The year was 1513. Don Juan Ponce de Leon, better known as "Pump" in transcontinental travel circles, was on a seemingly hopeless mission. Pump de Leon, after numerous world travels, many bodybuilding titles, and huge prize monies, had set out to find the fountain of GH. It had been rumored for centuries that in the midst of the islands of the Pituitary there was an amazing fountain. This fountain apparently possessed incredible powers of age restoration. In addition it had such a dramatic impact on fat loss and increased muscle mass that no man who walked away from its waters would ever hit a training plateau again. Being as hardcore as they come, Pump de Leon was possessed to find this fountain of GH, even if it cost him all the gold he had won throughout his competitive years.

Meanwhile, back in Spain, Ferdinand Patterson, Pancho de Luoma and Juan Jose Berardi were determined to get Pump de Leon's story first hand (and to get some of that damn GH!). Casting off on the rickety fishing vessel known as "The Male Hormone," Ferdinand, Pancho, and Juan set sail across the Atlantic in search of Pump de Leon and the Pituitary Islands. After the treacherous journey, they found Pump alone on his own "muscle beach" doing heavy tree trunk squats. After months on the Atlantic with minimal training and poor nutrition, the sailors of "The Male Hormone" were dying for a workout and some good muscle foods. So they began lifting the logs and boulders strewn about in Pump's makeshift gym. Later, over post-workout coconut shakes, Pump shared with his fellow Spaniards the fact that he had not yet found the fountain of GH, but was glad to have three more bodybuilders to help in his quest.

Sadly, Pump, Ferdinand, Pancho, and Juan Jose never made it back to Spain. Nor did they find the fountain of GH.

Now, about 500 years later, the fountain of GH has been found. But not in the area our ancestors sought. It has been found through recombinant technology. And although GH is now available for all, whether it really has the amazing powers that senors de Leon, Patterson, de Luoma, and Berardi sought is another story; one that I plan to tell today.

GH - The Hormone

Growth hormone (GH) is a 191-amino acid protein or peptide that's naturally released from the pituitary gland. GH, much like Testosterone, is released in a pulsatile or episodic manner. The GH pulse occurs every 2-3 hours so each and every day we get about 8-12 big doses of all-natural growth hormone (Hartman et al 1991). The sum of these GH peaks amounts to about 0.5 mg of GH produced per day. The following is an example of what normal 24 hr GH
According to the research review published in a new textbook entitled "Growth Hormone in Adults," the release of GH from the pituitary is governed by a balancing act between 2 hormones; GHRH (growth hormone releasing hormone) and somatostatin. GHRH is responsible for stimulating both the synthesis and the release of GH from the pituitary. Essentially, GHRH initiates the strength of the GH pulse.

GHRH's arch rival, somatostatin, counters these effects, however, by inhibiting GH release. Therefore, somatostatin prevents the GH pulse. In the end, GH release occurs when GHRH is at its peak in stimulating the pituitary, while somatostatin is at its low in inhibiting the pituitary. The result of this high GHRH and low somatostatin period is a big spike in blood levels of GH (Juul, 2000).

The following is a chart adapted from Basic and Clinical Endocrinology, 5th Edition depicting other factors influencing the GH secretion spike:

Once the GH pulse occurs, blood GH is free to affect target tissues. Some of the well-documented actions of GH are increases in longitudinal bone growth (longer bones), increased bone mineralization (thicker, stronger bones), anabolism (protein building), lipolysis (fat loss), and anti-diuretic actions (Bengtsson, 1999). GH treatment is common in congenital syndromes of GH deficiency and in cases of hypothalamic or pituitary damage.

In addition, it's been recognized that around the age of 30, there's a progressive decline in GH secretion from the pituitary, so much so that by the age of 60, GH production can drop as much as 60%! This means that an aging pituitary that once produced 0.5 mg of GH per day would now produce only 0.2 mg per day, and this is definitely physiologically relevant. In fact, these production levels are often equivalent to those of GH deficient young adults. This age-related GH decline has been termed somatopause by some researchers and treatment requires GH replacement therapy.

GH Deficiencies

GH deficiencies in different populations can occur as a result of impaired GHRH activity or increased somatostatin activity, impaired GH production and release within the pituitary, and/or impaired GH interactions with GH receptors on target tissues (Bengtsson, 1999). Basically GH either isn't produced or the GH is knockin' but it can't come in. Regardless of the mechanism behind GH deficiencies, these conditions can lead to a whole host of physiological abnormalities.

In children, GH deficiency leads to a reduced growth rate. This can occur due to the lack of GH specific effects on bone and connective tissue growth. In addition, skeletal muscle growth can be retarded due to other metabolic abnormalities associated with GH deficiency (decreased protein anabolism).

In adults, there are a number of abnormalities associated with GH deficiency. First, GH deficient adults tend to suffer from a host of psychological symptoms. These symptoms include reduced energy levels, reduced vitality, increased anxiety, reduced emotional reaction, depression, hampered learning capacity, and social isolation (Bjorck, 1989).

Secondly, GH-deficient adults suffer from negative changes in body composition such as increased fat mass, especially in the abdominal area (called android fat distribution), decreased lean body mass and muscle volume, and reduced bone mineral content (Binnerts 1992, Bengtsson 1993, Rosen 993). As a result of these negative changes in body composition, decreased muscular strength, poor exercise capacity, and poor power output are a result (Cuneo 1990, 1991).

Finally, GH deficiency can lead to other symptoms such as dehydration, reduced heart size, reduced cardiac performance (measured by cardiac contractility and output), hypertension,
Hey Doc, How's My GH?

So how do you know if you need GH treatment? That's a good question that scientists are still trying to answer. And you can bet that if they're having a hard time with this question, most physicians are quite a bit behind them. Since the symptoms of GH deficiency in adulthood (increased adiposity, decreased muscle mass, reduced strength and exercise capacity, and psychological disturbances) are non-specific, a deficiency based on clinical symptoms is difficult to diagnose. Therefore, biochemical markers must be used.

Random sampling of plasma GH isn't a sufficient measure due to the unpredictable pulsatile nature of GH secretion shown in the graph above. If you pull a sample at the peak of a GH burst, it looks like you're fine, but if you pull one at the "trough"; it looks like you need some GH. Normal fasted levels of GH are less than 5 ng/ml, but again, the utility of random sampling is limited. By taking a 24-hour integrated measure, you could get a good approximation of total GH secretion, but who wants to sit in the doctor's office for 24 hours and have 24 blood samples taken; one every hour? Not me!

Therefore, the best clinical test for GH secretory deficiency is an ITT or insulin tolerance test. With this test, a single dose of insulin is administered to promote hypoglycemia. If you check your chart above, you'll notice that hypoglycemia is a good GH secretory stimulant. So, as insulin goes up and blood glucose goes down, GH secretion should go up. Since this test only measures GH secretion and not GH action at the receptor level, other tests are required to determine GH deficiency.

Serum measures of IGF-1 and IGFBP-3 are two markers of GH activity but their utility has been questioned (more on these later). Since daily IGF-1 levels tend to be stable, in the clinical setting, low IGF-1 levels can indicate the need for further assessment of GH secretion and function. Normal IGF-1 levels are 90-318 micrograms/l while IGFBP-3 levels are 2.0-4.9 milligrams/l.

Effects of GH Replacement

Since GH deficiency leads to the aforementioned frightening list of psychological and physiological abnormalities, the treatment of GH deficiency has received much attention within the medical community. In clinical trials, most of which were referenced above in the "deficiency" section, GH replacement has been shown to remedy most of the physiological abnormalities. The major benefits of GH therapy include positive protein balance (synthesis exceeds breakdown), increased lean body mass, decreased fat mass, increased insulin sensitivity, normalized body water, increased bone remodeling, and increased T4 to T3 conversion.

What about side effects? In GH deficient patients, replacement therapy is usually associated with minimal side effects. The most common side effects typically occur with the onset of therapy but often tend to normalize within a few months' time. These negative side effects include fluid retention, carpal tunnel syndrome, myalgia (muscle pain), and arthralgia (joint pain). In addition, fasted and post-prandial (post-meal) blood glucose levels tend to be higher in GH replacement as a result of the mild insulin insensitivity that can occur with doses in excess of the exact requirement. Finally, it's been suggested, but not verified, that GH replacement may lead to a risk of malignancy and some cancers.

Although there are a few risks with GH replacement, the risk to benefit ratio of GH therapy in grossly deficient humans remains positive. Since GH can be relatively safe in replacement situations, as well as the fact that GH treatment can greatly impact body composition, researchers and clinicians have begun to explore the use of GH in treating the negative physiological conditions caused by HIV or age-related muscle wasting, obesity, severe physiological stressors (surgery or burn injuries), nutrient restriction, glucocorticoid-induced muscle atrophy, and impaired immunity. Unfortunately, the data are mixed in regard to GH therapy in these populations with some studies showing positive results in muscle mass and fat loss and others showing nothing but side effects.

One reason for this may be the fact that in some studies, GH treatment has been given alone while in others, GH treatment was given with several other hormones that may have acted synergistically with the GH to promote the positive changes. One thing is clear though: there is no clarity! At the doses given in research studies, there is no clear consensus on whether GH therapy is warranted in any population other than those with GH deficiency. More research is needed to make this determination.

How GH Works - The GH/IGF-1 Axis

Due to the rise in recombinant GH availability, the research has been abundant and a clearer picture is emerging of GH action. But make no mistake, the picture isn't all that clear. It may be more like one of those computer-generated 3D pictures that you have to look at in just the right way for just the right amount of time to make any sense of it at all. And no one has yet to look long enough at this particular picture.

With all of this GH floating around, the black market supply of GH has also been on the rise. So after we talk GH action, let's talk bodybuilding. If GH can potentially get bodybuilders big and ripped, then to some, it's a drug worth exploring. So for you die-hard muscle heads,
Circulating GH is thought to act through two distinct but interrelated mechanisms. The first is direct. GH can act directly on many cells in the body via the GH receptor. Once released into the blood from the pituitary, GH either circulates as free GH or circulates bound to GHBP (GH Binding Protein). Free GH is available to interact with cellular receptors to create a response.

Once free GH has interacted with the cellular receptors, it's thought that more GHBP is formed. With this increased GHBP, some researchers believe that more GH is rendered temporarily unavailable. But at the same time, it stays in the system for a longer amount of time. So although GHBP-bound GH has a much longer half-life, it cannot interact with cellular receptors while bound.

Unfortunately, there's no clear consensus as to whether it's more important to cellular GH action to prolong the half-life of GH (to allow for higher levels to circulate for longer), or to decrease GHBP to allow for higher levels of free GH. And this debate holds true for not only GH, but for other hormones like Testosterone as well. Although the researchers tend to contradict each other and sometimes even themselves on this point, the bottom line is that the effectiveness of GH (and other hormones) is tied up in this balance between bound and unbound GH and the presence of binding proteins.

Binding proteins aside, once free GH does reach the cells, its direct actions include the promotion of lipolytic and hyperglycemic effects. GH can decrease glucose utilization in favor of fat release and oxidation (lipolysis). Unfortunately, because of this shift from carb to fat use, GH also increases insulin resistance. Hyperglycemia is a result of this insulin insensitivity. So although GH itself can make you lean due to lipolysis, this might come at the expense of insulin resistance and might ultimately lead to a diabetic state. As a result, you'll be a lean diabetic rather than a chubby normal guy. I guess it's a trade-off.

The second mechanism by which GH exerts its effects is indirectly through IGF-1. In the liver, circulating GH is converted into IGF-1 and 2 which can travel through the blood to promote their effects. IGF is also bound to one of 6 plasma proteins (IGFBP's 1-6). About 1-5% of IGF-1 is free while 95-99% is bound. Again, this balance is important for hormone action. This systemic IGF is also free to interact with cellular receptors.

In addition to the systemic effects of liver IGF-1, IGF can act locally. Let me explain. GH binding to cells can lead to what is called peripheral conversion of IGF-1. At this specific location (skeletal muscle for example), IGF-1 acts in an autocrine or paracrine fashion to promote its effects. This means that unlike GH, which has endocrine function (it is produced in the pituitary and travels elsewhere to do its work), IGF-1 can both be produced in, and promote changes in, the same tissue or those immediately adjacent to it. Perhaps the most relevant effect of IGF-1 to this discussion is the ability of IGF-1 to increase protein synthesis by increasing cellular mRNA formation (mRNA makes protein) as well as increasing uptake of amino acids. This effect on protein synthesis can lead to increased lean mass. The research indicates that this effect is dependent on GH presence as well. So IGF-1 alone does not promote such effects. Nor does GH. It appears the combination of the two most consistently lead to increased protein synthesis.

In addition, IGF-1 can also counteract the hyperglycemic effects of GH via insulin-like actions on glucose uptake. Since IGF-1 is typically elevated to a small extent with GH elevations, IGF action is not sufficient to neutralize the hyperglycemic effects of GH, but perhaps it minimizes extreme insulin insensitivity.

The bottom line is that GH and IGF-1 seem to be necessary bedmates. Although each may act most strongly in different tissue types, they are thought to work together to promote anabolism and stimulate lipolysis (Ney 1999, Yarasheski 1994). But all this synergy comes at a price. Both hormones negatively feed back on the pituitary to slow GH production. And this impacts normal GH secretion as well as GH treatment.

When plasma GH levels and IGF-1 levels are elevated with GH treatment, this elevation is non-physiologic. What this means is that after a GH injection, GH levels are elevated for some time, and then come crashing down to normal, often being suppressed for hours thereafter. So the pattern seen in the graph above is not the one seen when using exogenous GH. This is probably due to the fact that both GH and IGF-1 are negative regulators of GH release so an increase in either (from a GH injection) reduces the secretion of GH.

So when examining the GH/IGF-1 axis, a few things should be considered. With strong feedback mechanisms in place, it's difficult to maintain consistently high levels of GH without constant exogenous dosing. And that's a hassle. In addition, just like with insulin, there may be something known as GH insensitivity (Grinspoon 1998). It appears that with chronically high levels of GH, liver and peripheral conversions of GH to IGF-1 are decreased. So even with the constant use of exogenous GH, the body may simply try to regulate itself and the actions of GH by preventing the availability of what is thought to be GH's partner, IGF-1.

It seems like a no-win situation. And perhaps this is best. The body has feedback mechanisms for a reason... protection. If GH action isn't kept in check, the medical condition known as acromegaly can result. Acromegaly is characterized by abnormal skeletal growth characterized by enlarged jaw and hands. Individuals suffering from this have abnormally high levels of GH, IGF-1, and IGFBPs. It's apparent, then, that the feedback mechanisms of these individuals aren't working all that well.

Often times, GH users smugly tell me that acromegaly is BS because they've been using GH for X amount of time and they didn't get it. Well guys, guess what? Normal individuals
The Perfect Physique?
GH, Muscle Function, and Body Composition Research

Since most of the benefits of GH were originally thought to impact muscle mass, scores of rodent studies were conducted to examine the effect of GH on muscle mass and contractile ability. The findings did indicate a small increase in muscle mass but no increase in contractile strength. One study looked at rat quads (no they didn't squat) and they did get bigger (quads), but not stronger (Bigland, 1953). In addition, in other rat studies, although there were small increases in body mass, there were absolutely no increases in strength. How could this be? More muscle equals more strength, right? Well, researchers concluded that the increase in quad mass was not contractile protein. The mass could have been fluid or connective tissue.

Since animals did benefit from increased muscle mass, the next step was to take these findings to humans. In cases of GH deficiency, small increases were found in muscle volume (~6-8%) and lean body mass (~11%). Exercise capacity was elevated in such patients (~12%), but strength was either not changed or mildly increased by about 8% (Jorgensen 1989, Salomon 1989). As stated earlier, most of the observed benefits of GH have been seen in GH deficient animals and humans.

Also, as mentioned earlier, there's certainly not much to get excited about in other populations. When GH is administered alone, very few studies have shown any increase in size or strength. In two recent HIV studies, patients given huge doses of up to 27 IU per day (9 milligrams) had no gains in muscle mass. But remember, according to what I said earlier, IGF-1 was the protein anabolic agent. And GH has its biggest effect on lipolysis. And the combination of the two may lead to the greatest results.

So in examining the research, it's been speculated that the levels of IGF-1 administered weren't great enough (in conjunction with GH) to make an impact, or that the individuals became GH resistant. Also, since IGF-1 would lower GH secretion, it doesn't make much sense to give it alone. Remember, GH and IGF-1 often work together to change body composition. Newer studies have shown that when adding IGF-1 to the mix, it appears that there's a definite increase in protein synthesis and muscle mass as well as some increase in strength.

So perhaps GH alone is useless at increasing muscle mass while a combination of GH and IGF-1 may be effective if protein anabolism and increased contractile protein is the goal (Kupfer 1993, Snyder 1988). But even the increases seen in these studies were moderate and a cost/benefit analysis is warranted since this combination might also lead to severe side effects.

So what about GH and fat mass? Most studies have shown modest decreases in body fat and skinfold measures with GH treatment (Jorgensen 1989, Salomon F, Tagliaferri 1998). Decreases in fat mass of about 16% and decreases in thigh adipose mass of about 7% have been reported. But remember, a 16% fat decrease doesn't mean they went from 20% to 4% body fat. It more likely means that a 200 lb person with 20% bodyfat or 40 lbs of fat would have their fat mass decreased to about 35.5 lbs. This would put them at about 193.5 lbs and 18% fat.

In another study, obese women on GH lost 2 more lbs than placebo group in a one-month period. So although it does appear that GH can decrease fat mass in clinical populations, when looking at the actual fat loss numbers, it appears that the good old ECA stack or MD6 would be more effective than GH.

GH and The Athlete

I've never been sure why the use of GH has become popular in athletes and bodybuilders. Perhaps it's the name... Growth Hormone. Sounds like it'll make me big. Or perhaps it's the legend of Pump de Leon. Either way, the research on GH use in bodybuilders and men on resistance training programs has shown it to be all but useless. And this is probably due to the feedback mechanisms like the negative feedback on the pituitary and the GH resistance discussed earlier.

In two landmark GH studies conducted at the Washington University School of Medicine, a world-renowned GH researcher named Kevin Yarasheski studied the effects of GH in combination with weight training (Yarasheski 1992, 1993).

In the first study, 18 untrained men were given either GH and exercise or placebo and exercise for 12 weeks. GH subjects were given 40 micrograms/kg of recombinant GH and all subjects were evaluated before and after treatment for fat mass, fat free mass, total body water, whole body protein synthesis, insulin sensitivity, muscle size and muscle strength. Due to the development of carpal tunnel syndrome, 2 subjects were forced to withdraw from the study.

When comparing the GH+exercise group with the placebo+exercise group, the data showed that there was no fat loss, no change in insulin sensitivity, no increase in muscle size, and no increase in strength! Whole body protein synthesis was increased in the GH group relative to the placebo, but muscle protein synthesis wasn't. In addition, lean body mass was increased, but again, this wasn't muscle mass, but probably a combination of water retention, organ mass, and connective tissue instead. The researchers, who seemed quite objective in their conclusions, decided that non-muscle proteins were being formed instead of muscle contractile protein.
In the follow-up study, Dr. Yarasheski pursued the effects of GH on experienced weightlifters. Since the GH didn’t positively impact strength or body comp in the untrained guys, Dr. Yarasheski wondered if well-trained athletes might be different. So another study was conducted to examine protein synthetic rates in GH-treated athletes. After 2 weeks of GH treatment (40micrograms/kg), the data were clear that short term GH had no effect on whole body protein synthesis or breakdown. The reason they chose 2 weeks was that in a number of previous studies on clinical populations, any increases in protein synthesis had only lasted for about a month and then ceased due to some type of down-regulation (Perhaps GH insensitivity?). In this population, however, GH didn’t even promote protein synthesis within this time frame.

With all this negative data, it should be mentioned that one study showed something positive happening, but again, it wasn’t all that exciting (Crist 1988). This particular study showed a small 4% gain in lean body mass and a modest 12% loss in body fat with GH doses of 8IU per day (2.6 milligrams). Muscle mass wasn’t measured, so there was no way to determine the make-up of the increased LMB (lean body mass).

So it’s pretty apparent that in weight trained men, GH alone doesn’t increase muscle mass. Resulting lean mass gains from GH treatment are probably a combo of water, connective tissue, or organ mass. I say probably because organ mass and connective tissue mass are hard to measure. The indirect evidence is pretty strong, though.

Since non-muscle protein gains and the development of carpal tunnel syndrome (due to growth in the connective tissue sheath in the wrist) were apparent in these studies, connective tissue gain is a reasonable speculation. In addition, acromegaly patients have increased organ mass as a result of the high responsiveness to GH, so it would stand to reason that this could have occurred in these studies, too.

The next logical question is: Since a lot of guys are still using GH, what are the implications of increased organ mass and connective tissue? Well, to be honest, we don’t know.

Acromegaly patients do not have high rates of organ malfunction or pathophysiology, so although growing large organs isn’t ideal, the current literature doesn’t indicate that the problem is immediately life-threatening. But, acromegaly patients do die prematurely, so if they were to live longer, perhaps these organ changes could have long-term impact.

As far as the issue of increases in connective tissue, the increases themselves may not be too terrible, as long as they don’t become pathophysiological. Of course, developing carpal tunnel syndrome is no picnic. On the other hand, if the strength of connective tissue increases with connective tissue growth, athletes could become more injury-resistant. Connective tissue growth will not lead to strength increases in well-trained guys if contractile protein mass doesn’t go up, but these connective tissue increases may allow individuals to train with heavier weights with less risk of injury. This, however, merely results from me taking off the “science hat” and speculating a bit.

Let’s Get Ready to Rumble

GH vs Testosterone and Beta-Agonists

With all this data flying, I think it’s important to put things into perspective. Currently, far and away, the most popular bodybuilding drug for building muscle mass is Testosterone, while the most popular fat-loss drugs are the beta agonists clenbuterol and ephedrine. So if GH is to have any relevance to bodybuilders and athletes, it has to show itself to be superior to these drugs in terms of effectiveness, safety, or price. Since we all know that the price of GH is astronomical (it can run $1000 ++ for a month’s supply), the price situation is a losing one on the GH front. What about the other two factors?

As stated in the above sections, fat loss with GH is moderate and GH can probably be outperformed with a simple ECA stack. In addition, it appears that even Testosterone, while not known for its fat-burning abilities, does a nice job of its own. In two studies, Testosterone was shown to decrease fat mass by 5% and 6% (Anawalt 1999, Blackman 1999). In one of the comparing the costs of different drug therapies if you were to obtain them legitimately with a prescription. I’ve also added the cost of MD-6 for a little comparison:

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So Long GH
New Options in GH Manipulation

Over the last few years, GH has been a relative disappointment in terms of treating catabolic/wasting disorders. And it has obviously been a disappointment for athletes and bodybuilders. So the pharmacologists got to work and built a better mouse trap. It has been proposed that GH has been disappointing because of the feedback mechanisms described earlier as well as the non-physiologic nature of GH treatment. What this means is that since GH is normally pulsatile, the body may be best adapted to this situation. Perhaps it likes to see frequent short bursts of GH rather than huge single increases followed by hours of suppression.

Since GH treatment results in these non-physiologic GH responses, pharmacologists have speculated that an oral GH secretagogue that could increase the burst frequency and burst amplitude (height) might offer the distinct advantages of less negative feedback, less GH resistance, a better risk profile, and a better mode of delivery (oral).

Lo and behold, such secretagogues, called Growth Hormone Releasing Peptides have been found. Growth hormone releasing peptide 6 (GHRP 6), Hexarelin, and MK-0677 are available and fit the bill. Whereas a GH injection might cause a large spike in GH and the suppress GH for hours thereafter, these drugs, increase GH frequency and amplitude in a more physiological manner as shown below:

As shown, the GH secretagogues offer a pulsatile GH release that is more physiologic than the GH burst that a GH injection gives. Of additional interest is the fact that the inclusion of GHRH injection with GHRP (not shown) can lead to this same profile with huge, rapid peaks in GH release.

With an understanding of natural GH release it is clear that these new types of GH therapy may offer future treatment options for GH deficiency. In the absence of good safety or body composition data, it is uncertain as to how they will be used or what populations will benefit the most from their use. If these drugs do become more popular treatment options, I would expect that bodybuilders will be testing them out as well and will provide feedback on their efficacy.

If you'll permit me to speculate about potential body comp implications, since GH has shown to be a more effective fat loss agent than anabolic agent, these secretagogues may offer a new and better fat loss approach. Since even just a physiological burst of GH increases lipolysis (Gravholt 1999), especially in the abdominal area, the very large bursts seen with GH injections may not be necessary. They may not lead to increased lipolysis above normal or mildly supraphysiological pulses. And since GH secretagogues mildly increase frequency and amplitude of GH secretion, this increased GH activity may be even more effective at promoting fat loss than GH alone. So if some supplement company comes out with a real-deal, honest-to-goodness, GH secretagogue that really works, it may be a great supplement to promote lipolysis. But for now, the only effective secretagogues I know of are the ones discussed in this article.

GH Plus

Within the last few years, the bodybuilding community has taken drug use to a new high. Being extremists by nature, bodybuilders are always looking for the next drug or combination of drugs to take their muscle mass to the next level. To this end, the new generation of bodybuilders have sworn by a combination of Testosterone, GH, IGF-1, Insulin, and Thyroid drugs. A discussion of these combinations is beyond the scope of this article and beyond the scientific literature at the current time. There is quite a bit of indirect evidence suggesting that, in theory, there may be a synergistic response to a polypharmacy of this type, but there have been very few trials looking directly at such combinations (Mani Maran 2000, Painson 2000, Demling 1999, Grinspoon 1998 and 1999, Juul 1998, Keenan 1996).

The body of anecdotal evidence is greater and I've talked to tons of guys who have used GH, T, Insulin, Thyroid, etc. Many feel that the addition of GH to a drug stack results in some pretty good gains while some say that they don't think the GH helps them at all. But who really knows how much each drug contributes? Since each person is different, uses different doses, and may or may not have real drugs, comparisons are difficult. At a price tag of $1000+ per month for the GH alone, I just don't think that the gains would be worth it either way.

My personal feeling is that when drug use gets to this extreme level where it is "necessary" to take 5 or 6 dramatically powerful, incompletely understood, and potentially dangerous hormones to compete. I think it has gone way too far. Although it's pretty interesting to think that we could control our body compositions by taking the endocrine system off auto pilot and controlling it manually for a while, we may get more than we bargained for.
Auto pilot may never work again and you'll be trying to figure out how you're gonna pay the hormone replacement bills for the rest of your lives. I just don't want to be 65 years old and still giving myself a dozen injections per day because I turned my pituitary into a shriveled, dangling waste of endocrine tissue hanging from my atrophied brain mass.

References:

Great read......

Awsesome post !!

So what if you were to shoot it 2-3 times a day? Also, why did the guy lose his 12am pulse when he shot the synthetic gh at noon?

great read bro.
IGF-1 stays elevated quite long after GH administration and is one of the reasons for GH suppression.

So what if you were to shoot it 2-3 times a day? Also, why did the guy lose his 12am pulse when he shot the synthetic gh at noon?

Any thoughts on the Nutropin Depot? Any word getting to you about this long-acting formulation?

I don't have much info on Nutropin, Miracle Man over at Elite seems to like it. Ironmaster has started using it also.

I don't think there will be much difference in efficacy.

Awesome post brother. And Bouncer.....quit lying, we all know it took you 3 1/2 hours to read that post, not '25 minutes'.

This is an article from Testosterone, the online magazine (T-Mag). The author started with the premise that GH didn't work, and took only the parts from the research that supported his pre-determined conclusion. He also ignored more to-the-point studies that undermine his conclusion, including the research results of the top name GH manufacturers. And he has NO explanation for the huge difference in appearance of today's top pro's compared to those of yesteryear. (the difference is GH and insulin).
Still, there is some very good info here.....especially regarding the complex interactions of the human endocrine system.
The drive to popularize growth hormone began about 20 years ago with publication of the book "Life Extension: A Practical Scientific Approach™", by Durk Pearson and Sandy Shaw. In other words, while a "Fountain of Youth" pill sounds like a pretty nice invention for those struggling toward middle age and beyond – and the lower metabolism and waistline battles that go along with it. Ultimately, the adage from Joan Welsh still holds true: "A man’s health can be judged by which he takes two at a time – pills or stairs." Do yourself a favor, and opt for the stairs. You’ll be better off for it.

References & Image Credits: (1) PR Newswire (2) PR Newswire (3) PR Newswire (4) NIH.gov (5) Quackwatch.com (6) LeavingBio.net.

Growth hormone boosts bone metabolism. Since GH stimulates bones to grow longer and larger in children and adolescents, it makes sense that it continues to stimulate bone growth in adults by maintaining and increasing bone density. Bone remodeling is the process of new bone formation by osteoblasts and bone resorption by osteoclasts, the essential out-with-the-old and in-with-the-new system by which our body keeps our bones young.

Knowing what exercises to do and when can be daunting, so I’ve taken the guess work out of working out by creating a simple and effective program that activates your body’s natural systems for creating bone growth.