HORMONAL NUTRITION

Hormones are Controlling Your Metabolism
Eat Food to Change Your Hormoes
Lose Fat, Gain Muscle, and Get Healthy

Kevin Kuhn, M.S.Ed., CSCS

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Introduction

They say that KNOWLEDGE is POWER. I believe this very strongly; however, I believe that knowledge with practical application is even more powerful. So, my first question to you is: Do you feel that you have POWER over your metabolism? In other words, do you feel that the diet you eat places you in control of how you look, feel, and move? If you eat the "Standard American Diet" (SAD), the answer is probably "no." This is because the "western" diet is full of foods that are high in calories but contain little nutritional value, but also because the amount and types of foods that we, as a society, have been told to eat have caused a shift in our metabolic balance because of the burden they place on our hormones. The issue of calories is part of the equation, but should no longer be the focus.

When you put something into your digestive system, it is NOT the energy value of that type of food that decides how your body will use it. The amount of energy needed to digest protein, carbohydrates, and fats is not equal, and the hormones needed to breakdown, use, or store protein, carbohydrates, and fats are all different. Yet all we have ever been told is that too many calories cause weight gain, and reducing calories will reduce weight. If all we did in life was sit in a chair and eat only 1 kind of food, the previous "equation" would be fine, but no one can do that! Different kinds of food affect your metabolism differently. This is because your hormones dictate how your body uses food, and different foods dictate which hormones can do their job. The Standard American Diet results in specific

hormones being produced to such a great extent that opposing hormones that are needed to maintain metabolic balance are unable to be produced so that they can do their job. The SAD diet and "calorie-focused" standard of nutrition results in a lopsided metabolism and takes YOU out of the driver seat. You can be a passenger with no say in where you and your body are going, or you can take back the wheel and decide for yourself where you metabolism is headed.

We are MALNOURISHED. As a society, we are under-nourished. If you are reading this, you probably have 3 meals a day, and have no financial issues with buying a snack when you need one. So are we a "fat" society because we eat too much, or because we do not eat enough? I think we, as a society, eat too little of the things that provide the most benefit to our bodies. We are over-fed, but under-nourished. This, in my opinion, is one of the biggest factors contributing to the obesity, diabetes, and inflammatory disease epidemic in western society. Everyone "knows" they should eat more fruit and veggies, but the issue is not that simple. There is more to it than that!

The issue of obesity and weight gain is much, much more complex than ingesting too many calories and not expending enough calories. The days of eating less than normal while exercising more than normal should be done away with. The fact of the matter is that this approach to fat-loss just does not work for people. If it were this simple, than why is fat loss and muscle-gain so difficult? Part of the problem comes from where we get our information. Across the board, there seems

to be no consensus on what actually works. High-carb/low-fat? High-fat/low-carb? Atkins? The list is virtually endless. I wrote this book, not to defend a specific diet type, but to see what science and research explicitly says about how our food choices affect our hormones, because it is our hormones that are running things. We can argue about the "value" of calories all day, but when it comes down to it, the presence, or absence, or specific hormones dictates how our body processes food, and the food we eat dictates how we produce hormones. The nutrition standard we have right now prevents the optimal production and function of specific hormones, and thus causes not only weight gain (and all the diseases and conditions associated), but also prevents gains in muscle mass. So I propose a paradigm shift. If we shift the focus from "I can only eat 'x' amount of calories," to "I need to eat food 'x' because it will have a specific effect on the production or role of hormone 'y,'" we can lose fat, increase muscle, and improve our health.

After reading this book, I am sure you will agree.

I confess: This paradigm shift is not originally mine. Many others have been promoting this ideal. So I am indebted to individuals who worked to better understand these concepts and its out-workings, and have thus taught me different aspects of hormone nutrition. Though this concept is not original, the task of compiling the information in this book is. I hope my words and the research presented further promote this concept and provide a foundation for better health in your life.

So what hormones are we talking about? Well, there are very many, but this book will cover 12 specific hormones involved in metabolism. These hormones are: insulin, adiponectin, leptin, ghrelin, glucagon, CCK, PYY, GLP-1, cortisol, testosterone, growth hormone, and IGF-1. We will cover the specific role that these hormones have in relation to metabolism. We will also cover the role that specific food types (Protein, Carbs, and Fat), dietary supplements, and lifestyle factors and behaviors have on the production of these hormones, and how that affects your metabolism. Finally, I'll explain how to put everything together into a plan of action so you can improve your health, lose fat, and gain muscle.

Chapter 1: **DIETING, FAT-LOSS, AND MUSCLE GROWTH**

If you and I have ever had a conversation about weight-loss, I probably asked you this simple question:

"Do you want to lose weight, or do you want to lose fat?"

I've asked this question quite a few times, and typically get one or the other of these two answers:

"I want to lose fat"

"Is there a difference?"

The reason I ask this specific question is because most people do not know the difference between weight-loss and fat-loss. To illustrate this point, I'll give you an example. I've never had someone say to me:

"Kevin, I just want to get rid of this 10 pounds of muscle I've put on."

Or

"Kevin, I just wish I wasn't so hydrated, because I would love to get rid of 10 pounds of water-weight."

That seems silly, doesn't it? Perhaps it is silly, but that is the exact difference between weight-loss and fat-loss. You can lose weight on ANY diet, however, there is no guarantee that the weight is entirely fat. In fact, research on most diets (when calories are limited to a certain amount) show that if weight-loss occurs, up to 50

percent of the total weight lost can be muscle, water, and other lean tissue, while the other ~50 percent is fat.

You read that correctly. Only ~50 percent is fat. I think that is unacceptable, and I hope you agree. But, perhaps you do not care if approximately 50 percent of weight-loss is muscle. If that is the case, then I have a few things to tell you that I think may change your mind.

First, I would like you to think about you goal body weight. Most of us have a specific number, or at least a range, we would like to see every time we step on the scale. I think a much more important number to strive for is your goal fat percentage. Let's just say, for example, that you want to weigh 100 pounds. Do you care if you weigh 100 pounds, but 50 percent of that is fat? Or to take it one step further, would you be happy if you were at your goal weight, but you were 75 percent fat? Hopefully you care about what that 100 pounds is composed of. This is where the issue of body composition comes into play. Body composition is just a fancy way of saying "this percentage of your body weight is lean tissue (muscle, bone, tendons, ligaments, etc.), while this percentage is fat." Generally speaking, and from the health and wellness perspective, it is better to have a higher percentage of lean tissue and a low percentage of fat. The exceptions deal with those who have too low fat percentage, which has its own list of concerns and complications that will not be discussed here. So back on track.

Calorie restriction diets do not take into account that weight-loss is not the same as fat loss. If you are currently on a calorie restriction diet or point-limiting diet, you may not care what you are losing as long as you are losing something, but in the long run, you may not be so happy with your results. The reason you will not be pleased in the future is because most of the weight you lost (50 percent muscle and 50 percent fat) in muscle and lean tissue will be replaced with fat. That's just another way of saying that dieting slows your metabolism. When your metabolism slows, the body becomes very good at storing fat, and not using it. This is a simplified analogy, but the idea holds much from a practical standpoint: You are either storing fat, or building muscle. Ladies, you do not have anything to fear from gaining muscle mass. Your cloths with fit better, you will feel better, you will move better, and you will be healthier! Men, the same goes for you! In fact, if fat-loss is your goal, then increasing your muscle mass will allow you to better reach your goal, since increasing the amount of lean tissue you have increases the amount of calories you use (speeds up metabolism). But, you cannot increase muscle mass while restricting the number of calories you eat. So from the big picture standpoint, calorie restriction decreases the body's muscle and lean tissue, and thus slows down metabolism. The weight that is lost (50 percent muscle and 50 percent fat) during the diet will be gained back, and most, if not all, of that will be fat. Calorie restriction for most people is just a short-term weight-loss fix that results in long-term fat gains. This increase in fat mass can both directly and indirectly lead to issues of metabolic syndrome, type II diabetes, cardiovascular disease, and other serious issues. Simply put: Calorie restriction for weight-loss is pretty unhealthy.

Secondly, as we age, both our muscle mass and bone density begin to decrease. Research shows that on average, most people will loose 8 percent of their muscle mass every decade starting at age 30. If we are not proactive about maintaining both muscle mass and bone mineral density, we are increasing the risk of falls, broken bones, and just not being able to get around! Age does not have to mean limitations and frailty. Calorie restriction speeds up the aging process by reducing the muscle we need later in life. What about bone mineral density? Ladies, I'm sure you have been told by your doctor or a friend that you need to make sure you get enough calcium in your diet. The truth of the matter is that most people get enough calcium and other minerals to maintain bone health and prevent osteopenia/osteoporosis. So why do so many people have bone density issues as they age? Well, the first issue is that the bones will not become stronger if they are not stressed. This just means that in order for the calcium in your diet to be used to strengthen your bones, you have to exercise (especially exercise the involves a loading effect, like resistance training). The second issue is related to sarcopenia, which is just the medical name for reduction in muscle mass as we age. It is hard to load the bones to maintain their strength without the muscle to move and exercise! So your muscle mass right now is directly related to your muscle mass and bone health down the road.

Lastly, I want you to know there is a better way! It is possible to alter your diet to increase muscle mass and the amount of fat you use (and thus lose!) while

limiting the amount of water, muscle, and other lean tissue that is lost. This is accomplished by switching our focus to how food affects our hormones.

HORMONES

Hormones play a major role in the body, dictating whether we gain weight or lose weight. Hormones are responsible for just about every process that takes place in your body. Simply put, they decide if you store more fat than you use or build more muscle than you break down. The basic formula for consistent fat-loss or consistent muscle-growth then is: use more fat than you store and build/repair more muscle protein than you break down.

Or perhaps it is easier seen this way:

- Increase the amount of stored fat that is used as fuel
- Decrease the amount of fat that is stored
- Increase the amount of muscle that is repaired / Increase muscle mass
- Decrease the amount of muscle that is broken down

Simple enough, right? Well, the hormones that control these processes, which are occurring all the time, are directly influenced by the food we eat. So fat-loss and muscle growth can be easy once you know what foods affect specific hormones, and what those hormones do specifically to your body.

If you want to just lose weight, then stop eating, but keep in mind that in order to continue to lose weight or maintain the weight that is lost, you have to stop eating food all together, or eat fewer and fewer number of calories. If you follow the logical outworking of calorie restriction dieting, the metabolism continues to slow as fat mass replaces muscle mass. By the way, this is not healthy. Your entire body becomes the victim of starvation.

So what is the alternative? Prepare yourself for a paradigm shift.

YOU HAVE TO EAT FOOD TO LOSE FAT, AND YOU HAVE TO EAT FOOD TO GAIN MUSCLE!

That's the bottom line. The type of food dictates the hormone response, and whether fat is stored or muscle mass increases. What foods do you need to lose fat? We'll cover that! What foods do you need to gain muscle? We'll cover that, too! So please allow me to lead you through the rabbit hole of confusion that food can be, so YOU can take control when it comes to changing your body.

Chapter 2: **HORMONE TYPES AND HORMONE RESISTANCE**

Anabolic vs Catabolic

Since hormones are running the show, it is important to understand if they are helping your, or hurting you. The human body is an amazing and complex system, so we will only cover the ones that are currently the most important with regards to body composition and weight loss and gain. Before we go into the specific hormones, it is best to categorize them into two major groups: Anabolic and Catabolic. "Anabolic" simply means that a reaction occurs to combine small (simple) molecules into bigger (more complex) molecules. "Catabolic" is just the opposite, where a reaction occurs to break down bigger (more complex) molecules into smaller (more simple) ones. Typically, anabolic reactions build a new molecule or store some sort of energy, while catabolic reactions release energy. Both are completely necessary for normal functioning, and both are occurring to some degree all the time. So metabolism is the sum of all the anabolic and catabolic reactions that take place within you. The issue of fat-loss and muscle-gain is to maximize skeletal muscle's anabolic reactions (muscle building) and minimize catabolic reactions (muscle break-down), while simultaneously minimizing the storage of fat (anabolic) and maximizing fat breakdown (catabolic). So now lets dive into some of the "key players". This is by no means an exhaustive list of the hormones involved in metabolism. As science progresses, I am sure that we will gain a better

understanding of how some of these hormones (as well as those not in this book) function in relation to metabolism.

Before we dive into the different hormones, I want to discuss hormonal resistance. Perhaps you have heard the term "insulin resistance." Insulin resistance is just one example of hormonal resistance; there are quite a few others mentioned in this book. The human body is constantly seeking out balance, or homeostasis. So in order to provide a system of "checks and balances," the body produces hormones that work with other hormones, as well as hormones that work to oppose, or counter the effects, of other hormones. Using insulin resistance as an example: when insulin is produced too much for too long, it becomes less effective at doing its job. Your body becomes resistant to the effects of insulin. So, in order to prevent this from occurring, it is important to either stop eating things that cause insulin production or eat things that cause glucagon (a hormone that opposes insulin) to be produced. This is an important concept to keep in mind because we need to be conscious of the food eat, since too much of anything can cause a detrimental or negative hormonal effect.

With that said: Lets dive in!

Chapter 3: **INSULIN**

I would like to start out by exploring the role and function that insulin plays in metabolism. Due to the obesity, metabolic syndrome, and type II diabetes prevalence in the United States, insulin is typically given a bad rap. However, there is absolutely nothing inherently bad about insulin. In fact, insulin is extremely important with regards to increasing muscle strength, size, and performance. So for the next section, lets wipe the slate clean with regards to the things you've heard or read about insulin, and take a look at the way it is supposed to function in metabolism.

Insulin is a hormone that is produced by the pancreas. Its main job is to regulate how much glucose (blood sugar) is circulating in the blood at any given time, and "deliver" glucose to cells, specifically muscle and fat cells. Though there are a few different roles that insulin plays in the body's immune system, brain function, and with other organ function, we will focus on its role in muscle and adipose (fat) tissue. After you eat a meal, for example, digested carbohydrates are broken down into sugar that then enters your blood stream. This causes your blood glucose concentration to go up. When the body notices this is happening, it signals part of the pancreas to produce and secrete insulin, which then travels to specific spots or "doors" along muscle cells. These muscle cells need the glucose for fuel. The problem is that glucose can't just flow in and out of muscle cells whenever it wants. There are "doors" that can only be opened by a key (insulin) to let glucose

into the cell. So, insulin acts as a "gate-keeper" to allow glucose to exit the blood and enter muscle cells so it can be used as fuel. Without insulin, it would be very difficult to refill the muscle's gas tank.

So now we can discuss why insulin has such a bad reputation. I'd like for you to think about what happens when you put gas in your car. What happens when the tank is full? The pump should automatically shut off, right? Well, when the muscle's "gas tank" is full, any left over glucose in the blood is sent to adipose tissue and is stored as fat. I like to describe glucose as "high-intensity fuel" and fat as "lower-intensity fuel." The human body can't store very much "high-intensity fuel", so as soon as the tanks are full, the body starts to convert the sugar to "low-intensity fuel."

So, weight gain (from fat) occurs when there is consistent over-load of carbohydrates in the diet with a lack of high-intensity activity to empty the muscle cells' fuel tank. The consistent over-load of carbohydrates causes a huge spike in the concentration of glucose in the blood. The pancreas has to pump out an appropriate amount of insulin to open the fuel tank "door," but the fuel tanks are full, so insulin cannot do its job adequately. The glucose in the blood can not stay there, so it slowly makes its way to adipose tissue, where it goes through chemical processes to be stored as fat. As this occurs more often and over time, the body assumes more insulin is needed to get the sugar out of the blood, so the pancreas produces more insulin. But the issue is not a matter of the amount of insulin. The issue is that there is too much glucose in the blood, which is there because of too much carbohydrate

in the diet or not enough physical activity. This is where insulin resistance comes into play. The body becomes progressively less sensitive to the amount of insulin that is produced. The whole process is kind of like "the boy who cried wolf." The brain says, "there is too much glucose in the blood, we need insulin to get rid of it!" So the pancreas makes more insulin. Then it happens again at the next meal, and the next meal, and so on. In the case of type II diabetes, the pancreas gets so exhausted from pumping out so much insulin that it cannot keep up with the demand signals from the brain, so it begins to makes less insulin. When the body gets to this point, the brain cries "wolf" when the blood glucose level gets to a certain point, but the pancreas is too tired from all the previous "wolf!" cries, so it cannot function to produce enough insulin. Some cases of this situation result in sugar- or diabetic coma. That's one end or extreme of the spectrum, but I want you to know the step-by-step outworking so you can prevent it, and regardless of where you are on the spectrum, begin moving back towards a healthy insulin sensitivity level. Another issue related to this thing called "insulin resistance" is that insulin not only stores fat in adipose tissue, but its presence also prevents other hormones from doing their job of releasing stored fat so it can be used as fuel. So the over-production of insulin not only increases the amount of fat you gain, but it also limits how much you can use!

I do not know where you are along the spectrum of insulin resistance, but I do know that if you are trying to lose fat or trying to increase muscle mass or performance, increasing the body's sensitivity to insulin is a great place to start.

The first thing to do to increase insulin sensitivity is to decrease the degree or amount of glucose (above normal) in the blood, especially following a meal. This is accomplished by decreasing the kinds of food in your diet that cause spikes or significant increases in blood glucose, such as high glycemic carbohydrates and sugars. By reducing the types of food that spike blood sugar, there is less demand on the pancreas to produce insulin. If you do not know where a specific food is rated on the glycemic index chart, go to glycemicindex.com. The University of Sydney set up this website as the "home of the glycemic index," and is a great place to learn more about how different foods affect blood glucose differently.

The next thing to do is to get some exercise. If you get the body moving, and do some high-intensity activity, you will use "high-intensity fuel," and thus empty the muscle's "gas tank." When the glucose "gas tank" is empty, the body has to find fuel from some other source (like fat) and insulin can do its job of opening the tank door to replace high-intensity fuel. Depending on the duration and type of exercise, among a few other things, the body will either begin to tap into stored fat (which is ideal) or it will break down muscle to convert it to high-intensity fuel (not really ideal).

The severity of insulin resistance and intensity of physical activity should dictate the amount, timing, and type of blood glucose spiking carbohydrates in your diet. So, the general rule of thumb when it comes to increasing insulin sensitivity or

decreasing fat-mass is this: the more "sugar" in your diet, the more weight and body composition issues you will have.

There are some things you can supplement or add to your diet to improve the function of insulin. Research has shown that adding cinnamon (approximately 3-6 grams) to your diet every day can decrease the amount of glucose in the blood following a meal as well as decrease fasting levels of insulin.

Although there is not complete consensus on the issue, some research shows that ingesting 2 teaspoons of vinegar (acetic acid) immediately before, or during a carbohydrate rich meal may have antiglycemic (limits the severity of blood spike, and thus the amount of insulin produced) properties, but only when the carbohydrates are complex in nature. In other words, downing a spoonful of vinegar before eating a big pasta dinner may be helpful at reducing the severity of a blood glucose spike, and thus an insulin spike, but the vinegar will not be helpful if ingested right before you pound a candy bar and some soda.

A relatively new face in the supplement industry is magnesium. It has been shown to be an effective supplement, when taken for at least 6 months, at decreasing fasting blood glucose as well as improving insulin sensitivity.

ALA, or alpha-lipoic acid, is an essential fatty acid that functions to reduce oxidative stress and damage that occurs during carbohydrate and glucose

metabolism. As the body converts carbohydrates into usable glucose energy for individual cells, inflammatory byproducts are also produced. ALA works as an antioxidant to neutralize and scavenge these free radicals. Supplementing with ALA has been shown to not only improve insulin sensitivity, but also reduce inflammation in individuals with metabolic syndrome. Some research has even found that supplemental ALA can reduce LDL cholesterol.

Recent breakthroughs in vitamin D research have found it to be a major player in glucose metabolism and insulin resistance. There is a correlation between individuals with vitamin D deficiency and insulin resistance, and it is now understood that many of the pathways that regulate insulin's role in metabolism and immune function are directly controlled by vitamin D. Though more research on vitamin D supplementation is necessary to determine the exact role it plays, vitamin D is becoming a popular addition to many individual's who are trying to improve their body composition and health.

Other foods, or ingredients in specific foods, such as bitter melon (momordica charantia L.,), stevioside, garlic, onion, ginseng, fenugreek, and gymnema sylvestre have been shown to have specific effects on either insulin sensitivity, or on other aspects of metabolic syndrome. One of the major issues with these specific foods is palatability. Bitter melon, specifically, can be a pretty unpleasant thing to have to eat consistently. For this reason, attempts are being

made to put the active ingredients from these foods into other forms that are less stressful on the taste buds.

Though some supplements may help improve insulin resistance for individuals who are diabetic, this does not mean they will be helpful for those who are not diabetic. Chromium, for example, has been used as treatment for insulin resistance in individuals with Diabetes; however, research shows that supplementing chromium in non-obese, non-diabetic individuals found that it actually made their body more resistance to the effects of insulin!

It is now understood that other hormones play a role in how insulin functions. Fixing the levels of adiponectin and leptin, which are typically less than optimal in over-weight individuals, results in a shift towards normal insulin functioning.

One more thing that is extremely important to discuss with insulin sensitivity is the sugar fructose. Fructose, in and of itself, is different from most other types of sugar because it does not cause insulin to be released into the blood stream, nor does it cause leptin to be released from fatty tissue. Due to this specific characteristic, fructose has a much greater "chance" of being stored as fat. Since it does not signal the insulin response, it does not have the chance to be transported to the muscle to be stored as high intensity fuel, and is therefore much more likely to go right to fatty tissue and stored as fat. Ingested fructose is absorbed in the small

intestine, and then transported to the liver. The liver converts it into a form that can be used by the body; however, this usable form is not under the control or influence of insulin or other carbohydrate metabolic enzymes. Without the regulatory role of insulin and other enzymes, fructose increases the amount of very low density lipoprotein (VLDL, a type of LDL cholesterol) in the blood, which in turn, increases the total amount of triglycerides in the blood. When there is an overload of triglycerides in the blood (known as hypertriglyceridemia), the body tries to normalize the triglyceride level by depositing some of the triglycerides into visceral fatty tissue (abdominal fat, or fat that surrounds your internal organs), which leads to an increase in abdominal fat mass. The increase in abdominal fat causes an overload of triglycerides in the liver. This overload of triglycerides in the liver decreases the liver's sensitivity to insulin. When the liver becomes resistant to insulin, the production of VLDL goes up even more. The increased production of VLDL in the blood results in a greater amount of triglycerides being sent to muscle tissue. This increased concentration of total triglycerides causes havoc to normal carbohydrate metabolism and moves the body's metabolism from being locally insulin resistant (liver) to globally insulin resistant (muscle). The consumption of fat has a similar metabolic response in that it does not elicit an insulin response either; however, this should not be an argument for low-fat diets, since specific kinds of fat are necessary for normal body functioning and health. As briefly mentioned above, fructose does not elicit an insulin response, nor does it trigger the leptin response (which will be discussed later).

The other "side" of the insulin coin is the fact that it is considered to be the most "anabolic" of hormones. The reason it is given this title is because in order for muscle growth, increases in strength, or other desired changes within the muscle cells to occur, insulin is still the gate keeper. Muscle growth occurs due to a cause and effect chain where all links in the chain have to be connected in order for the end result to occur. Insulin and the receptors on muscle cells that insulin connects to are links in this metabolic chain, so understanding how to improve insulin sensitivity as well as when to elicit an insulin response is vital for improving body composition, decreasing fat mass, and increasing muscle mass.

Bottom line: To Improve insulin sensitivity:

- **Getting up off the couch and doing some exercise**
- **Reduce the amount of carbohydrates in your diet (especially fructose!)**
- **Increase the amount of cinnamon and vinegar (acetic acid) in your diet (especially before high complex-carbohydrate meals)**
- **Supplement with magnesium**
- **Supplement with ALA**
- **Supplement with vitamin D**

Chapter 4: **ADIPONECTIN**

Adiponectin is a protein hormone that is made by fatty tissue. Ironically though, the greater percentage of body fat you have, the less adiponectin you produce. Due to this inverse relationship, low adiponectin levels are associated with weight-gain, obesity, cardiovascular disease, metabolic syndrome, and diabetes.

So what does adiponectin do exactly? Well, it is responsible for a few things. First, it reduces triglyceride content in muscle tissue. High triglyceride content interferes with the muscle's ability to pull glucose (sugar) into the cell, which results in resistance to insulin. So adiponectin levels directly affect insulin sensitivity, which is directly related to obesity and diabetes. Secondly, adiponectin increases the amount of stored fat that your body can use for fuel. More adiponectin means there are fewer restrictions on what can be used for fuel. To sum it up, adiponectin increases insulin sensitivity, which increases the amount of stored fat you can use for fuel, and therefore improves body composition and muscle efficiency.

How do you increase adiponectin? Well, you "may" do it the "traditional" way. You can go on a calorie restriction diet (that will not work in the long run) and exercise. But…here's the million-dollar question: Do adiponectin levels increase so you lose weight, or does losing weight increase adiponectin levels? The research is not exactly clear. We know there is an increase in adiponectin levels when weight-loss occurs, which is associated with improved insulin function and a reduction in

inflammation. However, some research suggests that there is a minimum necessary threshold that must be lost (5-10% weight-loss) in order for a significant increase in adiponectin levels. Recently, research has been conducted to see if diets that cause weight-loss can affect adiponectin levels. The studies that examined calorie restriction diets for weight-loss showed mixed results for adiponectin level changes. So just reducing the number of calories in your diet may not be enough, and looking at things from this perspective, it appears that adiponectin levels need to increase to lose fat and reduce risk factors, but adiponectin levels can not go up until body weight is reduced! What comes first: Significant weight-loss or significant adiponectin level increases? If you are addressing the issue from this "traditional perspective, I don't know what comes first, but, I can tell you there is a better way to look at this issue. Yes, I've got good news for you.

There are things you can add, and remove, in your diet that will increase adiponectin levels, and thus lead to greater losses in body fat. First, due to the mixed results from the weight-loss diet research, new research looked into whether low-carb and low-fat diets would affect adiponectin levels. The findings from studies like this have shown that diets that are low in carbohydrate content show greater increases in adiponectin levels, especially when compared to diets that are low in fat content. In fact, some research shows that low fat diets can have no effect on adiponectin levels, even when the diet results in 5% body weight-loss. So, low fat diets may actually be counter-productive for increasing adiponectin levels, and thus

less than ideal for maintainable fat-loss and preventing insulin resistance and cardiovascular disease.

What else increases, besides exercise and low-carbohydrate diets, adiponectin levels? Glad you asked. Omega-3 supplementation, fiber supplementation, specific antioxidants, monounsaturated fatty acids, and magnesium supplementation have all been shown to increase adiponectin levels.

Lets talk about Omega-3's. Fish oil and Omega-3 supplements are a huge fad right now, but for good reason. A review of research on Omega-3's and adiponectin found that eating fish daily or taking an Omega-3 supplement daily can increase adiponectin levels 14-60%. If you are not taking a high quality Omega-3 supplement yet, I encourage you to get on it! I am a big fan of Flameout (Biotest) or Functional 03 (Original Nutritionals). Fiber may be the most under-rated supplement on the market. A review of research on fiber supplementation found an increase of 60-115% in adiponectin levels! I hope that fires you up, because I think data like that is really exciting!

Some specific antioxidants, like curcumin, raspberry ketones, and cyanidin 3-glucoside (C3G) have been found to be effective at increasing adiponectin, as well. Raspberry ketones are found in raspberries and in supplement form, and have been show to increase the amount of adiponectin that is produced and released from fat cells. C3G is found in blueberries, and has also been shown to increase the

production and availability of adiponectin. Curcumin is found in turmeric, and can not only reduce inflammation in fat cells, but can also increase adiponectin levels. The body has a hard time absorbing curcumin by itself, but research has shown that combining it with peperine (found in black pepper) or olive oil increases the body's ability to use it. The types of fat typically considered good for the heart (monounsaturated fatty acids), such as those found in avocados, nuts, olive and sesame oils, have been shown to increase adiponectin levels when these fats take the place of saturated fat in the diet. Lastly, increasing dietary magnesium is associated with increases in adiponectin levels. Eating more magnesium-rich foods or magnesium supplementation have both been shown to be effective at increasing adiponectin levels. The Cedars-Sinai Medical Center put together a great resource list of specific foods and their magnesium content. This list can be viewed by Googling "magnesium rich food cedars sinai".

Bottom Line: To Increase adiponectin levels:

- **Consistent Exercise**
- **Reduce the amount of carbohydrates in your diet**
- **Supplement daily with high quality Omega-3 pills or oil (or eat cold-water fish every day)**
- **Supplement daily with high quality fiber (or eat a lot more fiber rich food)**

- **Supplement daily with a high quality antioxidant blend (specifically one containing antioxidants from raspberries and blueberries)**
- **Supplement daily with curcumin (or add turmeric to your food, along with pepperine and olive oil to boost bioavailability)**
- **Increase the amount of monounsaturated fatty acids in your diet (MUFA's)**
- **Supplement daily with high quality magnesium (or eat more magnesium-rich foods).**

Chapter 5: **LEPTIN**

Leptin is a hormone that is produced in adipose (fat) tissue. It's main job is to tell the brain when you are full, or satiated. Looked at from a slightly different perspective, leptin keeps the brain informed on how much fuel is in the tank. In a normal functioning person, leptin levels are high during periods of "over-eating". This increase in leptin production signals the brain to increase the metabolic rate. During a meal or period of "plenty," leptin is produced and signals the brain that energy stores are full, so intake should stop and metabolism should increase. Conversely, during periods of dieting and calorie restriction, leptin levels decrease. A week of dieting, for example, can decrease leptin levels by as much as 50%. One reason diets are unsuccessful can be linked to this reduction in leptin, since the "satisfied" feeling that leptin is responsible for providing becomes weak during calorie restriction. If you are counting calories to lose weight, you probably do not feel full or satisfied after you eat, and that is a problem.

The strange thing about leptin is that the concentration in your body is highly correlated to the amount of fat mass you have. So, in a not so politically correct explanation: the "fatter" you are, the more leptin you produce, with a few exceptions. The amount of leptin that the body produces is also directly dependent on the amount of insulin that is infused into the blood. Research has shown that individuals who are obese may have up to 4 times the amount of leptin than a person who is considered lean, though in obese individuals, leptin seems to be less

successful at executing its function of telling the brain the gas tank is full. Recent study on hyperiemia (where the body produces and secretes excess insulin, explained previously in this book using the "boy who cried wolf" analogy) shows that this excess of insulin actually prevents leptin from acting out its normal role. This phenomenon has been described as leptin Resistance (similar to insulin Resistance).

The main issue with leptin resistance is highly connected to insulin resistance, since leptin production is directly related to insulin production and secretion. When an individual has some degree of insulin resistance, they will also have some degree of leptin resistance. Therefore, fixing leptin resistance may parallel fixing insulin resistance. The previous section on insulin gives examples of ways to improve insulin resistance, and these should also play a role in correcting leptin resistance. Some specific ways to improve body composition, lose fat, and increase leptin sensitivity deal with fructose and its effects on other hormones. Since fructose does not cause an insulin response, and therefore no leptin response, it may be "responsible" for increased risk of over-eating because the "full" signal is slowed or not sent to the brain. Ingesting fructose may be preventing you from correcting both insulin and leptin sensitivity levels. Due to this, it may be very beneficial to steer clear from food that is high in fructose (like soda and junk food). There has been some debate, especially within the past couple years, as to whether fruit should be avoided since fructose is the type of sugar found in fruit. The amount of fructose found in fruit typically pales in comparison to the amount of high

fructose corn syrup you would find in soda or candy. The specific vitamins, minerals, antioxidants, and phytonutrients found in fruit should not be avoided just because of a little fructose. I do not think fruit should be avoided, but if you are really worried about the amount of fructose that some types of fruit contain, then eat the fruit either right before or right after you work out, and that sugar will be used as fuel.

Another very important aspect of male metabolism that leptin is involed in is the endorcrine pathway of testicular steroidogenesis. High levels of leptin have been shown to block the normal production of testosterone in men, and as previously mentioned; overweight individuals typically have up to 4 fold greater production of leptin due to insulin/leptin resistance. The prevention of normal testosterone and Androgen production not only limit the growth and repair of damaged tissue, but also limit the amount of stored fat that can be used for fuel. For this reason, resistance training should be a part of a healthy lifestyle due to the positive effects it has on anabolic hormone production.

Since leptin was first "discovered" in 1994, research on whether specific foods or ingredients affecting leptin functioning have been limited, though it is growing. One specific ingredient (IGOB131) from the seed of a West African plant, Irvingia Gabonensis (also called African Mango), has been found to improve the function of both leptin and adiponectin in overweight humans. This ingredient was also responsible for other beneficial changes in metabolism, such as reductions in

body fat and decreases in plasma total cholesterol, LDL cholesterol, blood glucose, and C-reactive protein (a marker of inflammation). IGOB131 extract is now a commercially available in pill or drop forms. Another supplement providing potential benefits to metabolic syndrome and leptin resistance is Acetyl-L-Carnitine (ALCAR). Though there have been numerous animal studies on ALCAR supplementation, there have been few human studies that show any legitimate benefit. It seems as though ALCAR supplementation may improve leptin sensitivity, but only when there is a significant deficiency in normal ALCAR levels.

There are other ways to improve leptin sensitivity. First, reduce inflammation. If you have some degree of insulin and leptin resistance, then you have some chronic inflammation, too. The best ways to decrease inflammation are to increase the amount of Omega-3's in your diet, decrease the amount of carbohydrates in your diet (especially those simple carbs), and increase fruit and vegetable intake. Another important factor that is often overlooked is the amount of sleep an individual gets. Research is clear that both acute and chronic sleep deprivation affects leptin production and sensitivity. Getting 7-8 hours of sleep every night is a must to keep inflammation and inflammatory processes under control. Recent research has shown that increasing the amount of calcium in the diet may correct leptin resistance, though the exact mechanism of why this occurs is still unknown. Supplementing with taurine has also been shown to be beneficial at improving leptin resistance as well as other symptoms of metabolic syndrome. For individuals with above normal levels of leptin production, or leptin resistance,

research has shown that high-intensity and long-duration resistance training may have the effect of reducing the total leptin production, and thus aiding in weight loss and correcting leptin resistance. The caveat to this type of high-volume training (some studies report the need for 50 total sets per workout) is that the reduction in leptin levels is delayed, so this type of training has to be consistent in order for the effects to be significant.

Bottom Line: To Improve leptin (and perhaps insulin) sensitivity:

- **Remove, or at least limiting, the amount of fructose in your diet**
- **Decrease the amount of simple carbohydrates in your diet**
- **Supplement with IGOB131 extract**
- **Supplement with high quality Omega-3's**
- **Supplement with calcium**
- **Supplement with taurine**
- **Get plenty of sleep every night (7-9 hours)**
- **Resistance train consistently and vigorously!**

Chapter 6: **GHRELIN**

If leptin tells your brain that you are "full," then ghrelin tells your brain that you are hungry. It has other duties, including the stimulation of growth hormone, signaling the need for new fat cells, increasing triaglycerol formation, and decreasing lipolysis. This hormone is produced mainly in the stomach, and though it has been previously studied because of its appetite-stimulating effects, it is now being studied for its role in the regulation and control of glucose in the blood, since it is now understood that it is also produced in the pancreas. In general, the concentration of ghrelin in the blood increases until food is ingested. After feeding, the concentration drops, but then begins to rise again until the next feeding. Ghrelin produced in the stomach enters the blood and increases in concentration until the brain gets the "I'm hungry" signal. What do you do when you are hungry? You eat! Ingesting food, carbohydrates specifically, elicit the insulin response, which causes the removal of ghrelin (and thus the "hunger pangs"). The thing about calorie restriction (aka…dieting) is that it causes a chronic increase in ghrelin levels, so you are always hungry. If you are always hungry, then losing weight, and more importantly, maintaining a specific weight, become more and more difficult. This is perhaps another big reason weight loss diets are so unsuccessful. No one wants to feel hungry all the time. How do you deal with, or work around, this hunger signal?

Well, first of all, I do not want you to think that the hunger signal is bad. Your body is letting you know it needs something when you get the ghrelin signal. I do

not think the goal is to "give in" to the signal, I think the goal should be to proactively control the signal. How is this accomplished? Specific meal timing and intense exercise, I believe, are the answer. There has been some research on the effects of specific supplements, such as oligofructose, on ghrelin levels. Though the outcome for this supplement was very desirable (weight loss due to significant reduction in ghrelin levels with a significant increase in the hunger-suppressing hormone, PYY, as well as improved glucose regulation), subjects reported on the final day of testing that almost 50% of the time they were supplementing, they experienced some sort of gastrointestinal discomfort, including flatulence and intestinal pressure. These subjects who reported these negative side effects also said that they would not continue the supplement dose because of the discomfort.

What are the other options? There has been other research looking into the composition as well as timing of feeding on ghrelin levels. One specific study sought to find the effects of a protein-carbohydrate rich breakfast or an ultra low-carbohydrate diet on ghrelin levels and weight-loss. This study came about due to both scientific proof and anecdotal evidence that significant portions of protein at breakfast can be beneficial at controlling hunger, and adding complex carbohydrates to protein at this meal may be even more helpful at controlling hunger. This study showed that after 16 weeks both groups had a significant reduction in both body weight and ghrelin levels. They retested again after another 16 weeks, but do to some study design flaws and compliance issues, the data from the retest is less clear. What is clear is that both groups had beneficial reductions in both body weight and

ghrelin levels, and both groups were limited to less than 100 grams/day of carbohydrates. The authors of this study suggest that beginning the day with a high protein and complex carbohydrate meal (within a high protein/low carb diet) will assist in long-term weight-loss and prevent weight gain that often comes after weight loss.

Research is also quite clear that exercise duration and intensity can play a role in the production of ghrelin. Studies on different types of exercise as well as differing exercise intensity show that the specific type of exercise is not as important as the consistency and specific intensity of exercise. In other words, a single bout of low intensity exercise, like walking on a treadmill, using an elliptical machine or even low intensity resistance training, will not decrease ghrelin levels. In fact, research shows that low intensity exercise may have no effect on ghrelin levels, or may increase the level of ghrelin. A single bout of intense aerobic exercise (like running on a treadmill for 60 minutes) or intense resistance training (90 minutes of weight lifting) can cause an immediate decrease in ghrelin levels, but only for about 2 hours. If this type of training becomes consistent, then total ghrelin levels will actually increase, but only when weight loss occurs from the exercise. The overarching conclusion from research on exercise and ghrelin is that total ghrelin levels may increase as an individual loses weight due to exercise, but the active form of ghrelin (acylated ghrelin) stays the same. When body weight is lost due to calorie restriction alone, not only do total ghrelin levels increase, but the specific amount of active ghrelin also increases.

The amount of sleep you get also affects ghrelin levels. Sleep deprivation is known to increase ghrelin levels. A specific study, by Spiegel *et al.* showed that sleep deprivation not only caused significant increases in ghrelin and thus appetite, but there was a trend for study participants to crave calorie-rich carbohydrate foods. It appears that this issue may be cyclical in that high levels of ghrelin can contribute to shorter amounts of REM sleep, which cause increases in ghrelin production. Conversely, research also shows that improved sleep patterns lead to decreases in ghrelin levels.

Bottom line: To normalize ghrelin levels:

- **Stop dieting!**
- **Eat a protein and complex-carbohydrate rich breakfast**
- **Exercise consistently and vigorously**
- **Get plenty of sleep every night.**

Chapter 7: **GLUCAGON**

If insulin were a chess player, then glucagon would be its opponent. Glucagon is called the insulin antagonist due to its blood glucose regulatory role. Since most people who eat the "Standard American Diet" (SAD) have extremely active insulin production, the role and function of glucagon may be blunted or limited. When blood glucose levels increase to a specific threshold, insulin is released into the blood to stimulate the removal of the glucose out of the blood and into muscle or fat cells. Glucagon, conversely, is released into the blood when glucose levels dip below a specific threshold. Glucagon acts to break down glycogen (the stored form of glucose) in the liver so that blood glucose levels can return to normal. During exercise or fasting, when muscles are damaged or when muscle proteins are broken down into amino acids by cortisol in order for the body to make its own glucose, glucagon works to deliver these amino acids to the liver so new glucose can be made. These are considered to be the "immediate glucose regulatory roles" of glucagon. Now we can dive into the other ways it can affect metabolism.

Since glucagon serves to oppose insulin, and since insulin serves to store glucose and fat in both muscle and fat tissue, glucagon should release glucose and fat in muscle and fat tissue, right? Well, it is very good at releasing glucose in muscle tissue, but its exact role on releasing fatty acids from adipose tissue (a process called lipolysis) has been less clear. Research conducted in the early 2000's showed that glucagon (by itself) was quite ineffective at increasing lipolysis. More recent data

shows that although glucagon may not directly increase fat breakdown, its presence and availability may prevent or counter some of the fat storing effects of insulin, and thereby indirectly increase the availability of fat to be released from storage. The term glucagon-stimulated lipolysis (GSL) has been thrown around in the research realm since the discovery of glucagon related fat-loss mechanisms. The major mechanism of GSL comes when glucagon is produced and secreted to a much greater and more consistent extent.

Though the immediate effects of glucagon on the liver elicit glucose release and the production of new glucose from amino acids, the consistent presence of glucagon at the liver causes a shift in the type of fuel the body sees as consistently available. In other words, if you eat "a lot" of carbohydrates, then the body assumes that there will be a consistent supply of this "high intensity fuel," so there is no need to utilize very much "low intensity fuel" (fat) that is stored. However, when the amount of carbohydrates in the diet is consistently limited, the body begins to recognize that it should save as much glucose as possible and increase the amount of stored fuel it uses for everything else. When this principle is taken to the "extreme," like very-low carbohydrate diets, the body's fuel source can shift even further to ketones.

Research just published at the end of 2012 showed that glucagon stimulates the production of Fibroblast Growth Factor 21 (FGF21), which is responsible for improving metabolic functioning in type II diabetics as well as obese individuals by

increasing energy expenditure, improving glucose and fat metabolism by correcting insulin sensitivity, and reducing body weight. Though it is involved in all of these metabolic functions, it is now understood that elevated levels of FGF21 are typically found in individuals who have some degree of insulin-resistance or are obese. It appears that FGF21 is over produced in a physiological state of insulin-resistance, but it seems to be unable to execute its function in this state (FGF21 resistant state). Limiting the insulin response not only has the effect on all the previously insulin-related metabolic responses, but it also allows glucagon to have its effect of increasing the lipolytic effects of normal FGF21.

How do you increase glucagon production to combat insulin and increase GSL? The first thing is to increase dietary protein intake. Research shows that consistent dietary protein intake of about 1.5-2.0 grams of protein per kilogram of body weight is sufficient enough to increase fasting plasma glucagon concentration by greater than 30%. This amount of dietary protein has also been associated with significant improvements in insulin sensitivity and glucose metabolism. The summation of all the protein/glucagon research comes down to this: there is a linear relationship between glucagon production and the concentration of amino acids in the blood. To significantly increase the amino acid concentration in your blood, you have a two options: Fast, or eat more protein. I find that most people struggle with fasting, so I will recommend ramping up your protein intake. The other way to increase glucagon production is via exercise. High intensity exercise, especially exhaustive aerobic exercise, causes an increase in plasma glucagon

concentration. It is now understood that this increase is not only to provide fuel to maintain exercise intensity, but it also plays a role in the process of eliminating nitrogen from the body via the urea cycle. Research also shows that exercise also plays a role in increasing the body's sensitivity to glucagon by increasing or changing the characteristics of the receptors that glucagon binds to. In this way, the physiological "influence" of glucagon can increase without the need for more glucagon to be produced.

Bottom Line: To increase glucagon concentrations in the blood:

- **Eat more protein**
- **Eat fewer simple carbohydrates**
- **Exercise intensely**

Chapter 8: **CHOLECYSTOKININ (CCK)**

CCK is a hormone that is produced in the duodenum (intestinal wall) as well as the brain. It also plays a role in satiety (feeling of fullness). Unlike leptin, which has a longer effect on satiety, CCK is more of an immediate signal to the brain. When CCK is released, the signal travels quickly to the brain to deliver the message: "I am full right now." One way it functions to do this is by slowing down gastric emptying (delays the movement of food from the stomach into the intestines). CCK also functions to stimulate the liver to produce bile, and the gall bladder to release the bile. This is a very important step in digestion because bile acts like a detergent in that it makes fat molecules smaller and more easily to digest. CCK also acts on the pancreas to produce and release pancreatic fluid and pancreatic enzymes that are necessary for normal digestion. CCK is produced when fat and protein enter the stomach or intestine. Let me be clear: Carbohydrates do not elicit a CCK response. In fact, high levels of glucose in the blood elicit an increase in the hormone somatostatin, which can actually work to block the production of CCK. In other words: Lots of carbs in the diet means less than optimal "I'm full" signal when you eat.

There exists a correlation between individuals who are considered very obese and lower than normal levels of CCK. Though there is no definitive answer behind this mechanism, I theorize it is related to the level of carbohydrates in the diet as well as the level of insulin and leptin resistance. Research related to the

issue has looked into administering CCK injections during meals to see the effects on the sensation of fullness as well as the amount of food eaten. Some studies have examined the effects of CCK administration with a high-carbohydrate or carbohydrate only meal. The results indicate that even when a meal composed of carbohydrates only, a physiological dose of CCK results in an early onset feeling of fullness as well as a reduction in the total food consumed. Other studies have examined the effects of CCK administration on meal satiety. Across the board, when CCK is administered in conjunction with a meal, regardless of what the meal type is, the subjects eat less food. The issue with potential CCK therapy is that it is very unpractical to get a CCK infusion or shot every time you eat. What is the alternative? As previously mentioned, protein and fat are responsible for CCK production and secretion. Instead of waiting for pharmaceutical companies to try to develop a drug that replicates the role of CCK, just eat more protein and fat. By the way (and this is a bit of a tangent, but please bear with me, as I feel this is very important), there exists no legitimate research or data that shows that fat, even saturated fat, is inherently bad for you or causes heart disease.

The low-fat diet "ideal" and craze that we have been told is the best way to live, in my opinion, has been the biggest variable responsible for the current obesity-epidemic and inflammatory-disease state in America. Fat does not make you fat; plain and simple. I am not crazy or alone in this position, either. Plenty of doctors, physiologists, nutritionists, and dieticians agree that dietary fat is not the enemy or demon it has been made out to be. The issue of saturated fat causing

problems comes when this type of fat is part of the Standard American Diet, or a high-carbohydrate diet. In this type of diet, ingested fat is not efficiently used, so forget about it being used as fuel. On a low-carbohydrate diet, however, research shows that ingested saturated fat is used quite readily as fuel, and there are few, if any, negative health effects. So, all that to say, do not be afraid to eat fat, unless it is hydrogenated or trans-fat. Back to CCK.

Eating more protein and fat and decreasing carbohydrates will increase CCK production, and lead to improved sensation of fullness while reducing the amount of food ingested. For individuals afraid of "over-eating," this is a big deal! Brand new data shows that CCK plays a vital role in glucose metabolism, and actually has the effect of stopping the production of glucose in the liver. This occurs via a "gut-brain-liver" neuronal axis. This is great news for those who are seeking to reduce the over-production of insulin (hyperinsulinemia) as well as those who just want to improve glucose metabolism and insulin sensitivity. The problem is that this signal to stop the liver from producing glucose is blocked in individuals who are on a "high-fat induced insulin-resistant diet." The thing about this type of diet is that the insulin resistance associated comes from the accumulation of bad fats, or too many carbohydrates. A high-fat diet will not cause insulin resistance unless the fat in the diet is from hydrogenated and trans-fat sources, like fried foods and margarine. So if you are eating a diet high in bad fat, you probably have some degree insulin resistance, and your CCK signal to correct blood glucose levels probably does not work.

Bottom line: To increase CCK production:

- **Increase the protein and fat content in meals**
- **Decrease the carbohydrate and bad fat (trans-fat and hydrogenated fat) content in meals.**

Chapter 9: **PEPTIDE TYROSINE TYROSINE (PEPTIDE YY or just PYY)**

Peptide YY is yet another hormone involved in satiety and the feeling of fullness when eating. It is produced and secreted from the small intestine, though it does its job when it travels through the blood and binds to specific receptor sites on the brain. When food, especially protein, and to a slightly lesser extent, fat, make their way through the stomach and into the small intestine, Peptide YY is produced and put into circulation. When PYY binds to its receptors on the brain, the fullness signal is flipped like a light switch. This specific mechanism takes place in the hypothalamus, at the receptor sites of neuropeptide Y (NPY). NPY and PYY oppose each other in the NPY stimulates food intake via hunger signals, while PYY stimulates satiety signals. When PYY binds to its receptor site, it not only intiates the satiety signal, it also suppresses NPY neurons and thus the hunger signal. PYY also works to slow down the movement of food through the digestive tract, specifically in the ileum, where the small intestine connects to large intestine, and thus indirectly assists in satiety.

The total amount of PYY that is produced and secreted depends on the amount and caloric density of the food eaten. In other words, the more food that is ingested, and the more calories contained in the ingested food, means more PYY production. This is not the only variable associated with PYY production, though. The production and secretion of bile contributes to PYY production, as does the production and secretion of CCK. The important thing to note with these two

variables (bile and CCK) is their production is heavily influenced by protein and fat, and less influenced by carbohydrates. This serves as important information for those who restrict caloric intake as well as those who eat the Standard American Diet, since the amount of food as well as the specific composition of food (Ratio of carbs, protein, and fat) directly influences PYY production and thus the feeling of fullness, or lack thereof.

It is now understood that a high-fat diet has the most influence on the initial release of PPY into circulation, while the effect of protein in the diet causes a delayed release of PYY into the blood about 2 hours after ingestion. A study comparing the post-meal production of PYY in obese individuals who ate either a high-carb/low-fat diet or a high-fat/low-carb diet showed that after just 7 days the high-carb/low-fat diet was significantly less effective at eliciting PYY production compared to the high-fat/low carbohydrate diet. The high-fat/low-carb diet resulted in PYY secretion 55% higher than the low-fat/high-carb diet. This serves as more evidence that the "LOW-FAT" message constantly thrown at us is not ideal for fat loss or weight maintenance.

Though it is very rare to have a genetic deficiency of this hormone, it has been established that individuals who are considered obese as well as those who are considered pre-diabetic (type II) have lower than normal levels of PYY production. Again, this is most likely due to the specific composition of an individuals diet (amount of carbohydrates) and potential PYY resistance. If PYY, CCK and leptin

were in a race to see who would suppress appetite first, CCK would be the winner, followed by PYY, and then leptin. PYY thus works as more an intermediary "fullness" or "satisfied" signal from the end of a meal to an hour or two post feeding, though the effects may persist longer in some cases.

Research on PYY infusion shows across the board that there is a physiological signal that reduces the amount of food ingested. Multiple studies comparing how much people eat *ad libitum* (at one's pleasure, or at your own discretion) at a buffet after either a PYY infusion or a saline solution infusion (placebo) showed the CCK results in ~30% decrease in the amount of food ingested at that meal, as well as ~30% reduction in the food consumed in the following 24 hours. That sounds awesome, right? We should all just get an infusion of PYY before we eat. Unfortunately, the situation is not that simple. The issue is that many of the subjects in the PYY infusion studies experienced nausea to the extent that they were unable to finish an entire infusion (the infusion took 90 minutes).

Other modes of PYY delivery have been studied, such as a intra-nasal form, but this, too, caused many subjects some level of nausea, and many were unable to complete the study. PYY, by itself, in an oral form seems unable to be absorbed across the intestinal wall, and has thus been an unsuccessful therapeutic modality. When PYY was combined with sodium N-caprylate in an oral form, however, the concentration of PYY in the blood reached levels greater than normal PYY production. This increase in plasma PYY was in conjunction with a 12% reduction

in the amount of food ingested 15 minutes after PYY/sodium N-caprylate administration when compared to the placebo. This oral form of PYY had no long-term effect, however, as the amount of food ingested in the next 24 hours remained unchanged.

There does seem to be potential for PYY administration as a therapeutic modality for obesity in the future, but it is not here yet, so do not wait around for it. Be proactive! Since protein and fat are primary stimulators of PYY production and secretion, eat more protein and fat. Since carbohydrates do not stimulate PYY production and secretion, eat fewer carbohydrates. Seams simple, right? What else can you do? Exercise is now known to have a significant effect on PYY production. Though PYY typically increases during all modes of exercise, long-term, moderate intensity aerobic exercise, as opposed to other forms of training (Sprint intervals or resistance training), seems to have the most impact on increasing total PYY levels and thus satiety.

Bottom Line: To reduce NPY (hunger) signaling and increasing PYY signaling:

- **Increase the amount or protein and fat in your diet**
- **Decrease the amount of carbohydrates in your diet**
- **Exercise consistently, especially aerobic exercise.**

Chapter 10: **GLUCAGON-LIKE PEPTIDE-1 (GLP-1)**

GLP-1 is a hormone that is part of the incretin family, which is responsible for increasing the production and secretion of insulin. GLP-1 serves other functions as well. It is made from the molecule preproglucagon, which when broken down, also creates glucagon. Due to this shared "parent" molecule, GLP-1 has similar qualities and functions as glucagon, which is why it is called "glucagon-like." The L-cells of the small intestine are the main producers of GLP-1, though it is also produced, to small degrees, within the central nervous system as well as the pancreas. It functions to oppose glucagon by promoting insulin secretion, by increasing the volume of the cells of the pancreas that produce insulin, and by preventing glucagon release. Due to its inherent connection to insulin, GLP-1 levels are related to glucose metabolism and insulin sensitivity.

Though the exact reason is still unknown, obese individuals may have lower than normal production and secretion of GLP-1 after a meal. This is likely due to the composition of the diet and the effects of the Standard American Diet on glucose metabolism and insulin function.

The GLP-1 that is produced in the central nervous system works on the appetite centers of the brain to elicit the feeling of fullness and by slowing down gastric emptying. Both human and animal studies have shown a dose-dependent relationship between intravenous GLP-1 administration prior to a meal and a

reduction in food and liquid ingested, with an associated feeling of fullness. However, once again, in order for this to be of therapeutic benefit, it has to be injected prior to each meal.

Since GLP-1 is plays a significant role in glucose metabolism as well as hunger satiation, research has recently been conducted to figure out the exact mechanism of GLP-1 production and secretion. Research in the mid 1990's showed that significant GLP-1 production occurred in conjunction with food intake. From the studies conducted since then, we now know that GLP-1 is produced in a nutrient-dependent fashion. GLP-1 works in conjunction with PYY to "stop" digestion via the "ileal brake" when unabsorbed nutrients have made their way to the ileum. In doing this, GLP-1 works as a potent "full" signal within the gut by delaying gastric emptying and by slowing gastrointestinal function. It is understood that fat, protein, and carbohydrates (Glucose and sucrose, specifically) elicit a GLP-1 production and secretion response; however, it appears that oleic acid (a type of fatty acid that occurs naturally in high amounts in animal and plant source fats) has a specific role in GLP-1 production. Oleic acid, which is a very common mono-unsaturated fatty acid, has a very direct effect on the cells that produce GLP-1. Research in this area has shown that oleic acid, as well as other free fatty acids, increases GLP-1 production. The exact mechanism are still not fully understood, though it may have something to do with the effects of free fatty acids on uncoupling-protein 2 (UCP2), and the regulatory role that UCP2 has on GLP-1 secretion within the gut. From this data we can conclude that although most food

types elicit some GLP-1 response, fatty acids, even saturated fatty acids, have the most profound effect on GLP-1 production and secretion.

There is some debate over whether increasing oleic acid and other fatty acids in the diet will have negative effects on GLP-1 production and function down the road. This issue deals with chronically high levels of fatty acids increasing oxidative stress as well as causing an increase in uncoupling-protein 2 (UCP2), and this increase in UCP2 negatively affecting the pancreas (especially the parts that produce insulin and glucagon). Research dealing with this theory seems to point to the increase in UCP2 potentially associated with a high-fat diet is due to insulin resistance more than the amount of free fatty acids in circulation. In other words, even if you go on a high-fat diet that causes chronically elevated levels of free fatty acids and cholesterol (Hyperlipidemia), if you are doing things to improve your insulin sensitivity and decreasing oxidative stress, there is little to no risk of UCP2 induced damage to the pancreas, or anything else for that matter. I should also mention there is a multitude of pharmaceutical drugs and GLP-1 receptor agonists that have been used, and are currently used, to increase the production or the function of GLP-1, including Acarbose, Exenatide, Liraglutide, and Metformin.

Bottom line: To increase GLP-1 production:

- **Increasie the amount of fat in your diet, especially unsaturated fatty acids like oleic acid,**
- **Improve insulin sensitivity**

- **Reduce oxidative stress (eat fruits and veggies, exercise consistently, and get plenty of sleep).**

Chapter 11: **CORTISOL**

Cortisol is a member of the glucocorticoid family of steroid hormones and is produced by in the adrenal cortex of the adrenal glands. This is not the type of steroid athletes get in trouble for using. Cortisol is quite different from anabolic steroids, because unlike anabolic steroids, cortisol is responsible for breaking down protein. Cortisol functions to break down body tissue protein (especially muscle protein) into amino acids so they can be transported to the liver where they go through a process to be converted into sugar (gluconeogenesis). This occurs to provide the body with its preferred energy source to combat stress. For this very reason, it is called the "stress hormone." Acutely, this process is good for you. In fact, acutely cortisol is also lipolytic (breaks stored fat down to be used for fuel). The problem comes up when cortisol release becomes chronic, due to chronic stress.

Since cortisol is produced and released to help the body combat stress, it is considered a very big part of the "fight or flight" response. Again, cortisol serves to break down protein structures into amino acids to convert them to sugar under stressful situations. It is responsible for providing high-intensity fuel when the tank is empty, or getting low. But, in today's world, there seem to be more and more stressors than previously. The body does not know the difference between stress that comes about from running away from a rabid dog and the stress that comes from not enough sleep, fatigue, hunger, and poor dietary habits. Stress is stress, and

the body produces cortisol when it is stressed. Here is the main point of this chapter: The cortisol "switch" is not supposed to be on all the time. A chronic release of cortisol is like red-lining your engine every time you drive. You may not blow your physiological engine, but it will have lasting effects on your metabolism.

When the body senses stress (physiological or psychological), the hypothalamus secretes corticotrophin-releasing hormone (CRH), which then stimulates the anterior pituitary gland to release adrenocorticotrophic hormone (ACTH), which in turn, causes a release of glucocorticoids, mineralcorticoids, and androgenic steroids from the adrenal cortex. Cortisol is the main glucocorticoid released in this domino effect cascade of the hypothalamic-pituitary-adrenal (HPA) axis. When this cascade happens consistently, the "fight or flight" role that cortisol plays is replaced with some pretty unhealthy side effects. There have been numerous studies on the effects of sleep, or sleep deprivation, and the stress response. Research has shown us that cortisol that is produced in response to chronic stress causes increases in fat mass, the breakdown of stored glucose, reductions in muscle mass and bone tissue, suppression of the immune system, while increasing insulin resistance. When you add all these effects of chronic stress-induced cortisol production, the end result is a big shift towards obesity, diabetes, cardiovascular disease, neurological disorders, as well as many other detrimental health effects.

Normal cortisol production and release works on a negative-feedback loop, where the presence of cortisol inhibits the production and release of more CRH and ACTH, so that more cortisol is not produced. This is important since some studies show that obese individual have lower plasma concentrations of cortisol than non-obese individuals. However, some data shows that there is a high correlation between obesity and levels of CRH, ACTH, and other glucocorticoid metabolites (An intermediate product of metabolism; in this case, the broken down products of cortisol and other glucocorticoids). Research has also shown that ACTH and CRH injections, as well as mental stress test increase cortisol levels more in obese individuals than in non-obese individuals. From this we can conclude that increased fat mass results in a "hyper-responsive" HPA-axis to stress that is simultaneously less responsive to the negative-feedback loop. So generally speaking, the more fat mass you have, the easier it becomes to produce cortisol in response to stress, and the more difficult it becomes to shut off the "cortisol" switch. This does not happen over-night, and there many variables involved stress-related weight gain.

Since cortisol increases insulin resistance, research has shown there to be a direct relationship between stress and cortisol-induced triglyceride accumulation in the liver, which leads to increases in abdominal fat and further insulin resistance. Research published in 2011 explored the relationship between stress and food cravings. In some individuals, an over-active HPA-axis, or even acute stress, can influence the central reward centers in the brain as well as appetite. Research in

this area show that increased HPA-axis activity may result in individuals craving low-quality food and/or "comfort food" (food that is typically very high on the glycemic index, high in calories, and not very nutrient dense). An interesting thing to note is that HPA-axis activity decreases when an individual experiences long-term weight loss, which shows that the effects of obesity on HPA-axis activity and the effects of HPA-axis activity on increases in fat mass and obesity can be reversed, to certain degrees. Though this is the case in many, if not most, people, there are some individuals who lose weight during periods of chronic stress. This is thought to occur due to individual differences in coping mechanisms, dietary choices, as well as changes in "fat-burning" metabolism.

Cortisol also plays a role in immune function. Chronic stress not only causes an increase in the HPA-axis, it also increases the size of the adrenal organs so they can produce more cortisol. Another associated part of chronic stress is a reduction in the size of lymphatic organs, which play a major role in immune function. Enlarged adrenal glands produce more cortisol, and reduced lymphatic organs produce fewer white blood cells to combat infection. Chronically elevated levels of cortisol has been shown to not only reduce the production of white blood cells, but it has also been shown to inhibit the role of existing immune cells as well as the body's natural antioxidants. For this reason, chronic stress and chronic cortisol production is highly correlated to increases in infections and a suppressed immune state.

Exercise is also known to have distinctive effects on cortisol production. In general, all types of physical activity cause an acute increase in cortisol production to stimulate an inflammatory response and tissue remodeling. Due to this, cortisol is a very necessary hormone involved in increases in muscle size and strength due to its direct connection to the inflammatory response (which will be discussed in the chapter on growth hormone) as well as its role in remodeling skeletal muscle tissue. High-intensity resistance training is known to cause a significant increase in cortisol production, though it is typically matched with an increase in growth hormone. Aerobic exercise, especially running, of moderate to high intensity (consistent endurance training greater than 50% of VO2 max) is known to cause an increase in cortisol production more so than consistent resistance training. This is most likely due to the body preferring glucose production from broken down protein as opposed to stored fat. For this reason, it is very important for those who are chronically stressed as well as those who train and participate in endurance training to understand the role of cortisol in metabolism and immune function, and apply dietary and lifestyle interventions to combat the over-production and negative side effects of chronic cortisol production.

There are many ways to combat chronic stress, and thus the chronic production of cortisol. Since sleep deprivation is one of the major causes of stress-induced cortisol production, address this first. Be sure you getting plenty of restful sleep. Some studies looking at only addressing sleep time have shown that increasing your sleep time over 6 hours is associated with less fat gain. Increasing

the amount of time you sleep results in a reduction of HPA-axis activity and thus cortisol production.

Nutrient support seems to be the best line of defense during periods of chronic stress. Hyperactivity of the HPA-axis, but the adrenal glands specifically, seems to deplete vitamins B5 and B6, as well as vitamin C. Stress may have the effect of dumping these specific vitamins and result in less than optimal functioning of the adrenal glands and the hormones produced there.

The specific mechanisms of cortisol feedback are directly dependent on magnesium, zinc, manganese, and calcium, and indirectly dependent on the supporting agents L-theanine and L-tyrosine. Due to this, HPA-axis and cortisol functioning are enhanced when these specific nutrients are enhanced in the diet or supplemented.

Magnolia (magnolia officinalis) supplementation has been shown to reduce stress and "induce" restful sleep, while aiding in relaxation and improving mood, though in most studies, the exact amount of cortisol was not reduced by a significant amount. A proprietary blend of magnolia officinalis and phellodendron amurense (Relora ®) has been shown to assist in achieving normal cortisol and DHEA levels, and may therefore aid in satiety signaling and fat metabolism.

Though a definitive answer has yet to be reached, these exists some data that supports the theory that a diet high in Omega-3 fatty acids, or supplementation of Omega-3's, may reduce systemic inflammation and thus reduce the amount of physiological stress markers, and thereby limiting the total HPA-axis stress response. This has been a very popular research topic within the exercise and sporting world, though at this point in time, there is still conflicting data.

In relation to chronic exercise-induced stress and cortisol production, there exist many dietary interventions to prevent both cortisol release to spare muscle breakdown as well as suppression of the immune system. Carbohydrate ingested before, during, and after high-intensity exercise has shown to be beneficial at preventing the degree of exercise-induced immune suppression and limiting the amount of cortisol release during exercise. Glutamine, an amino acid, is a major player in the immune function as a potential fuel source for specific immune cells; however, specific research studies, as well as reviews of literature, on glutamine supplementation and immune function shows little, if any, evidence that it is helpful at preventing exercise-induced immune suppression or cortisol release. Branched-chain Amino Acids (BCAA's) supplementation before and after exercise, on the other hand, have been shown to be very beneficial at preventing exercise-induced muscle damage, increasing muscle protein synthesis, reducing cortisol production and the inflammatory response, and protecting and regulating the immune response.

The phospholipid Phosphatidylserine (PtdSer), plays a role in cell membrane function, and when supplemented orally (100 to 500 mg/day), has been shown to improve cognitive function. However, when orally administered at a dosage of ~800 mg/day, exercise-induced changes in HPA-axis moderated, and there is a reduction in the amount of exercise-induced cortisol. This reduction in exercise-induced cortisol production may account for reductions in muscle soreness and increased feelings of wellbeing, which were reported by the study participants.

Cortitrol®, is a dietary supplement that claims to help individuals stay relaxed and calm by controlling cortisol levels. The proprietary blend of ingredients includes many of the previously mentioned vitamins, minerals, and extracts, such as magnolia officinalis, L-theanine, and phsophatidylserine. Research on Cortitrol® and exercise show it to be very effective at reducing the stress-induced production of cortisol and the free radical production that is associated with high intensity exercise-induced cortisol levels.

Some other potentially effective nutritional interventions for HPA-axis functioning include: American ginseng, Indian ginseng, Asian ginseng astragalus, codryceps, holy basil, licorice, 4-amino-3-phenylbutyric acid, and NAC.

Bottom line: To decrease stress, HPA-axis activity, and cortisol production and secretion:

- **Get plenty of restful sleep (7-9 hours/night!)**

- **Employ lifestyle stress reduction strategies**
- **Eat a nutrient rich diet**
- **Supplement with Carbohydrates (before, during, and after high-intensity exercise)**
- **Supplement with BCAA's**
- **Supplement with PtdSer**
- **Supplement with Cortitrol**

Chapter 12: **TESTOSTERONE**

Testosterone is a steroid hormone within the androgen family that plays specific roles in bone and muscle growth and repair, the breakdown of fatty tissue (lipolysis), immune function, libido, the production of blood cells, as well as many "masculinizing" effects, both in men and women. In men, testosterone is produced primarily in the testes, and in women, it is produced primarily in the ovaries and adrenal glands. Men typically have a 10-fold+ greater production of testosterone than women. This is partly due to the total amount of testosterone that male physiology produces, but also due to the conversion of most testosterone to Estradiol that occurs in female physiology. Somewhat related to this is the correlation between testosterone production and muscle mass with an inverse relationship between testosterone and fat mass. An important thing to note is that estrogens play a role in fat metabolism as a negative effector of lipolysis. In other words, estrogen can bind to receptor sites on adipose (fat) tissue, and thus "block" the breakdown of fat at that specific location. Though oversimplified, the more testosterone you can produce, the more muscle and less fat mass you will make up.

Testosterone production is controlled by gonadotropin-releasing hormone (GNRH). GNRH is produced and released from the hypothalamus at specific intervals, or pulses throughout the day and night. A pulse causes the pituitary gland to secrete Luteinizing hormone (LH), which is directly responsible for initiating the conversion of cholesterol into testosterone. Did you catch that? Cholesterol is the

"backbone" of testosterone, and it serves as the backbone for every steroid hormone we produce. For this reason alone, low-fat diets should raise some red flags.

The production and secretion of testosterone works on a negative feedback loop. If the brain senses there is too much testosterone circulating, then the signal is sent out to convert the "extra" testosterone into dihydrotestosterone (DHT) or estradiol. Along these lines, testosterone is vital to the production of follicle stimulating hormone (FSH), and thus is a major part of male (sperm production) and female (ovulation and estradiol production) reproductive processes.

It is now understood that the "pulsating" production and release of testosterone is one of the main characteristics that makes it anabolic (muscle growing) in nature, and that a steadily consistent level of this hormone would wear out its receptors and have much less of an effect. It is also understood that testosterone has both direct as well as indirect effects on skeletal muscle tissue. One mechanism in which it does this indirectly is by increasing the pituitary gland's production of growth hormone (GH), which also plays a major role in muscle repair and growth. Another indirect mechanism of action for muscle growth is through the neuromuscular system and its neurotransmitters. By binding to or interacting with specific sites of the neuromuscular system, testosterone can change the amount of neurotransmitters (and thus enhancing the potential force production of muscle contraction, i.e. strength!) as well as directly influencing the structural proteins that make up the muscle cell, and thus increase the mass of the muscle cell. The direct

effects of testosterone on muscle are complex, but very beneficial. Testosterone released into the blood is bound to a sex hormone-binding globulin (SHBG) that acts kind of like a chaperone to escort testosterone to specific tissues. Once testosterone arrives at its "target tissue," it is released by the escort (SHBG) and enters the cell. After crossing the membrane of the cell, it binds itself to the nuclear androgen receptor, which then binds itself to DNA. This causes an increase in DNA transcription, and an increase in protein synthesis. When this is consistent and compounded, it results in increases in proteins that are within muscle cells as well as the protein that make up the structure of the cells, and thus increases muscle density and size.

So how does a person maximize all these direct and indirect effects of testosterone on muscle tissue? Lets start with an overview since there are so many different mechanisms. The various mechanisms come about because of the multiple nutrition/exercise intervention theories that have been explored to "time" testosterone pulses in order to maximize the anabolic effect. Some ways to address these specific mechanisms include: Altering macronutrient diet ratios and feeding times, supplementing with specific foods or ingredients to increase androgen/steroid hormone production throughout the day/night or to increase the catecholamine response during exercise, executing a specific type, time, or intensity of exercise, as well as reducing the amount or effects of the hormones and mechanisms that oppose testosterone. Since testosterone levels, and muscle mass, tend to decrease as we age, it is very important to utilize both nutritional as well as

other mechanisms to combat the age-related decline in lean tissue, bone density, and functional mobility.

First, I would like to explain that muscle growth does not occur exclusively through testosterone. The combined effects of testosterone, and other anabolic hormones (growth hormone and IGF-1) can be hard to separate in order to provide an exact cause and effect relationship. Due to this, the effects of these variables on muscle protein synthesis will be explored.

What is the effect on differing dietary macronutrient (Carbs, Proteins, Fats) ratios on testosterone and muscle protein synthesis? First off, it is well established that calorie restriction (dieting), regardless of the macronutrient composition, results in decreases in testosterone levels, and chronically high caloric intake that results in obesity results in reductions in testosterone production, too. So, there is a total caloric "range." Secondly, research is quite clear that when the total percentage of daily calories from fat is held to 20% or less over time, there is a reduction in testosterone production. This is perhaps due to an inadequate amount of available cholesterol for steroid hormone "backbones." Increasing the amount of fat in the diet is correlated to increases in testosterone production, though when fat makes up 40% or more of the total caloric intake (again, over time), this increase in testosterone peaks, and then actually declines. For this reason, many nutrition professionals who specialize in body composition alterations suggest a diet with "moderate" intake of fat (between 20-30% of total daily calories come from fat) to

have the most beneficial effects on testosterone production. There has been conflicting data regarding diets high in protein versus diets high in carbohydrates and their effects on testosterone. Though the evidence seems to lean towards high protein diets resulting in reductions in testosterone in comparison to high carbohydrate diets (whether exercising or not), the overall effect of muscle growth should not be forgotten. In other words, though testosterone production may decrease on a high protein diet, research is clear that a minimum threshold of protein (and slightly less important, the total daily protein intake) or essential amino acids, specifically Leucine, must be met in order to maximize muscle protein synthesis. Recent research on this topic has shown that supplementing with the "building blocks of protein," amino acids, (specifically the Branched Chain Amino Acids: Leucine, Isoleucine, and Valine) during peroids of high-intensity resistance training results in significantly higher levels of testosterone with simultaneously significantly lower levels of cortisol and creatine kinase (a marker of muscle damage).

Related to feeding times and maximal muscle protein synthesis is the issue of the Leucine threshold. Dr. Layne Norton's research on this specific topic provides great insight. In Dr. Norton's writings and presentations, he explains that maximal protein synthesis is achieved by ingesting a threshold dose of ~3.2 grams of Leucine at each meal (5 per day, eating every 4-6 hours). Since the Leucine content is variable in different types of protein, the specific amount (in grams, for example) of protein you eat will depend on the exact source of protein.

Dr. Norton uses this graph to show the differing Leucine content of some protein sources, as well as the amount of each that must be ingested to achieve the threshold dose.

Protein Source	Total Protein	Amount of protein from source to reach 3.2-4.4g Leucine	Amount of food source required
Beef	8.0%	40g	133g (4.7oz)
Chicken	7.5%	43g	139g (4.9oz)
Pork	8.0%	40g	140g (4.9oz)
Egg	8.6%	37g	296g (10.5oz) or approx 5 large eggs)
Fish	8.1%	40g	170g (6.0oz)
Whey	12.0%	27g	variable depending upon whey powder type
Casein	9.3%	34g	variable depending upon casein powder type
Milk	9.8%	33g	932g (33 oz) or approx 4 cups of milk

So, achieving maximal protein synthesis in order to increase muscle growth is accomplished by ingesting the threshold dose of leucine, either from food or through amino acid supplementation.

Exercise, itself, is a strong stimulus for testosterone production, though not all types of exercise are created equal. In general, exercise stimulates testosterone production through catecholamine stimulation of beta-adrenergic receptors. In other words, exercise stimulates the release of hormones and neurotransmitters that in turn, stimulate the nervous system to promote testosterone production and secretion. Resistance training is known to result in acute, and potentially chronic elevations in testosterone that contribute to an amplification of exercise-induced increases in muscle mass. High-intensity endurance exercise also increases

testosterone; however, it also results in high levels of cortisol and highly catabolic processes that often out-weigh the anabolic nature of testosterone. For this reason, dietary measures to counter the catabolic nature of high-intensity endurance exercise must be employed to prevent injury, promote recovery, and maximize adaptations to training (competitive runners and cyclists…I am talking to you!) The greatest and most significant increases in testosterone levels from resistance training are associated with a few specific training variables. The National Strength and Conditioning Association's position on this is as follows: "Independently or in various combinations, several exercise variables can increase serum testosterone concentrations in boys and younger men:

- Large muscle group exercises (e.g., dealift, power clean, squats)

- Heavy resistance (85-95% of 1-repition maximum [1RM])

- Moderate to high volume of exercise, achieved with multiple sets, multiple exercises, or both

- Short rest intervals (30 seconds to 1 minute)

- Two years or more of resistance training experience"

Though the previous statement was male specific, this type of training has been shown both scientifically and anecdotally to increase testosterone and muscle

protein synthesis in both male and female populations, and almost every age group. Long story short, increases in testosterone production come about from consistent, high-intensity resistance training that favors multiple muscle groups across multiple joints. Since testosterone levels normally peak in the early morning, exercise and training performed at this time may result in exercise-induced elevations in testosterone that are easier to come by; however, exercise and training done later in the day seem to be more effective at maintaining elevated testosterone levels. In the end, the time of day seems to have less of an impact on testosterone levels and muscle protein synthesis than consistency, intensity, and exercise selection.

Zinc plays a very important role in metabolism, immune and antioxidant function, as well as in maintaining cell structure stability. It is also a very major player in the normal production and functioning of the "sex hormones." Research is clear that exhaustive exercise has the effect of reducing testosterone levels as well as inhibiting thyroid hormones. Research indicates that Zinc supplementation has a potential to prevent these negative effects of exhaustive exercise; however, the prevention of these effects appears to be "exercise dependent" and may only be significant in anaerobic exercise to exhaustion, as opposed to aerobic exercise to exhaustion.

Calcium supplementation in addition to exhaustive training has been shown to increase testosterone levels to a greater degree than exhaustive training alone. Along these same lines, a combination supplement of Calcium and Vitamin D results

in an increase in testosterone levels in elderly men and women, and this increase is highly correlated to a reduction in the risk of falling. Vitamin D plays a significant role in male reproductive organs, and there exists a correlation between Vitamin D levels and testosterone levels. Some research suggests a distinct seasonal variation in testosterone production, with testosterone production being highest in the summer and fall months and lowest in the winter months. It has been theorized that this is connected to the amount of Vitamin D we can make from skin exposure to sunlight. Related to this, research has shown that supplementing Vitamin D results in significant increases in testosterone levels in men.

There exists some conflicting evidence on caffeine consumption and testosterone production. The theory of increased testosterone production due to Caffeine ingestion is based on the effects that certain stimulants have on catecholamine response (increased epinephrine and norepinephrine). Increased production of catecholamines results in greater stimulation of beta-adrenergic receptors, and this, in a dose-dependent response, promotes testosterone production and secretion. Many studies show an increase above normal exercise-induced elevated levels when Caffeine is ingested prior to exhaustive exercise. One specific study showed that a caffeinated chewing gum improved repeated cycling sprint performance, increased testosterone levels, delayed fatigue, and reduced cortisol. Due to Caffeine's potential effects on testosterone stimulation, it is a very popular ingredient in pre-exercise/pre-competition nutritional supplements.

Though touted for its potential anti-cancer characteristics, research shows that consistent ingestion of garlic actually reduces testosterone production, among other negative effects to the male reproductive organs. Just like everything else in life, moderation is key. I do not think having garlic a couple times a week will have significant effects on testosterone production, especially if you are doing other things to enhance normal production.

Finally, since testosterone is opposed by cortisol and insulin, it is very important to reduce or manage lifestyle stressors as well as limit dietary sugar. Research shows that elevated levels of glucose and fructose inhibit the proper function of SHBG (the chaperone that escorts cortisol to muscle tissue), and thus results in immediate reductions in testosterone levels. The exception to this would be ingesting these sugars immediately before, during, or immediately after exercise, as simple carbohydrates at these times improve performance, aid in recovery, and promote an anabolic state.

Low-testosterone is a legitimate issue that can affect both men and women. Some specific symptoms related to chronically low levels of testosterone include: reduced libido, fatigue, low energy, reductions in bone mineral density and muscle mass, and reductions in work/exercise capacity, strength, and stamina. Blood and saliva samples are typically used to establish circulating plasma testosterone levels. It is now quite common for an individual diagnosed with "low-testosterone" to be

placed on testosterone stimulating drugs and medications to achieve normal/optimal plasma levels.

Bottom line: To increase testosterone levels:

- **Eat a diet with moderate levels of fat (~20-30% of daily caloric intake)**
- **Do not restrict calories (dieting)**
- **Ingest adequate amounts of protein to reach the Leucine threshold (~3.2 grams every 4-6 hours)**
- **Participate in consistent high-intensity exercise (especially resistance training that uses large muscle groups, heavy resistance, moderate to high volume, with short rest intervals)**
- **Supplement with Zinc**
- **Supplement with Calcium**
- **Supplement with Vitamin D**
- **Supplement with Caffeine**
- **Address cortisol (stress) and insulin (sugar intake and glucose metabolism)**

Chapter 13: **GROWTH HORMONE (GH, or SOMATOTROPIN)**

The name pretty much gives it away. Simply put, it promotes growth in children. In adults, the role of GH changes slightly to work directly with metabolism. In this capacity, GH is highly involved in muscle protein synthesis and the breakdown of fat. It is also heavily involved in the shift of fuel preference away from glucose to fatty acids, aiding in amino acid delivery across cell membranes, decreasing the storage of glucose (as glycogen), stimulating collagen growth and synthesis, enhancing immune function, improving the filtration function of the kidneys, as well as promoting the retention of specific elements (nitrogen, potassium, phosphorus, and sodium).

GH, like testosterone, is produced in a pulsatile fashion, though it peaks at night during sleep. This is thought to be a physiologically deliberate occurrence since most tissue repair and growth (especially exercise-induced muscle growth) takes place during rest. The hypothalamus is responsible for producing and releasing growth hormone releasing hormone (GHRH), which then makes its way to the pituitary gland and stimulates growth hormones production and secretion. The hypothalamus, as well as the pituitary gland, is stimulated to produce its respective hormones, in general, when signaled by stress, specific amino acids, or hypoglycemia (low blood sugar). Like
, GH can exist in its "free" form in circulation, though most of it has to be bound to a transporter protein, which puts it in an inactive form. Women tend to have higher

plasma levels of GH in comparison to men, especially through the early follicular phase of the menstrual cycle.

GH works to increase muscle protein synthesis and fat lipolysis both directly and indirectly. GH works directly on these mechanisms when it travels through circulation to target tissues, and binds to GH receptor sites. When GH binds to receptor sites in fat tissue, it causes the breakdown and release of fatty acids into circulation and also prevents circulating fatty acids from uptake and storage in fat tissue. In fact, growth hormones' most potent effect on metabolism may be its role in releasing stored fat and simultaneously preventing circulating fat from being stored. When GH binds to its receptors in muscle tissue it directly affects protein and fat metabolism. GH binding on muscle cells increases the delivery of amino acids across the cell membrane while simultaneously inhibiting free amino acids from leaving the cell to enter circulation. By providing muscle cells the amino acids required for the production of more muscle protein and limiting the amount of amino acids that can "escape" these cells, GH directly affects the synthesis of new muscle. GH binding on muscle cells also encourages free fatty acids to enter muscle to be used as fuel. In doing this, there is a shift to use more fat, and less glucose, as fuel for muscle work. Due to this accumulation of fat within muscle and as well as an accumulation of circulating glucose, GH has the potential effect of increasing insulin resistance, though this should not be an issue if other dietary measures are taken to maintain and promote sensitivity to insulin. Growth hormone therapy, when supervised by physician to correct GH deficiency, should not result in glucose

metabolism/insulin sensitivity issues; however, unsupervised GH administration (injected GH to increase muscle mass or improve athletic performance) can potentially end in diabetes due to the compounding effects of decreased insulin sensitivity.

GH works both directly and indirectly on muscle protein synthesis and lipolysis when binding to receptor sites on the liver. When binding at the liver, GH causes the breakdown of stored glucose (glycogen) as well as the production of new glucose from circulating amino acids (gluconeogenesis). Indirectly, though very potently, GH affects muscle protein synthesis and lipolysis by stimulating the production of insulin-like growth factors (IGF-1).

The amount of restful sleep you get every night may be the biggest factor related to maximizing total daily GH production. Since GH levels peak at night (reaching levels up to 20 fold higher than baseline levels) and are maintained longer than exercise-induced levels, getting 7-9 hours of restful sleep every night should be a priority. Though some studies show acute (every once in a while) sleep deprivation may result in no change in the GH pulsatile pattern, other research shows the overall production remains the same in a 24 hour period, but the night peak is reduced and the rest of the pulsatile bursts are slightly more robust. When sleep deprivation happens more consistently, like 2-3 days in a row, the body actually produces more GH (as well as a wide variety of circulating immune cells) in order to combat the increase in total physiological as well as psychological stress.

Chronic sleep deprivation has been linked to many negative outcomes, the most prominent being obesity. It is now understood that chronic sleep deprivation causes decreases in GH (and testosterone!) by direct and indirect mechanisms. Directly, chronic sleep deprivation fatigues the hypothalamus and pituitary gland due to an elevated and constant stress response. Indirectly, chronic sleep deprivation increases the production of cortisol and somatostatin (explained later in this chapter), which oppose and inhibit GH signaling and production.

Exercise is known to be a significant stressor that directly increases GH production acutely; however, the exercise variables must fall within a specific criterion. In order to get a significant increase in plasma GH levels, resistance training must reach or pass a specific intensity threshold. Research shows that "heavy resistance training" of loads around the 10 repetition max range (10RM) with shorter rest (greatest GH production with rest periods of 60 seconds or less) and higher volume (3 or more sets per exercise) results in the most significant increase in growth hormone.

Related to the exercise-induced increases in GH is nutrient supplementation. Research is quite clear that a meal composed of carbohydrates and protein a couple hours before training results in even greater exercise-induced GH production. Similarly, pre-exercise and intra-exercise supplements containing protein and carbohydrates have also been shown to increase exercise-induced GH production.

The next issue is macronutrient ingestion immediately after exercise. Since GH levels peak immediately after exercise and reach normal levels with 30-60 minutes after exercise, the potential "problem" of carbohydrates causing an insulin response that blunts GH after exercise may not be that much of a problem. In fact, some studies have shown that a liquid supplement (post-workout shake) containing both carbohydrates and protein ingested after resistance training can actually increase GH (and other anabolic hormones) production immediately, as well as result in greater production over the next 6 or so hours. So if you want to maximize GH production without supplements, just be sure to get a mixed meal of simple and complex carbohydrates and protein about 2 hours before training and as soon as possible after training.

There is an extensive list of supplements that have been studied to see their effects on GH. The most prominent are: Creatine, BCAA's, GABA, Arginine, Ornithin, Taurine, Lysine, Glutamine, L-Dopa, ZMA, A-GPC, and Melatonin.

Studies show that a single 20-gram dose of Creatine can cause increases in GH to levels similar to exercise-induced GH levels, though there is some inter-individual variability.

Though to date, no human research has shown definitive proof that Branched Chain Amino Acids have any effect on GH. BCAA's may or may not increase GH. If they do, it is probably in conjunction with high intensity exercise. What is known

about BCAA supplements is that they have been shown to be extremely effective at increasing muscle protein synthesis.

GABA, or gamma aminobutyric acid, is an amino acid as well as inhibitory neurotransmitter. When GABA levels increase within the brain, somatostatin neurons are blocked and inhibited which results in greater production of growth hormone. Research shows that when oral (3 grams) GABA is taken at rest, the plasma concentration of GH can increase by as much as 400%, which is similar to exercise-induced elevations. When oral (3 grams) of GABA is ingested prior to high-intensity resistance training, the GH response was elevated up to 600% above resting levels. These results show that GABA supplementation by itself increase GH levels, and in addition to heavy resistance training results in greater increases in GH production than exercise alone. One very important thing to note is that research shows that when GABA is taken consecutively for 4 days and then not taken, normal GH production may be blunted. Due to these findings, I do not recommend GABA supplementation on consecutive days.

Glutamine, a non-essential amino acid (although it is considered "conditionally essential" in cases where the body cannot make enough of its own Glutamine), when ingested orally as a supplement, has been shown be a significant stimulator of GH production. When 2 grams of Glutamine is mixed into a sugar-containing liquid (some studies used soda!) and ingested over the course of 20 minutes, GH levels increase gradually, but significantly, over time, peaking around

90 minutes after ingestion. One study has shown that at 90 minutes after ingestion, individuals supplementing with Glutamine had 4-fold greater levels of plasma GH than those who did not ingest Glutamine. For those of you who assume "more is better," please stick to the specific 2-gram (or ~27mg/kg of body weight) recommendation. Ingesting less than 2 grams may not provide a strong enough stimulus to cause GH release, and more than 2 grams may cause the body to "reflexively" remove the Glutamine from circulation, and also prevent the GH stimulus from occurring. More is NOT always better. Another important note is that the majority of research on Glutamine supplementation with resistance training shows no greater improvements in strength, muscle mass, or measure of recovery in comparison to resistance training alone, so it may be a potentially beneficial supplement to add on days or periods when you are not training.

The amino acid Ornithine, which was originally thought to only play a role in the body's nitrogen balance and urea cycle, has been used medically to decrease the amount of time it takes for wounds and burns to heal in malnourished patients. When it is combined with an alphaketogluterate (AKG) molecule to become Ornithine-AKG (OKG), it has been found to promote the release of some anabolic hormones, including GH and insulin, during calorie restriction or fasting states. By itself, Ornithine or OKG seem to be ineffective at increasing GH unless you stop eating…which seems very counterproductive.

Though supplementation of Arginine in a resting state (no exercise) results in increases of GH (about double), supplementation of Arginine in conjunction with resistance training results in GH levels that are lower than levels induced by exercise alone. If you are resistance training, supplementing with Arginine for GH elevations will not be beneficial.

Research shows that when Ornithine and Arginine are combined (and in conjunction with heavy resistance training) and supplemented for as little as 3 weeks, plasma GH as well as IGF-1 concentrations were elevated higher than levels caused by resistance training alone, showing that, at least for short term improvements, an Arginine-Ornithine combination supplement works! Due to this research, supplement companies have begun to make this combination supplement so you do not have to buy two supplements and measure specific doses of each.

A review of other amino acids that are claimed to increase GH, including Lysine and Taurine, shows that though these may cause GH production in rats, the effects on humans is different. In some cases, a GH response can occur when these amino acids are intravenously administered. From a practicality stand point, how easy is it for anyone to set up an amino acid IV drip on his or her drive to the gym? This review showed that oral doses may also cause a GH response, but the specific amount needed to do so would cause gastrointestinal distress, stomach pain, diahrea, and that these effects would prevent anyone from wanting to do any exercise, let alone high intensity resistance training for maximal GH response.

L-Dopa (Levodopa) is a precursor to dopamine, which is a neurotransmitter. It is an ingredient in medication to treat Parkinson's disease. In some studies, L-Dopa has been shown to increase levels of growth hormone Releasing Hormone (GHRH), and thus may indirectly increase GH production, though research on humans has yet to find convincing evidence, especially as it relates to GH associated fat loss and muscle growth.

Zinc Magnesium Aspartate, or ZMA, has been touted as a supplement to improve recover as well as increase levels of testosterone and growth hormone. Studies on ZMA show that though it may increase plasma levels of zinc, there are no changes on markers of muscle strength or changes in either testosterone or growth hormone.

Alpha-glycerylphosphorylcholine (A-GPC) is a phospholipid substance made from soy lecithin. It has been claimed to improve cognition, as it relates to certain "aging" diseases, as well as potentially increase GH production. Research in this area is still very new, and the total GH effect seems to be more significant in elderly populations, so until some solid data on A-GPC supplementation with exercise and GH is done, I would not put it on the list of supplements to try.

Melatonin is a very common on-the-shelf sleep aid. It does this by aiding in the setup of circadian rhythms and changes in sleep patterns to seasons. Since most research on Melatonin has dealt with its role in sleep and sleep disorders, there is

not as much data to go on as it relates to GH production. Research shows that normal doses (.5 – 5 grams) can cause rapid and significant increases in plasma GH levels. The interesting thing about Melatonin is that it can increase GH without having any effect on growth hormone releasing hormone (GHRH). The exact effects of Melatonin supplementation in conjunction with exercise are not fully known. Some studies show it may increase GH when ingested prior to cycling, but its effects on resistance training are still a mystery. You should not supplement with Melatonin for longer than 2 weeks at a time as this may negatively affect normal circadian rhythms and mess up sleeping patterns, which will negatively affect normal GH production.

Though at this point, no definitive research on humans exists on the effects of copper supplementation on GH, research published in 2011 studied these effects on growing pigs. They found that doses of 125 mg/kg copper sulfate or copper methionine resulted in increased growth performance and feed efficiency, eating the same amount of food as the pigs in the control group. This increase in dietary Copper increased the production of growth hormone releasing hormone while reducing the production of somatostatin.

There are a few things that play a direct role in preventing normal or optimal levels of GH. As previously stated in the ghrelin chapter, ghrelin is also a stimulator of GH production. This has been made very evident by research on calorie restriction and fasting, where ghrelin (and hunger) levels increase, resulting in

increased GH production to minimize the use of glucose and increase the amount of stored fat as fuel. Research shows less than optimal levels of GH are quite typical in obese individuals, as is a reduction in ghrelin levels or ghrelin function. Altered ghrelin levels may be one reason for alter GH levels in over-weight and obese individuals.

Due to the fact that insulin inhibits and opposes GH, diets that are high in carbohydrates may result in less GH production throughout the day. Though the short term effects may be insignificant since the majority of GH is produced at night or in response to exercise, the long-term effects of this type of diet may result in chronically lower levels of GH production and secretion, especially if the diet results in increases in fat mass. Related to this is the issue of carbohydrate timing. Regardless of the make up of your diet, ingesting carbohydrates before bed, or perhaps even a few hours before bed, may have the effect of blunting GH release. Since this is the time of day when the GH "mother-load" is produced, I strongly recommend steering clear of anything that would negatively affect it. So, no carbs before you sleep.

As previously mentioned, GH is also called somatotropin. A similar sounding hormone, somatostatin, is a hormone that opposes the production of many other hormones. In fact, it may be one of the strongest inhibitors of GH production. Due to this, a great percentage of research in the area of GH production and secretion is devoted to ways of inhibiting the production of somatostatin. Since somatostatin

can be produced and released due to hyperglycemia (high blood sugar), a very easy way to limit somatostatin production is to prevent unnecessary spikes in blood glucose, i.e., limit simple carbohydrates and sugars to only right after you exercise.

Bottom line: To increase growth hormone secretion:

- **Get plenty of sleep every night (7-9 hours!)**
- **Consistently participating in high-intensity resistance training (high volume, heavy weight, and short rest breaks)**
- **Eat a carbohydrate and protein mixed meal or supplement prior to, and immediately after training**
- **Supplement with Creatine**
- **Supplement with Branched Chain Amino Acids**
- **Supplement with GABA**
- **Supplement with Glutamine**
- **Supplement with Arginine/Ornithine**
- **Supplement with L-Dopa**
- **Supplement with Melatonin**
- **Steer clear of Taurine, Lysine, ZMA, and A-GPC supplements that claim to increase GH**
- **Limit high carbohydrate diets and sugar before you go to sleep.**

Chapter 14: **INSULIN-LIKE GROWTH FACTOR (IGF-1)**

A group of hormones called Somatomedins, or insulin-like growth factors, act similar to insulin (they cause glucose to enter muscle and fat cells) and also work to mediate the role of growth hormone. When GH reaches the liver, it signals the production of somatomedin C (also called IGF-1). The IGF-1 stimulation by growth hormone is "delayed" in that it takes anywhere from 8 to 30 hours for liver DNA to synthesize IGF-1. Specific types of stress, such as exercise, cause an immediate increase in plasma IGF-1 levels, indicating that other tissues (muscle and fat) can produce IGF-1.

So here is where things can get really difficult. Research shows that the level of IGF-1 in the blood is highly correlated to increased risk of many types of cancer, including prostate cancer. The higher your level of plasma IGF-1, the higher your risk of getting cancer. Research also shows that the level of IGF-1 in the blood is directly related to the amount of dietary protein (amino acids). The more dietary protein or amino acids you eat, the greater the amount of IGF-1 the liver produces and puts into circulation. One caveat to the amount of protein deals with the protein source. Vegans who eat high amounts of plant source protein do not seem to have high levels of circulating IGF-1, though most people would find it difficult to increase muscle mass on a true vegan diet. So based on this, a high protein diet sounds like a very bad idea. Well, the issue of IGF-1 and cancer is not that simple. Research has shown that IGF-1's ability to increase or promote cancer is related to the body's

ability to use vitamin D. In fact, research shows that high IGF-1 levels do not increase the risk of cancer when vitamin D levels and status are "high."

Once again, I think the issue comes down to some common sense. Though perhaps in theory, a high protein diet may increase cancer risk, studies looking at these types of diets have shown them to be beneficial at preventing cancer! I see it from two main perspectives: You can cut way back on protein and calories and live a long, but frail life (Sarcopenia and Oseoporosis from inadequate protein and calories), or you can eat a diet full of fruits, vegetables, and enough protein to allow for maximal protein synthesis, and live a long, active life. If you think having high levels of IGF-1 will increase your cancer risk, then exercise more and stay away from the standard American diet, fast food, and other garbage. Protein is not the problem.

How exactly does IGF-1 work to increase muscle mass? IGF-1 plays a major role in muscle protein synthesis by activating anabolic pathways, specifically the IGF-1Akt pathway. The names and specific processes involved are very confusing and complex, so for the sake of practicality, we will avoid that. The important thing to remember is that when IGF-1 binds to its receptor on specific tissue, it causes a domino effect of a sequence of events within a cell that results in less protein broken down and more amino acids combined into specific proteins.

In the case of hypertrophy (increases in muscle size), it is most likely that locally produced IGF-1 (within the muscle cells as opposed to IGF-1 produced by the liver) is the main contributor to increases in muscle protein synthesis since the liver production of IGF-1 is delayed and increases in muscle size are not correlated to increases in circulating IGF-1 in the blood (put into circulation by the liver). In fact, when IGF-1 is administered acutely, increases in muscle protein synthesis can be observed; however, when administered over the course of a year, no changes occur in muscle mass or body composition.

When IGF-1 is produced within muscle cells, it is given the name mechano-growth factor (MGF). As previously mentioned, the primary actions of IGF-1 in muscle protein synthesis and increases in muscle mass are due to MGF. MGF may also contribute to increases in muscle size and strength by activating and then making available satellite cells. Satellite cells are muscle stem-cells that donate their nuclei to muscle cells so that more protein synthesis can occur. A very important note is that production (or perhaps release) of MGF by muscle cells only occurs under mechanical loading, especially eccentric loading (resistance or weight training). Multiple rat studies have shown that rats that are deficient in plasma IGF-1 (IGF-1 produced by the liver) can still increase muscle mass when their muscles are placed under the tension of a mechanical load (weight training for rats!) due to the production of MGF.

What a perfect segue to the next issue: Exercise and IGF-1 response. Research is very clear that exercise, especially resistance training, causes an increase in local IGF-1 (MGF), as well as an increase in plasma IGF-1 levels if these levels are low before exercise. If IGF-1 levels are high prior to exercise, there is typically no change in plasma levels. Once again, plasma levels of IGF-1 after exercise do not tell us what is going on at the cellular level within muscle cells because plasma levels do not necessarily differentiate between IGF-1 and mechano-growth factor. Though the exact relation is not understood, it seems very plausible that exercise-induced increases in plasma IGF-1 will also lead to increases in cellular MGF. Research does show that consistent resistance training that promotes plasma IGF-1 production also increases the activity of DNA/RNA involved in MGF replication and synthesis, and thus potentially increase MGF production.

Due to this, specific supplements that increase plasma IGF-1 either in conjunction with exercise, or at rest, most likely play a very small role in muscle protein synthesis. What should be noted; however, is that even though increasing plasma levels of IGF-1 may not promote muscle protein synthesis; elevated levels of IGF-1 do prevent muscle protein breakdown and degradation. Research is clear that plasma IGF-1 has an "anti-catabolic" effect on muscle tissue and helps prevent negative nitrogen balance (catabolic state). Therefore, supplements that are claimed to increase IGF-1 in conjunction to resistance training or in a resting state may be beneficial at maintaining muscle gains and limiting losses in muscle mass.

Since GH stimulates IGF-1 production, the supplements listed in the GH chapter may be effective at increasing plasma IGF-1 levels.

So to sum this potentially confusing hormone: IGF-1 production is dependent on a growth hormone stimulus as well as the amount of dietary protein you ingest. The plasma levels of IGF-1 may do more to prevent muscle protein loss than they do to promote muscle protein synthesis. The most significant changes in muscle protein synthesis related to IGF-1 are actually the effects of MGF, which is produced due to mechanical load and stretch of muscle tissue.

Bottom line: To increase plasma IGF-1 levels:

- **Promote GH production**
- **Increase dietary protein or amino acid intake**
- **Promote MGF associated muscle protein synthesis by mechanically loading muscle tissue (resistance or weight training).**

Chapter 15: **PUTTING EVERYTHING TOGETHER**

When it comes to nutrition, regardless of your goal, you can never eat too many fruits and veggies. Everyone knows that eating more of each is good for you. So eat more! Next time you are at the grocery store, try a fruit or vegetable you have not had recently, or ever. I cannot stress enough how dependent our bodies are on the nutrients that are found only in fruits and vegetables. Add some to every meal. Regardless of what your goals are, the addition of one or two more servings of fruits and veggies (replacing grains, pasta, or other carbohydrates if you think calories are still the problem) will help you get there as well as provide for easier maintenance when your goal is reached. When I say carbohydrates at all in this book, I do not mean fruits or vegetables. Eat as many fruits and veggies as you can stand, and then some. Along these same lines is the issue of water. Again, regardless of your goals, drinking more water will help. Water is one of the most over-looked or neglected aspects of nutrition. Drink more of it!

I hope in reading the previous chapters you picked up on the fact that many of these hormones work very closely with each other, or oppose each other. I think that is a comforting notion because instead of worrying about the tiniest issue related to one specific hormone; I know that eating a specific kind of food will result in a "general effect."

So, what general effect, or effects, are you looking to achieve: Fat-Loss or Muscle-Growth? Lets break down both scenarios so you can see the practical outworking and application of each to change your hormones. Obviously, you cannot take every supplement discussed in this book, so I will only include the ones I think show the most research or potential.

FAT-LOSS

If the effect you are seeking is a decrease in fat mass or improvement in body composition, based on the previous chapters, here is a basic way of planning out your day.

Role of Protein: Promote "fullness" and repel "hunger" by activating the hormones involved in appetite-control more often.

Role of Carbs: Provide pre-workout and intra-workout fuel, as well as post-workout recovery. Other than that, limit the amount of carbohydrates you eat.

Role of Fat: Promote "fullness" and repel "hunger" by activating the hormones involved in appetite-control as well as provide sustained energy.

NOTE: These are not the ONLY roles of the macronutrients as they relate to Fat-loss, these are just the main goals.

Sleep 7-9 hours.

Wake up:

Drink some water. Since you are drinking, now is the perfect time to take: Vitamin D, Magnesium, and Calcium.

Breakfast:

Drink more water.

Eat a protein/complex carbohydrate rich breakfast to maintain "Full" signal.

Try a 3-5 egg omelet (cage-free/organic eggs, real butter or coconut oil, with bacon, onion, spinach, and cheese (season to your liking with turmeric, pepper, basil, oregano, and cumin).

Skip the fruit juice, but eat the real thing, like apples, oranges, berries, or bananas.

OR

Oatmeal with protein powder (about 30 grams) and fresh fruit.

Another option is a shake/smoothie. Try blending:

- 1 cup organic whole milk or unsweetened almond milk
- 1 cup frozen blueberries or raspberries
- 1 cup frozen strawberries or blueberries
- 1 scoop of protein powder (about 30 grams of protein)
- 1 handful of fresh baby spinach

After you have some food in you, take: Omega-3's and ALA.

Snack:

Drink more water.

Hard boiled eggs and carrots/celery/broccoli

Or

Walnuts/pecans/cashews and a salad (spinach, kale, green pepper, tomatoes, etc.)

Or

Protein bar (Quest Bar) or Protein Shake and some fruit or veggies: apples/oranges/raspberries/blueberries/carrots/celery/broccoli/

Or

Full Fat cottage cheese and fruit

Lunch:

Drink more water.

Pack your lunch with lots of protein rich foods, like tuna, chicken, or turkey sandwiches on high fiber bread (Or skip the bread!).

All natural unflavored full-fat Greek Yogurt with berries

Fresh fruits and veggies or a salad (with olive oil and vinegar dressing:

apples/bananas/oranges/grapes/peppers/ broccoli/carrots/celery/

Supplement with more ALA and Omega-3's

Snack or Pre-workout:

Drink more water.

Protein bar/shake with some fruit:

apples/oranges/grapes/pears/bananas/berries

OR

Branch Chain Amino Acids (BCAA's) with fruit:

apples/oranges/grapes/pears/bananas/berries

Snack or Post-workout:

Drink more water.

All natural unflavored full-fat Greek Yogurt with berries or honey

OR

Full fat cottage cheese with fresh fruit:

apples/oranges/grapes/pears/bananas/berries

OR

Chocolate milk and some walnuts/pecans/cashews

OR

Protein bar/shake with fresh fruit:

apples/oranges/grapes/pears/bananas/berries

Dinner:

Drink more water.

Make sure the last meal of the day is full of high quality protein and as many veggies as you can handle.

Try grilled or baked wild-caught salmon, free-range chicken breast, pork chops, or grass-fed beef.

Steamed, baked, or fresh veggies:

Broccoli/carrots/green beans/zucchini/squash/eggplant/sweet potatoes/beets

Salad: Olive oil and vinegar dressing

spinach/kale/other greens/tomatoes/beets/avocado/cucumber/

onion/celery/mushrooms

Supplement with more ALA and Omega-3's

Before Sleep:

No SUGAR!

Drink more water, and try adding in a fiber supplement.

Casein protein shake

OR

6-8 ounces of Lactose-free Kefir

MUSCLE-GROWTH

If the effect you are seeking is an increase in muscle mass, strength, or other lean tissue adaptation, based on the previous chapters, here is a basic way of planning out your day. The over-arching goal is to maximally stimulate protein synthesis as many times as possible (Every 4-6 hours). Shoot for 4-5 times throughout the day: Each meal, immediately before/after exercise, and right before bed.

Role of Protein: Promote anabolic state, trigger maximal protein synthesis, aid in recovery and repair

Role of Carbs: Provide fuel for pre-/intra-/and post-workout, as well as promote anabolic state by refueling and increasing the amount of nutrients that can get into working cells.

Role of Fat: Promote anabolic state by providing building material for anabolic hormones, and providing sustained fuel.

NOTE: These are not the ONLY roles of the macronutrients as they relate to Muscle-growth, these are just the main goals.

Sleep 7-9 hours.

Wake up:

Drink some water. Since you are drinking, now is the perfect time to take: Vitamin D, Magnesium, Calcium, and Zinc.

Breakfast:

Drink some water.

Get out of a catabolic state and signal maximal protein synthesis by getting that ~3.2 grams of Leucine (Either by protein or Amino Acids)

Protein shake (~30 grams of protein)

3-5 egg omelet (cage-free/organic eggs, real butter or coconut oil, with bacon, onion, spinach, and cheese (season to your liking with salt, pepper, basil, oregano, and cumin).

Skip the fruit juice, but eat the real thing, like apples, oranges, berries, or bananas.

OR

"Beefed-up" protein shake:

1 cup organic whole milk or unsweetened almond milk

1 frozen banana (peel it before freezing)

1 BIG spoonful of almond butter or peanut butter

Protein powder (30-50 grams)

1 handful of fresh baby spinach

Supplement with Omega-3's

Lunch:

Drink some water.

Pack your lunch with lots of protein rich foods, like tuna, chicken, or turkey sandwiches on whole-wheat (high fiber) bread

A couple handfuls of: Walnuts, almonds, pecans, or cashews.

All Natural unflavored full-fat Greek yogurt with berries

Salad or fresh fruits and veggies:

Spinach/kale/tomatoes/peppers/broccoli/carrots/celery

apples/bananas/oranges/grapes

Supplement with Omega-3's

Pre-workout:

Drink some water.

Protein/Carb supplement

AND/OR

BCAA's

AND/OR

Caffeine

AND/OR

Creatine

AND/OR

Glutaime

AND/OR

GABA

AND/OR

L-Dopa

AND/OR

Arginine/Ornithine

Post-workout:

Drink some water.

Protein/Carb shake (30-50 grams of protein)

A couple handfuls of: walnuts, almonds, pecans, or cashews

OR

Full fat cottage cheese with fresh organic fruit

A couple handfuls of: walnuts, almonds, pecans, or cashews

Dinner:

Drink some water.

Make sure the last meal of the day is full of high quality protein and as many veggies as you can handle.

Try grilled or baked wild-caught salmon, free-range chicken breast, pork chops, or grass-fed beef.

Steamed, baked, or fresh veggies:

Broccoli/carrots/green beans/zucchini/squash/eggplant/sweet potatoes/ beets

Salad: Olive oil and vinegar dressing:

spinach/kale/other greens/tomatoes/beets/avocado/cucumber/onion/ celery/mushrooms/olives

Supplement with Omega-3's

Before Sleep:

No SUGAR!

Drink more water, and try Melatonin

Pre-sleep shake:

Casein protein powder (30-40 grams of protein)

1 cup water or lactose free kefir

1 cup spinach or other greens

Handful of: pecans, walnuts, almonds, or cashews

That is it, folks. Now go change your diet and hormones so you can get the body you want!

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