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This significantly improved ATP/CP regeneration rates while inhibiting catabolism. This meant all other ATP dependant cellular activities returned to normal or above much more quickly. Supplements such as alpha lipoic acid, D-pinitol, and glucosamine may have increased insulin receptor sensitivity while drugs such as Glucophage or Avandia acted synergistically with insulin for this purpose.

Lactic Acid / Acidosis triggers GH release. This is, in part, the body's process of subsidiary energy pathway recruitment. GH triggers fatty acid release from lipocytes (fat cells) to be used as another energy /ATP source by cells. Cool huh?

Anyway, insulin hinders GH release when the body is at rest but not when the triggering pathway is due to acidosis. GH and Insulin must both be present in the liver for IGF-1 production. This is why GH release triggered by lactic acid build-up does not result in liver IGF-1 production. Too bad training also reduces blood insulin levels because as I said earlier, both are required in the liver for IGF-1 production/elevation.

IGF-1 can oppose proteasome and the good news is that all cells do contain some IGF-1 from prior training days. Lactic acid will destroy some of this IGF-1 and decrease normal IGF-1 receptor function/activity. But that is okay because this creates super IGF-1. (Huh?) We will get to that in a moment.

IGF-1 is very similar to insulin in structure and can stimulate both receptor types. 10-15 mcg of IGF-1 injected into the target muscle post-work out is a common hard-core bodybuilding protocol. This very effectively inhibits the catabolic activities of cortisol, BCAAs, and proteasome.

Since this protocol is followed 3-5 times daily through a series of injection-sites/target muscle a significant level of localized IGF-1 results. If IGF-1 is bound by its binding protein, the result is a twelve hour plus active-life.

### **SUPER IGF-1?**

Lactic acid /acidosis destroys some IGF-1, which is good. (Huh, again) There is always some IGF-1 in our circulatory system and muscle tissues from previous workouts and GH/insulin release. Most of this IGF-1 is bound to a protein called IGF-1 -BP-3 (Binding Protein 3) and is therefore inactive.

This means it sits in reserve waiting for something to bust it free so it can merge with its (or insulin receptors) receptor-sites. If the IGF-1 was not bound by IGF-1 -BP-3, it would be quickly destroyed by multiple metabolic factors. Lactic acid build-up is one of these factors that frees or unbinds IGF-1. Some of the free IGF-1 is destroyed but much of it is converted into a "super IGF-1" by lactic acid.

This new goodie is called "Des (1-3) IGF-1 and it is about 1000% more active than regular IGF-1! (WOW!) This newly formed "Des (1-3) IGF-1" can fit into and activate the lactic acid damaged IGF-1 receptor-sites and trigger major anabolism while inhibiting catabolism.

## **Hyper-Recovery**

When dealing with the catabolic environments of evil BCAA, the Ubiquitin-Proteasome-Pathway, and post-training inhibition of ATP formation by lactic acid, insulin holds a major key.

Whether of endogenous or exogenous source, an elevation in circulating insulin levels /activity not only significantly hindered the catabolic effects, but also promoted the return of anabolism to affected muscle tissue. The idea was the promotion of hyper-recovery resulting in hyper growth.

### **Hyper-Recovery Benefits of Insulin**

1. Signals muscle cell up-take of BCAA's while hindering BCAA (allowing for supra-compensation).
2. By triggering the storage rate-limiting enzyme responsible for glycogen replacement (Glycogen Synthase) Insulin increases cellular synthesis and storage of glycogen.
3. Significantly increases cellular up-take of creatine and other amino acids required for ATP synthesis, cellular repair, and growth.
4. Inhibits excessive cortisol release and activity.
5. Inhibits the glycogen-releasing hormone (Glucagon) to some extent. (Dose dependent)
6. Increases total cellular water and electrolyte content in muscle tissue.
7. Inhibits proteasome activity.
8. Acts synergistically with all other anabolic hormones and hormone-like substances to induce hyper-recovery.
9. Inhibits SHBG synthesis by the liver which then in turn results in an increase in AAS bio-activity.
  - Endogenous Insulin misuse can be deadly and administration should never be undertaken without qualified supervision.
  - AGR Nutrition is soon releasing an orally active insulin replacement.

## THE KEY TO FREAK STATUS WAS SENSITIVITY

### A Beast Has Got To Be Sensitive ...

Most beasts realized an increase in net lean tissue mass of 20 or more pounds during a "first time" insulin protocol. After the initial protocol, gains decreased with each subsequent cycle. In many cases athletes reached a point where fat anabolism exceeded lean tissue gains.

This did have some benefits, but health concerns exceeded benefits dramatically. (Unless an athlete's goal was to be one of those strange sweaty fat people in spandex on day time talk shows trying to convince us all that "cellulite is sexy". Hey whatever floats their boat? Huh?)

All too often, athletes assumed "more is better". (Okay re-think the day time talk show issue) Actually, the right amount was perfect. (Huh?) Rather than continuously increasing dosages of Insulin, or any substance, some athletes that became beasts had realized that the problem with decreasing positive results was often due to receptor insensitivity.

In the case of insulin, consider how many diabetics suffer from obesity. Both fat cells and muscle cells (and many others in the body) receive an anabolic signal from mergence occurring between an insulin molecule and its respective receptor-site.

When muscle cell insulin receptor-sites either decrease in number (concentration) or sensitivity, an individual is said to be insulin insensitive. Unfortunately fat cells seldom have this problem. This allows an excess of nutrients to be converted to, and stored as fat. None of the benefits or hyper-recovery could have over ridden insulin insensitivity. But there were drugs, supplements, and diet factors utilized by the largest beasts to over-ride insensitivity.

### WARNING: Science Geek Stuff Follows

Before we discuss "**Super -Cycles- Of -Serious- Sensitivity**", we should have at least set a basic understanding of how insulin actuates a cellular response.

The insulin molecule is much larger than an androgen molecule. For this reason, it cannot simply merge with its receptor-site and take a ride into the interior of a cell. When an insulin molecule merges with its cellular receptor-sites, the release of another compound group is actuated called GPI'S (Glycosylphosphatidylinositols). This in turn frees an insulin mediator group compound called IPG's (Inositol-Phosphoglycans) which enters the cell and triggers a variety of growth signals.

These signals include the synthesis of steroid hormones, proteins, and glycogen. They also initiate the transport of amino acids, glucose, and certain fats into the cell while signaling the synthesis of new transporters. This whole Insulin/GPI's /IPG's process and triggering mechanism is dependent upon a reaction called hydrolysis.

This basic explanation above should provide the realization that several substrates must be available at the cellular level to provide building materials for GPI's and IPG's as well as to act as mediators. If not, then insulin insensitivity/resistance occurs. Yes, this meant a sincere effort to clean up the diet, and some well chosen supplements for all insensitive beasts.

### **(There are Drugs for Insensitive People)**

There are several drugs prescribed by doctors for individuals with insulin insensitivity such as diabetics. Athletes had realized these drugs had a re-partitioning effect upon the muscle-to-fat ratio in favor of muscle gain and fat loss.

Additionally, the drugs acted to increase muscle cell insulin receptor-site sensitivity and number while they decreased fat cell sensitivity. The result was a decrease in dosages used for insulin during protocols and a return to progress for the athletes. Insulin "efficiency" was the actual goal.

### **PRESCRIPTION DRUGS FOR INSULIN INSENSITIVITY**

- Avandia (Rosiglitazone maleate)
- Actose (Pioglitazone HCL)
- Glucophage (Metformin HCL)

There are several others but these are the most commonly found drugs for the treatment of insulin resistance/insensitivity. It should be noted that most drugs used for the treatment of this condition require 12 weeks of continuous use to reach maximum effects. This of course was very beneficial to beasts with a long-term goal of continuous progress as we will discuss later.

### **SUPER CYCLE OF SERIOUS SENSITIVITY**

As you already are aware, insulin is the body's main storage hormone. An increase in insulin sensitivity strongly correlates with less fat accumulation and more protein storage (like uh, muscle). This translates into increased protein synthesis, a positive nitrogen balance/anti-catabolic environment, and muscular hypertrophy.

Several factors can effect insulin sensitivity and therefore affect nutrient utilization efficiency. Dietary, supplemental, training, and pharmacological practices

have had a profound impact upon the body's composition for this reason. In fact, consider this:

- An increase in circulatory insulin levels results in a 50% increase in muscle protein synthesis.
- Anaerobic training (like weight training) resulting in triggering an adaptive response increases muscle protein synthesis 100%.
- Supraphysiological amino acid concentrations in the circulatory system, such as is experienced from ingestion of whey protein, results in an increase in muscle protein synthesis of 150% above basal.
- However, when insulin levels were increased to supraphysiological levels immediately following a high intensity anaerobic work-out in the presence of supraphysiological amino acid concentrations and adequate glucose levels, a synergistic effect resulted in an increase in muscle protein synthesis of 400%!

The first layer of a "Super Cycle of Serious Sensitivity" demanded watching "Titanic" with Leonardo De Caprio and actually *not* cheering when he goes down for the third time. (In the ocean) Joking!

## **LAYER ONE**

The first layer of a "**Super-Cycle-of-Serious-Sensitivity**" utilized Actose, Avandia, or Glucophage. Since all three drugs required 12 weeks of continuous daily use to achieve maximum receptor-site concentration (increase in number) and sensitivity the additional layers took into account this time frame.

### **AVANDIA EXAMPLE**

WK #1-4: 2 MG 2 TIMES DAILY  
WK #5-8: 4 MG 2 TIMES DAILY {WITH MEALS}  
WK #9-12: 6-8 MG 2 TIMES DAILY  
(Maximum dosage for Avandia was 8 mg 2 times daily)

### **GLUCOPHAGE EXAMPLE**

WK #12: 500MG 2 TIMES DAILY (WITH MEALS) (Maximum dosage for Glucophage was 850 mg 2 times daily )



## **LAYER TWO**

Obviously the reason Frank and other beasts used drugs and supplement / diet protocols to increase insulin sensitivity was because the second layer was the administration of an exogenous insulin such as Humalog or Humulin-R.

No exogenous insulin was used for the first 4 weeks of the 12 week protocol due to the reality that supraphysiological levels of insulin down-regulate receptor-site concentration and sensitivity. Remember that the Frank's goal was to increase the positive muscle anabolism aspects of insulin not merely off-set the negative fat anabolism.

The second drug type beasts used to increase insulin levels is called a sulfonylurea such as glipizide or glyberide. These were oral drugs used to treat and control type 2 diabetes (discussed prior in Absolute Anabolic Phases). They worked primarily by causing the pancreatic beta-cells to release more insulin than normal in response to food.

The reason beasts used them in "layer two" was to minimize the inhibition of normal pancreatic insulin release during Humalog or Humulin-R protocols. Short sighted athletes simply injected insulin daily for 4-12 weeks hoping not to become a diabetic for their stupidity.

The wiser beasts used some form of an on-off weekly, or even daily, protocol and used a second drug to decrease the risks of inducing diabetes. Sulfonylurea use required an increase in carbohydrate and BCAA's intake (total caloric intake, as well). Failure to do so could have resulted in hypoglycemia, dizziness, lightheadedness, and shakiness. (Coma and death were possible with inadequate glucose substrate intake)

### **HUMALOG - GLIPIZIDE EXAMPLE (LAYER TWO)**

WK #5-8: Day #1 Humalog 7 iu 2 times daily.  
Day #2 Glipizide 5 mg 2 times daily.  
Day #3 off both drugs. (Repeat)

WK #9-12: Day #1 Humalog 10 iu 2 times daily.  
Day #2 Glipizide 10 mg 2 times daily.  
Day #3 off both drugs. (Repeat)

Between WK #5-12 there were 8 weeks or 56 days so there were nineteen 3 day rotations possible. This meant the on-off protocol of "Day #1-Humalog, Day #2-Glipizide, Day #3 off " was repeated 19 times in 56 days. Ya, I know  $3 \times 19 = 57$  but day #57 was off (duh).

The logic behind this protocol was multifaceted. First there were only 19 actual days of Humalog use out of 56 days total thus drastically reducing negative reactions such as insulin insensitivity and diabetes. Second was the synergistic effect sulfonylurea drugs had upon insulin use.

These drugs actually increased the total amount of insulin "utilized" by avoiding the day after crash normally created through exogenous insulin administration. On day three of each rotation, the pancreatic beta-cells remained sensitive and still up-regulated insulin production above normal. And of course, by maintaining the increase in insulin receptor-site sensitivity, progress was not inhibited.

### **LAYER THREE**

The third layer of a "Super-Cycle-of-Serious-Sensitivity" intended for mass gain was AAS. Exogenous insulin use worked best for sheer growth when a high androgenic environment was created. (Max Androgen Phases worked quite well) In this example though, the goal was to create a long term AAS layer that acted synergistically with the three day Humalog-Glipizide protocol and matched the numbered sequence /rotation.

Note: AAS increase Insulin sensitivity, and insulin decreases SHBG and Albumin.

### **Testosterone S. - Winstrol D. - Finabolan Example (Layer 3)**

WK #5-12: Day #1 -testosterones 50 mg 2 times daily with Humalog. Day #2 Winstrol D. 50 mg 2 times daily. Day #3-Finabolan 30 mg 1 time daily. (Repeat days 1-3 again)

Testosterone Suspension had an active-life of about 24 hours though blood plasma levels remained elevated slightly longer. Winstrol Depot and the black market drug Finabolan both had an active-life of about 48 hours. The result of this three day stack rotation was minimal HPTA suppression and low estrogenic potential with excellent cortisol inhibition. The rotation allowed for minimum AAS receptor-site competition also.

Most Beasts looking for maximum hyper-recovery used this protocol site-specific with a 2 day on /one day off training schedule. This was due to the fact that non-esterified AAS were active upon administration. Let me be sure that this is understood and explain again.

When an AAS is joined to an ester such as a propionate, cypionate, or other, a time release drug is created. As example, when testosterone is a cypionate ester, the average active-life after administration is about 16 days (contrary to what some may believe). This is due to the activity of an enzyme in the body called esterase.

After administration it will take about 16 days for the esterase enzyme to unbind all of a single injection of testosterone cypionate. Until the testosterone is separated from its ester, it is inactive. Testosterone Suspension is raw test in water and Winstrol Depot is a c17-alfa-alkylated drug so both are active upon administration. Finabolan is an acetate ester that is quickly freed due to its short chain structure.

Site-specific refers to administration of a substance at a site "specific to" the area or tissue of a desired response. In the case of fast acting insulin and non-esterified AAS, the drugs were injected directly into the target muscle, in this case, each day after an early morning work-out.

Since disbursement of the site-specifically administered drugs was no longer dependent upon blood flow to the target muscle, hyper-recovery was greatly enhanced. The second reason this was so effective was due to a much lower concentration of the AAS binding proteins SHBG and Albumin in muscle tissue.

## **SIMPLE DIETARY CHANGES**

Simple dietary habits have profound effects upon insulin sensitivity. As example, saturated fats, such as those found in animal protein sources, and fried foods promote insulin insensitivity/resistance. Unsaturated fats such as non-hydrogenated vegetable and nut oils, or flax seed and olive oil, promote insulin sensitivity.

Obviously insulin levels and sensitivity play a vital role in hyper-recovery. But what about the ability to "absorb" the foods, supplements, and pharmacological items that were ingested? Remember, we are what we absorb, not what we eat.

## **ROTO ROOTER? Oust to point out a painful fact)**

The items we eat or swallow first take a trip to the stomach for initial digestive processes. Next, they enter the small intestines where further digestive processes and most absorption occur. Those nutrients and pharmacology items not absorbed continue their quest for freedom by entering the large intestines for continued digestion and minor absorption before exiting and sharing an intimate moment with you and a magazine.

The mucosal cells in the intestines allow water-soluble goodies to pass through to the blood stream and the liver portal vein while oils and fats hit the blood stream by way of the lymphatic system. Unfortunately, the intestines in most individuals look like a kitchen sink drain pipe and therefore greatly reduce the amount of ingested items actually being absorbed. (Back to those intimate moments and money down the toilet!)

One option for intestinal efficiency is a high colonic. It actually makes an impressive difference in the level of nutrient absorption and subsequent recovery/growth. "Butt", there is another option not relating to roto-rooter.

Fibrous foods such as bran, prunes, and vegetables act as a sort of intestinal scraper. It helps, but the best method requires packets of Questran and Metamucil. (Both can be found at most drug stores or pharmacies.) Basically, follow the directions on the containers for one-day-use and drink 8-16 oz of water every hour for 16 hours. This helps flush globules of undigested fats and toxins out of the system as well as reduce total cholesterol counts.

So a dietary game plan for the 12 week period of a "Super-Cycle-Of-Serious-Sensitivity" included a very high fiber intake, a decrease in saturated fat intake with an increase in unsaturated fats, and weekly Questran /metamucil therapy. Oh, and extra magazines in the restroom. Most beasts realized a 20-30 % increase in nutrient absorption and a notable improvement in nutrient utilization.

### **Supplements of Sensitivity**

The most important supplement for insulin activity mediation is the amino acid **L-Arginine**. In fact, simply ingesting 2-4 grams of L-Arginine with a post-work out protein/carb drink increases glycogen synthesis rates 30-40% in healthy individuals.

Arginine is also the precursor to nitric oxide (N.O.) synthesis. N.O. plays a key role in every anabolic metabolic pathway in the human body. The addition of L-arginine's N.O.2 synthesis cofactors, (400 mcg-Folic Acid, 400 mg-N-Acetylcholine, 1000mg phenylalanine) can increase the active-life of N.O. from only a few minutes to about 12 hours. Twice daily is best. Remember insulin, GPI's, and IPG's ? Guess what mediates their activity!

**L-Glutamine** is an amino acid that can be converted into a glucose substrate and utilized for glycogen synthesis. It also acts as a non-insulin dependent mediator to trigger cellular glycogen synthesis and glucose/amino acid up-take. This adds up to decreased insulin resistance. Best daily intake? Up to 120 grams daily, but 20 grams works well.

**The amino acid Taurine** is made in the body from methionine and cystein (e). It occurs in animal origin proteins but not in vegetable protein. Taurine is very similar in action to insulin and aids in prevention of insulin insensitivity by helping cholesterol to remain soluble. 2-4 grams daily with meals is an effective dosage.

**Chromium Polynicotinate** (niacin bound chromium) improves insulin receptor-site/insulin binding or affinity to some extent. The average diet only provides 25-33 mcg of chromium daily and is poorly absorbed (as are most chromium supplements). Chromium Polynicotinate is retained in the body at a rate of about 17% and about

300-311 % better than chromium picolinate. 300-400 mcg of chromium polynicotinate daily is effective.

**Colosolic Acid** acts similar to insulin in triggering a cellular insulin-like response in muscle cells but not fat cells. And it reduces blood glucose about 20% at a dosage of 620 mcg. Colosolic Acid occurs in glucosol powder at a rate of about 600-620 mcg per 50 mg of powder.

**1/8 teaspoon of cinnamon** 8 times daily with food can increase insulin efficiency "up to " 300%. (Cinnamon rolls are not an option).

**4oz daily of the herb fenugreek** reduces urinary sugars about 40-50 % and increases L.H. production. Fenugreek seed has a unique 0.9% content of the amino acid 4-hydroxy-Isoleucine which is a pancreatic stimulator and the ground complex contains HPTA stimulatory saponins. The ground seed is several times more active than the ground herb. However, 4oz. of the ground herb acts to slow the digestion of sugars and is an excellent roughage.

**Sage tea** (2oz of sage) has been shown in some research to increase insulin sensitivity up to 500 % (1 0-20% is more likely)

**Omega-3 fatty acids** increase insulin sensitivity by modulating cellular good PG (Prostaglandin) production. (3-6 grams daily).

We have discussed D-pinitol and lipoic acid in the past. Cinnamon seems to work just as well for some individuals.

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**Editor's Note:** *Wow ... I can't believe we FINALLY reached the end of this book. First, we 'd like to thank you for taking the time to read all the pages, including all the detailed "science geek" stuff. We are deeply grateful for your loyalty and we hope this book will allow you to reach your bodybuilding and "fitness " goals.*

*Of course, we aren 't finished just yet. We have two more books coming out. Part 3 of the CME series is called "Domination". It's finished right now in fact, but guess what ... ALR is writing the entire thing from beginning to end. He's discovered so many new "tricks " to make you GROW that it's almost scary. Anyway, that 'sfor you super hardcore guys.*

*Of course, not everyone wants to or can take steroids - at least not forever and nonstop. That's why we are also coming out with "Building The Perfect Beast ... NATURALLY" as well. That book hasn 't been written yet, but we assume both will be out late 2004 to 2005.*

*Please visit our site at [www.AnabolicBeast.com](http://www.AnabolicBeast.com) for the most recent updated news on our industry, business, books, supplements, chemistry and so much more.*

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A great deal of personal experience and research went into this project, the latter often being referred to as "available literature" or "commonly noted".

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