The Ultimate Diet 2.0

by
Lyle McDonald

This book is not intended for the treatment or prevention of disease, nor as a substitute for medical treatment, nor as an alternative to medical advice. It is a review of scientific evidence presented for information purposes only. Recommendations outlined herein should not be adopted without a full review of the scientific references provided and consultation with a physician. Use of the guidelines herein is at the sole choice and risk of the reader.

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Foreword

After my last little drug book on Bromocriptine, I wanted to get back to my main area of interest and real area of expertise: integrated methods of training and nutrition. I will be mentioning some drugs that might be useful along the way, but that's not the main focus of this book. Actually, the best way for me to introduce this book is with a story/history lesson.

The story of this book begins just over twenty years ago in 1982. That year, Dan Duchaine and Michael Zumpano introduced the Ultimate Diet to the world of bodybuilding. In a nutshell, the Ultimate Diet was a 10 day cyclical diet and training plan, incorporating three different training and eating approaches in a coordinated fashion. Drug options were suggested since the laws were less stringent then. It was radical and revolutionary for its time combining cutting edge science with good old intuition (and maybe a little lucky guesswork) to create a complete plan for generating extraordinary results in ordinary people. It achieved something few plans could claim: fat loss with muscle gain or at the very least fat loss with no muscle loss. With slight adjustments in calorie intake it could be used for muscle gain with minimal fat gain.

Fifteen years later, the same Dan Duchaine released an "updated" version of that diet in his seminal book Underground Bodyopus: Militant Body Recomposition. Now a 7 day plan, without nearly the complexity as the original Ultimate Diet, Bodyopus kicked off an entirely new interest in the bodybuilding subculture regarding cyclical diets and cyclical ketogenic diets in particular. This is where I enter the story.

In 1997, I was terribly bored with my life, looking for something to do. I latched onto the Bodyopus diet like a drowning man grabbing a life preserver and never looked back. In one sense, it mirrored Duchaine's original interaction with Zumpano and the Ultimate diet back in the 80's. At that time, Zumpano was the guru and Duchaine was the bored detail man looking for something new to do with his life. In 1997, Dan was the guru and I was the bored detail man. That's how I like to think of it anyhow; I don't know if Dan saw it like that or not.

In any event, what started as a whim, writing a weekly diary of my experiences on the diet turned into something far more. A couple of years later, I wrote the be-all, end-all book on ketogenic dieting. Even Dan admitted I knew more about "his" diet than he did which was as great an honor as I could receive. I became the keto-guy (a nickname I still can't shake no matter how hard I try) even though I never really advocated them in the sense that you'd think. Against all odds, considering how badly it was written and how boring it was to read, the keto book actually sold decently. If nothing else, it established me as a "name" in the industry. A detail-obsessed geek, mind you, but a "name" nonetheless.

In the 6 years since that book, research into human physiology, nutrition, biochemistry, etc. has advanced at an amazing and exponential pace. Science is finally getting to the mechanistic reasons that things happen in the body. Knowing how things happen in the body allows for a certain measure of control. This book represents an integrated approach to all of it.

A note on the title of this book

In case you didn't look at the cover, the title of this book is The Ultimate Diet 2.0. Before I go any further, I want to explain the reasoning behind the name. The first reason, mind you, is that I suck at coming up with creative names (the titles of my last two books prove that). At least I'm honest. But even if I were creative, I still would have chosen to call it The Ultimate Diet 2.0, so that's actually not the main reason this time.

If there's a primary reason I chose the title I did, it's out of tribute. As above, this book is basically an update and revision to the dietary approach that helped me gain whatever amount of respect and importance I hold in the bodybuilding or sports nutrition world. From the Ultimate Diet came Bodyopus. From Bodyopus came my first book. From my first book came my career.

Now, since I got involved with this whole mess at the Bodyopus stage of things, you may be wondering why I didn't call this Bodyopus 2.0 instead. There are a few reasons. First, it just seemed a bit inappropriate. Bodyopus was Dan's brainchild and the name held a very specific meaning to him. The Ultimate Diet 2.0 is a little more generic of a title and just seems a little less pretentious in that respect. Or not.

Also, Dan passed away three years ago (2000), and it just seemed disrespectful to confuse people with two books called Bodyopus. Even though it's a few years out of date and had a minor number of technical mistakes, I still think everyone should own a copy of Bodyopus. Having two books with that title would have confused the average person. Finally, I didn't know who legally owned the rights to the Bodyopus name and didn't want to get my ass sued.

Perhaps more importantly, this book doesn't really revise the seven day Bodyopus plan, with its two types of weight training (tension and depletion) and two distinct dietary periods (low- and high-carb). My first book was closer to that, more of a detail/technical manual for keto/cyclical keto diets. If anything, that first book should have been called The Bodyopus Companion or something equally silly.

Rather, what I'm going to describe here comes directly out of the original Ultimate Diet plan from 1982, which had three types of weight training, three types of eating, and a lot more complexity (and potential sources of confusion). On various levels, including the most fundamental ones, the original Ultimate Diet was the impetus for this book; as a tribute/revision to that diet, I think the names should be the same.

The title of this book, The Ultimate Diet 2.0, has several meanings. The first is simply one of tribute to Duchaine and Zumpano's original Ultimate Diet, released all the way back in 1982. It's fascinating reading now and they were ahead of their time by many years. This book is also an update to that same diet integrating findings about metabolism, fat loss and muscle gain to optimize it. Anyone involved with computers knows that new versions of stuff get a new number. Hence 2.0. Finally (hopefully), it will be the last diet you need. Hence the word "ultimate."

Introduction

Face it, you're normal. Well, more normal than you like. Even though you've trained like an animal, taken all the supplements and done everything you're supposed to, you're still closer to normal than not. Sure, you carry more muscle than the average person on the street (which isn't saying much) and you're leaner too (which is saying even less). You're as healthy as it gets (physically anyhow) and your doctor is thrilled.

But we both know that there's that nagging voice that isn't satisfied: normal isn't the same as content. Either you don't have as much muscle as you'd like or you can't get rid of the last little bit of fat. How do I know this? Am I psychic? Have I been watching you through your window? No. I know this because folks who have optimized metabolisms and good genetics don't buy books like these in the first place. We always notice them in the gym, the folks who seem to break all the rules of training and diet and still look better than we can hope to. They do nothing but bench and curls, eat a junk food diet and just get big, symmetrical and ripped. We can, and should, hate them. No, it's the folks who are looking for salvation who keep me in business.

In buying this book, you're continuing a long-held tradition of grasping at anything that might offer a solution. If at all possible, it should be simple and unobtrusive. Maybe a pill. Well, I hate to burst your bubble but this is the truth: if it were simple and easy to become supranormal, everyone would do it and you'd see a lot more examples walking around.

What I'm going to describe in this book isn't easy and it isn't simple. It's also not magic. I have some physiological tricks that may look like magic, but they're not. Since my biggest bodypart is my brain, I've used it to (re)develop an integrated system of training and nutrition (and some supplements and drugs) that let you sidestep some of the problems inherent in achieving a supranormal body.

In redeveloping the original Ultimate Diet, I've applied the most cutting edge research available, discussed various elements of the diet with many other smart folks, and done cycle after cycle testing the diet both in myself and in my multiple guinea pigs. After many years of testing, design and redesign, I give you the Ultimate Diet 2.0 (UD2 from here on out).

Chapter 1: What this book is and who it's for

So here we are again, another book, another chapter on defining the problems. If you read my last book, you already probably have some idea what I'm going to say. In short, dieting to low bodyfat levels sucks. Actually, dieting sucks across the board but the real problems start when you start to get far below normal. So what's normal?

In modern times, an average male may be carrying 18-25% bodyfat, an average female 21-28%. Many, many (too many) people are much fatter than that.

Healthy bodyfat levels are considered to be 11-18% for men and 18-25% for women. To the body-obsessed, except maybe at the lower levels, that's still fat. Male bodybuilders (and other athletes) think in terms of sub-10% bodyfat levels, females typically in the low to mid teens. Researchers would probably debate the validity of such beliefs but who cares; if you believe it, it's true to you. Perhaps more important is that it is your goal.

Most diets or diet books are aimed at the folks who are trying to get somewhere in the realm of average. There are tons to choose from out there. Any discovery or piece of research that might affect these folks can be turned into a quick fix diet book. One of these days, I'm going to write my own, make a zillion dollars and retire.

For obese folks just trying to lose weight, pretty much any non-retarded diet will work. The main issues to deal with there have more to do with anxiety and the issues involved in changing long-term eating and activity patterns. And even though some readers might disagree, getting a male to 12-15% bodyfat or a female into the 18-22% range usually isn't that difficult. Basic food control, adequate protein and exercise will usually get it done without too much trouble. This book isn't aimed at either group.

By the time folks get to the 12-15% (18-22% for women) range, anxiety, food control and changing habits usually aren't the problem. For bodybuilders and athletes meticulous food control and training is part of the lifestyle. It's when folks start trying to achieve the lower extremes of bodyfat percentage that other problems start to occur. Ravenous hunger, severe muscle loss, metabolic slowdown and screwed up hormones are a few of the usual problems. Women and some men have an additional problem mobilizing and getting rid of stubborn fat (hip/thigh area for women, ab/low-back fat for men).

In presenting the UD2, I'm going to assume that you already have the discipline and anxiety issues well under control. While they are less of a problem on this diet than on many others, it's the real physiological problems I'm setting out to address and fix.

Who am I?

I imagine most readers know me as the author of The Ketogenic Diet, which is more or less considered to be the be-all, end-all book on low-carbohydrate dieting. Fewer readers seem to be aware of my second book, which dealt with the drug Bromocriptine. If you've read either book, you'll have a better background to understand the information in this book. If not, don't worry, I'll try to give you enough background to understand the UD2. To be honest, to give the rationale for everything in detail would take more pages than I want to take. I'm going to cover the basics and you'll just have to take my word for the rest.

Who are you?

So who are you, the ideal UD2 candidate? Actually, let me backpedal a bit and talk about who this diet isn't for. It's not for rank beginners. The training and dietary recommendations simply aren't appropriate for someone just starting out. Get 3-6 months of basic training under your belt and get your basic diet dealt with first before even considering the system described in this book. As above, it's not for the general fat folks out there. In general, until males hit 12-15% bodyfat and females 20-22% bodyfat, a more standard approach is probably fine (and desirable). I recommend folks use the simplest approaches they can until those approaches stop working.

First and foremost, if you're a male, you should have no more than 15% bodyfat, female no more than 22% bodyfat. Most likely you want to get leaner while maintaining or even increasing muscle mass. This could be for a bodybuilding contest, for some special event, or simply because you want to see where the body has veins. Alternately, you may want to gain muscle without the accompanying fat gain (or even slight fat loss). Perhaps you're a performance athlete like a powerlifter or an endurance athlete who needs to lean out while maintaining performance. The UD2 can be used for all those goals.

It should go without saying that you have to be exercising for the diet to work. By exercise, that means weight training (I'll talk about endurance athletes separately). Again, if you're new to weight training, the UD2 isn't appropriate; get 3-6 months of training under your belt first. If you're not planning on exercising, this diet will not do you any good. In fact, it'll probably just make you fatter.

You'll need a reasonable (but not insane) amount of diet discipline and you should have a basic understanding of nutrition and diet setup. If you don't know what a protein or carbohydrate is, or how to set up a diet, you're going to be totally lost reading this. I've tried my best to provide all the information you need but I'm going to make some assumptions about basic knowledge. If you meet this rather narrow set of criteria, read on.

Why not just use standard dieting approaches?

You may be wondering why you shouldn't just use one of the myriad standard dieting programs out there. I mean, pick up any bodybuilding magazine, and there are tons of plans that claim to let you achieve everything the UD2 does without all of the hassle. Why is the UD2 superior?

The main problem I have with the standard advice is that it's just so standard. High protein, low to moderate fat, low to moderate carbohydrates, weight training and aerobics is the standard prescription for getting ripped up. If all of the magazines are writing it, it must work, right? Well, yes, up to a point.

Frankly, I have no problem with the standard advice as long as it's producing results. As I said above, I actually prefer simpler approaches as long as they work. In many people, who frequently have genetic advantages that they might not even be aware of, they work just fine. But based on observations at the gym and the feedback I get, not everybody is so lucky (I'll talk about some of the reasons the genetically lucky are lucky next chapter). The reality is, only a small portion of the people who try actually achieve their goals using the standard advice. That tells me that, standard or not, it's not effective.

And don't get me started on the advice given by pro bodybuilders. It shouldn't even be taken into consideration unless you've got the array of steroids, thyroid medications, thermogenics and appetite suppressants that they use to get ready for a contest. A current pro is reported to have said the following about contest dieting "There is no magic diet, buy as many drugs as you can afford and starve yourself for as long as you can stand it."

For the majority, the genetically average (or disadvantaged), any number of problems can stop the diet in its tracks. A metabolically average dieter may lose 1 lb of muscle for every 3 lbs of fat lost trying to get to single digit bodyfat levels. Women have even more problems with muscle loss, not to mention issues with lower bodyfat mobilization. For some, metabolic adaptation causes fat loss to slow or stop completely long before goals are reached. There are all kinds of reasons these problems occur, most of which can be traced to the body's many annoying ways of adapting to a diet. Those same individuals have an equally hard time adding muscle without gaining too much bodyfat at the same time. Fundamentally, this is an issue of partitioning, where the calories are going (or coming from) when you eat (or diet).

What you should expect during the diet

I'll say up front that the UD2 is not an easy diet. You'll have to count/decrease calories and carbohydrates 3-5 days out of every 7. While you don't get to eat everything in sight on the other days, it'll sure seem like it. On some days you can even eat some junk food.

If you use the fat loss variant, you should be losing a pound or more of fat per week, while gaining some muscle. At the very least you'll maintain muscle without loss which can be an improvement for most people. Performance athletes can lean out while maintaining or even increasing performance as well. For the muscle gain variant, it's a little harder to predict. Women, of course, will have slightly smaller changes overall for what should be obvious reasons.

Despite what you may be used to, you'll only be lifting 4 days per week. Each workout should take about an hour or so, with one running maybe an hour and a half. If you can't find 4 hours per week to train consistently, this diet won't do you much good. Cardio is optional for men, but generally necessary for women to lose their lower bodyfat at any decent rate. Still, you shouldn't need a ton of cardio with this diet, not nearly as much as you think anyhow.

There are only one or two required supplements, although there are some that can be genuinely helpful. Beyond that, the diet revolves around basic foods that you can get at any supermarket (I assume that bodybuilders and athletes have no problem with protein powder). While I'll mention drug options to further optimize the diet, they are by no means required.

Chapter 2: Your body hates you

As I'm fond of saying, your body hates you. Actually, that's backwards, your body loves you. It loves you so much that it will do everything in its power to keep you alive even if that means keeping you small(er) and fat(ter). Because to your body, that gives you a better chance of survival. That you want to do something different doesn't matter as far as your body is concerned.

I want you to put this book down and go look in the mirror for a second. Don't get so caught up in throwing most muscular poses that you forget to come back. What you just saw is perhaps the most complicated machine in existence. Over millions of years (or 7 days, depending on your personal cosmology), it has developed ways of adapting to just about anything that you can throw at it.

This most complicated machine, your body, the one that hates you (but really loves you) still thinks that you're living the rather plebeian existence of our ancestors. Our modern lifestyle has only been around for the last couple of thousand years or so, far too short a time for our bodies to adapt. As far as your body is concerned, you might as well be a paleolithic man named Og (no jokes about the mental capacities of athletes, please) living on the plains.

Let's look at the implications of this by trying to see things from your body's perspective. For the most part, your body has one overwhelming goal which is to keep you alive long enough to have children and ensure the survival of your genes. Everything else is pretty secondary to that goal. So what does that mean? Again, a few things.

First it means that your body needs a nice space-efficient way to store scads of energy. That's to get you through the times when there isn't food available (as it frequently wasn't prior to the advent of 7-11). That energy store exists, it's called bodyfat, and your body thinks it's great. Fat is space efficient, easy to store, doesn't take much energy to sustain, and can hold an unlimited number of calories. If your fat cells get full, your body can even make new ones to store more incoming calories. The new fat cells are a lot harder to get rid of then they were to gain, by the way, which is a very good reason not to get too fat in the first place. Bodyfat is truly an ideal way to store energy.

From your body's perspective it looks like this: If food becomes unavailable, the more fat you have, the more likely you are to survive long enough until food becomes available again. In societies with seasonal food availability, being able to store a lot of fat when food was plentiful was the only way to get through the times when it wasn't. The extra fat also helped keep folks warm during the winter. No central heat or Gortex parkas back then.

In many societies people would fatten up in the summer so that they could survive through the winter and repeat for as long as they lived. Now, we just stay in one long fattening cycle (if you're a powerlifter, you can call this a bulking cycle and not feel guilty) without a break.

That's at the root of the modern problem of obesity: constant availability of high calorie, high-sugar, high-fat foods. Decreases in daily activity is the other big part. Though our genetics are the same as they were 10,000+ years ago, our environment has changed drastically.

Lean individuals would have been at a big disadvantage hundreds of thousands of years ago when getting your next meal wasn't as simple as driving down to the local fast-food restaurant. Folks who didn't fatten up wouldn't have survived the food shortages, for the most part, so their genetics usually got weeded out of the pool. This probably isn't true for ethnic groups that lived in areas of the world where food was available year round: those are the ethnic groups that tend to stay lean pretty naturally.

The people who could store fat the best, who were most likely to survive the famines, were the ones who survived and passed on their genetic code down the line to us. In our current society, bodyfat is just a health-risk, not a necessary element to keep us alive for the most part. This fact is clearly shown in the survival times of lean versus obese folks during total starvation. A lean individual may die after 60 days of total starvation while an obese individual may make it for six months or longer. Extreme leanness is generally incompatible with survival if food becomes unavailable. I'll come back to this in a bit.

But what about muscle, that's useful right? You've got to be able to kill stuff to survive and that means muscle. Yes and no. Although it's wonderful to imagine Paleolithic man taking down wild animals with his bare hands like in all the "Tarzan" movies, it's more likely that man used his bigger brain to outfox animals when it came to hunting. Our brains are staggeringly large (relative to our bodyweight) than those of other animals; most likely we used our brains to compensate for relatively less muscle mass.

So while a modicum of muscle was necessary for survival, and our ancestors are thought to have had more muscle than the average American couch potato (which isn't really saying much), excessive muscle mass was probably a liability. Sure, you need enough to get around and get food but anything more than that is basically dead weight. In the wild, with the possible exception of impressing a potential mate, an 18 inch arm wouldn't have been much of a benefit. If anything, it might have slowed down your spear-throwing a bit.

In contrast to fat, muscle requires a lot of energy to build, requires a lot of energy to sustain, and doesn't provide much energy when it is broken down. Even then, your body will happily break it down when you diet. My point is that you run into an equally difficult set of adaptations occurring when you try to push your muscle mass beyond a certain point.

The end result of all of this is that, to your body, which thinks it’s still on the plains eking out an existence, being fat and small are beneficial, because they meant greater survivability. Our physiology reflects this which makes things really suck for folks who want to be bigger and leaner. In short, we're fighting against millions of years of evolution and adaptation to reach our goals of bigger and leaner. Usually, the body wins.

Now, you may be thinking that I'm full of it already, because you can look at any magazine and find many sterling examples of individuals who are both huge and lean. They are called pro bodybuilders. There are a few reasons why the images in the majority of the magazines aren't very relevant to the rest of us. First and foremost, pro bodybuilders (or athletes in general) have better genetics than the rest of us. They are the genetic elite. This isn't some type of personal grouse or whine, simply a statement of fact and reality. If you had their genetics, you wouldn't be reading this book.

If you look at pro bodybuilders in their early stages, they are still typically leaner and bigger than the normal individual. From a physiological standpoint, they probably have higher than average testosterone levels and don't overproduce cortisol. Thyroid levels are probably optimal or close to it, helping to naturally optimize metabolic rate, fat burning and protein synthesis.

They have good skeletal muscle insulin sensitivity and tend to put calories into muscle more effectively (i.e. they partition calories towards muscle instead of fat). They probably have fewer fat cells than most people and that fat is evenly distributed (although even female pros have problems with lower bodyfat). When they diet, they don't have as many problems with metabolic slowdown. Their evenly distributed fat comes off easily and, since they can use fatty acids easily for fuel, they don't lose as much muscle when they diet. All of these factors contribute to their success.

We can contrast that to the average individual who could have any number of potential metabolic defects that prevents them from reaching their desired goals. Testosterone might be on the low side of normal, cortisol production is elevated, thyroid or nervous system output may be low (meaning a lower than optimal metabolic rate). Skeletal muscle insulin sensitivity is low which means that excess calories get pushed towards fat cells more effectively. When these folks diet, the brain tends to overreact, lowering metabolic rate (which probably wasn't optimal to begin with). Fat loss slows to a crawl. Difficulties mobilizing bodyfat, along with problems with testosterone and cortisol, lead to increased muscle loss. I could keep going but you get the idea.

I'm not just telling you this to depress you; consider it more of a reality check to make you aware of what is and isn't possible. My point is that pro bodybuilders (hell, pro athletes of any sort) are the genetic elite. You are not like them and they have advantages naturally that you don't. Most importantly, trying to mimic what they do, or expecting their results, can only lead you down an endless path of frustration.

And then there are drugs

All professional bodybuilders (and most athletes) use drugs. Anyone who says differently is lying or trying to sell you something. Again, this isn't a grouse or whine, but rather a statement of fact. When you introduce the myriad anabolic drugs into the equation, it becomes possible to not only side-step but almost ignore "normal" human physiology. Couple better than average genetics with enough drugs and you get professional bodybuilders. You are not one of them, you will not be one of them. No amount of wishful thinking can change that. Even if you had access to all of their drugs, there's no guarantee you'd get as big; it's likely that one of the genetic advantages that professional bodybuilders have is a high sensitivity to the drugs that they do take.

Anyone who tells you that the various bodybuilding drugs (anabolic steroids, insulin, clenbuterol, etc.) don't work, or aren't necessary to reach a monstrous level of development, is bullshitting you. Usually they have an all-natural supplement or steroid replacement to sell you in the first place (I just have a book). I'd be lying if I told you that anything you'll read in this book could take you to the development level of even the worst pro. It can't. Without both their genetics and their drugs, it simply can't be done. At best proper/meticulous/crafty nutrition and training will let your maximize your own potential and move beyond ordinary. To go above your genetic potential requires drugs. The sooner you come to terms with this, the better off you'll be.

The fact is that drugs, even the relatively simple testosterone, can take you to a level of development otherwise unachievable by any natural training, diet and supplement methods. At even moderate doses, testosterone allows you to sidestep your normal physiology and reach a higher level. It raises the "setpoint" of how much muscle you can carry; it reduces your fat mass at the same time. Once you introduce all the other drugs endemic to pro-bodybuilding, you get a physiology that is unattainable in non-drug using individuals.

Still not convinced? A single example should help to make my point. In natural (read: non drug using) individuals who have dieted down to extremely low bodyfat levels, say 5%, you see a common hormonal pattern. Testosterone levels are typically bottomed out (some studies even find castrate levels, which is why a lot of natural contest bodybuilders can't get their dick hard, not that they have a sex drive in the first place), thyroid levels are bottomed out, IGF-1 levels are bottomed out, sympathetic nervous system output is way down meaning decreased caloric and fat burning, appetite is through the roof, cortisol is through the roof, on and on it goes. This makes good evolutionary sense: at 5% bodyfat, you are starving to death. Your body is turning off every system (metabolic, reproductive, immune, etc.) that it can to keep you alive until you get some food.

Contrast that to a dieting professional bodybuilder. With the choice of the right drugs, he can eliminate pretty much all of the above problems. Anabolic steroids replace natural testosterone, synthetic thyroid replaces what the body is no longer making, injectable insulin, GH, and IGF-1 fix the insulin, GH and IGF-1 problem, clenbuterol replaces sympathetic nervous system output, appetite suppressants can deal with appetite and anti-cortisol drugs deal with the cortisol problem. That's only a partial drug list, by the way.

Getting to the point

The drug using bodybuilder has completely shut the door in the face of his normal physiology while the natural bodybuilder is basically fucked (physiologically speaking). Again, my point in explaining this isn't so much to give you a metaphorical kick in the nuts before we get started; it's to explain the basic realities of the situation. One of the worst things that a natural athlete or bodybuilder can hope to do is to emulate the pros in terms of their results, training or diet. Pro athletes and bodybuilders have at least two major advantages that you don't have: genetic and drugs. Hoping that you can achieve what they achieve or, even worse, trying to use their approach to do it, is destined for failure.

But all is not lost. One of the goals of the UD2 is to mimic, to as great a degree as possible, some of the processes that occur normally in the genetic elite. We may not be able to do it 100%, but we can get in the ballpark and this will improve results. By using specific nutritional and training practices, the occasional supraphysiological level of supplements and even the occasional drug, we can duplicate some of what's going on.

Chapter 3: Why is it so hard (Part 1)

After last chapter, you're thinking one of two things. Either you can't wait to get into the nuts and bolts of this diet, or you're still not convinced. I mean, you can open any of the muscle magazines (or should we call them supplement catalogs?) and see any number of diet plans, training programs, or nutritional supplements all of which promise everything I told you this diet could accomplish. So how come I'm right and they're wrong?

As I said in chapter 1, the biggest problem with the standard advice is simply that it is generally **so** standard. Most bodybuilding writers, nutritionists and gurus are more concerned with maintaining the status quo, maybe refining it a bit, than going out on a limb and suggesting something new and radical. So let's look at the status quo and see why it won't ultimately let you achieve your goal (again, if it did, you wouldn't have gotten or even needed this book).

The standard prescription to do what I've described (more muscle, less bodyfat) is usually a fairly standard moderate to high carb, moderate to high protein, and low to moderate fat diet (depending on the personal philosophy of the magazine and the writer in question). High fiber, clean carbs, lots of quality protein, fats seem to be the most variable although everyone is finally getting onto the healthy fat bandwagon like they should have done years ago. Couple that with weight training and cardio and magic will happen, right? Well, sort of.

Most people remember those wonderful beginner days, when muscle gain and fat loss come without too much effort. It's true, beginners can pull off what seems like a magical body composition transformation without much difficulty. Folks coming back from a layoff or injury, where they typically gain fat and lose muscle, can do this too. Muscle memory allows the muscle to be regained while the fat is coming back off. As people become more advanced, gaining more muscle, or reaching lower bodyfat levels, this trick becomes more and more difficult until the point that it is more or less impossible. You usually end up either having to focus purely on muscle gain (accepting that some fat will come with it) or fat loss (accepting that you'll lose some muscle). Bulking and cutting phases, basically.

I say more or less impossible because there are a lot of inefficient ways of accomplishing it usually involving months and months of teeny-tiny caloric deficits(like 200 calories/day under maintenance) coupled with intensive training to achieve fairly small changes. Personally, I don't have that kind of patience. I've always wanted a faster solution even if it was more complicated.

Even without trying to gain muscle while you're losing fat, simply losing fat without losing muscle is problematic. This is especially true once you try to push the boundaries of normalcy (again, about 12-15% bodyfat in men and 20-22% bodyfat in women). Getting to those levels without muscle loss isn't too much of a problem but getting leaner tends to cause muscle loss at faster and faster rates. The usual advice is to up protein (which only works up to a point) or lose fat so abysmally slowly (0.5 pounds per week) that you go nuts dieting for months on end.

Gaining muscle without putting on too much fat is another, somewhat different problem, although the same advice is usually given. Maybe with less cardio, or slightly different training, but the same nonetheless. Once again, past the beginner stage, lifters find that putting on muscle at any sort of appreciable rate (without drugs anyhow) usually means gaining some bodyfat as well. The ones who don't want to gain any fat are the ones who will tell you that 3-5 lbs of muscle/year is the most you can gain past the beginner stage.

Then there's the folks who want to lean out significantly while gaining muscle (or strength) at the same time. As I said above, this is pretty easy in beginners, folks who are very fat, or those coming back from a layoff. For everybody else, it's more difficult, approaching impossible. For short periods, the UD2 will let you do it.

Defining the problem again

If you go around into any commercial gym, you typically see folks in a lot of different conditions. There are typically some big but fat guys, some small but lean guys, some small and fat guys, and a few big and lean guys. But, unless you belong to a gym with a large bodybuilder contingent, the last group tend to be few and far between. The question is why?

Why is it so difficult to get both big and lean at the same time? It's almost as if the body can do one or the other well, but not both, which really isn't far from the truth. Related to that, why is it so difficult to lose fat and gain muscle at the same time? Or to gain muscle without gaining fat at the same time? Hell, even losing fat without losing muscle is difficult enough. To answer the question of why things are so difficult, let's start simple and move towards more complex explanations, since that will lead us to an understanding of how to solve the problem.

The simplest answer I could give, I've already discussed in the last chapter and in my last book: evolution. To restate it all quickly and simply, 10 million+ years of evolution have left us with genetic propensities and physiologies that want us to stay smallish and fat, because that means better survival. On top of that, once you're past puberty, your body is far more concerned with homeostasis (remaining the same) than with anything else. To one degree or another, it tends to defend your bodyweight and bodyfat percentage at a certain level (which appears to be programmed in the brain). You can change that set point to some degree with training and diet, but your body always strives to maintain the status quo more than anything else.

But both of those are really just statements of the amazingly obvious, without really telling us much. So let's move a level deeper and start to get into the physiology of why accomplishing our goals is so difficult. That will lead us towards the solution.

Doing two things at once

One fundamental problem is that our bodies aren't typically good at doing two things at once, especially when those things are at odds with one another, or have different fundamental requirements.

For example, study after study has shown that combining heavy strength training with heavy endurance training compromises the overall results. Why? Strength training sends the muscle an adaptive signal to become bigger and stronger and more efficient at using glucose for fuel; endurance training send a signal to become more energy efficient (which typically means smaller because smaller muscle fibers can get energy more readily) and use more fat for fuel. End result is that the body can't do both optimally and you get less than stellar results when you try to do both.

How does this apply to losing fat and gaining muscle? In short, they have different (and, in fact, mutually exclusive) requirements. That is, gaining muscle and losing fat require different scenarios in terms of nutrition, hormones, etc. In fact, the specific requirements for gaining muscle are also the reason that you tend to gain fat at the same time. Similarly, the requirements for fat loss are part of the reason (along with your body's adaptations) that you will lose muscle at the same time. I'll discuss this in detail next chapter.

Synthesizing new tissue (whether muscle or fat) requires energy and that energy can't just magically appear. Synthesizing new muscle tissue is especially costly, at least when compared to synthesizing new fat. While it's wonderfully idyllic to think that the calories for muscle growth will magically be generated from burning fat, it rarely happens that way, at least not without powerful repartitioning drugs like clenbuterol. Which makes a rather nice segue into the next chapter.

Chapter 4: Why is it so hard (Part 2)
Partitioning

At a very fundamental level, the problem natural bodybuilders and athletes have is one of partitioning. At its simplest, partitioning refers to where the calories go (into muscle or fat cells) when you eat more of them or come from (from muscle or fat cells) when you eat less of them.

In an ideal universe, every calorie you ate would go to muscle tissue, with none going into fat cells; you'd gain 100% muscle and no fat. In that same ideal universe, every calorie used during dieting would come from fat stores; you'd lose 100% fat and no muscle. Unfortunately, we don't live in an ideal universe.

As I mentioned early in this book, some hapless individuals will lose as much as one pound of muscle for every 2-3 pounds of fat that they lose when they diet. Typically, those same individuals will put on about the same amount of fat and muscle when they gain weight. Thus is the balance of the universe maintained. More genetically advantaged individuals tend to put more calories into muscle (meaning less into fat) when they overeat and pull more calories out of fat cells (and less out of muscle) when they diet. They stay naturally lean and have few problems dieting. Once again, you aren't one of them, or you wouldn't be reading this book.

When talking about calorie partitioning, researchers refer to something called the P-ratio. Essentially, P-ratio represents the amount of protein that is either gained (or lost) during over (or under) feeding. So a low P-ratio when dieting would mean you used very little protein and a lot of fat. A high P-ratio would mean that you used a lot of protein and very little fat. It looks like, for the most part, P-ratio is more or less the same for a given individual: they will gain about same amount of muscle when they overfeed as they lose when they diet. P-ratio can vary between individuals, of course, but for any given person, it appears to be relatively constant.

So what controls P-ratio? As depressing as this is, the majority of of the P-ratio is out of our control; it's mostly genetic. We can control maybe 15-20% of it with how we eat or train. Supraphysiological amounts of certain compounds (supplements) and, of course, drugs, can also affect the P-ratio. Exercise is perhaps the most significant weapon we have in battling with our body and affecting P-ratio.

So what are the main determinants of calorie partitioning? Hormones are crucially important. High testosterone levels tend to have positive partitioning effects (more muscle, less fat) while chronically high levels of cortisol have the opposite effect (less muscle, more fat). Thyroid and nervous system activity affect not only metabolic rate but also fat burning. Thyroid also affects protein synthesis. Optimal levels of these hormones not only mean better fat loss (and less muscle loss) when you diet but better muscular gains (and less fat gain) when you gain weight. Unfortunately, levels of these hormones are basically "set" by our genetics; the only way to change them significantly is with supplements or drugs. Beyond that, there's not a whole lot we can do to control them.

Another factor controlling P-ratio is insulin sensitivity which refers to how well or how poorly a given tissue responds to the hormone insulin. High insulin sensitivity means that a small amount of insulin will generate a large response; insulin resistance indicates that it takes more insulin to cause the same effects to occur.

Now, insulin is a storage hormone, affecting nutrient storage in tissues such as liver, muscle and fat cells. In that same ideal world, we'd have high insulin sensitivity in skeletal muscle (as this would tend to drive more calories into muscle) and poor insulin sensitivity in fat cells (making it harder to store calories there). This is especially true when you're trying to gain muscle.

When you diet, it's actually better to be insulin resistant (note that two of the most effective diet drugs, GH and clenbuterol/ephedrine cause insulin resistance). By limiting the muscle's use of glucose for fuel, insulin resistance not only spares glucose for use by the brain, but also increases the muscles use of fatty acids for fuel.

In addition to hormonal advantages, it's likely that the genetic elite have high skeletal muscle insulin sensitivity. They store tremendous amounts of calories in their muscles, which leaves less to go to fat cells. Their bodies also don't have to release as much insulin in response to food intake.

In contrast, individuals with poor skeletal muscle insulin sensitivity tend to overproduce insulin, don't store calories in muscle well (this is part of why they have trouble getting a pump: poor glycogen storage in muscle cells) and tend to spill calories over to fat cells more effectively.

So what controls insulin sensitivity? As always, there are a host of factors. One is simply genetic, folks can vary 10 fold in their sensitivity to insulin even if everything about them is the same. Another is diet. Diets high in carbohydrates (especially highly refined carbohydrates), saturated fats and low in fiber tend to impair insulin sensitivity. Diets with lowered carbohydrates (or less refined sources), healthier fats (fish oils and monounsaturated fats like olive oil) and higher fiber intakes tend to improve insulin sensitivity.

Another major factor is activity which influences insulin sensitivity in a number of ways. The first is that muscular contraction itself improves insulin sensitivity, facilitating glucose uptake into the cell. Glycogen depletion (remember this, it's important) improves insulin sensitivity as well.

So what else controls the P-ratio? As it turns out, the primary predictor of P-ratio during over- and underfeeding is bodyfat percentage. The more bodyfat you carry, the more fat you tend to lose when you diet (meaning less muscle) and the leaner you are, the less fat you tend to lose (meaning more muscle). The same goes in reverse: naturally lean (but **not** folks who have dieted down) individuals tend to gain more muscle and less fat when they overfeed and fatter individuals tend to gain more fat and less muscle when they overfeed.

The question is why, why does bodyfat percentage have such a profound impact on P-ratio? There are a few easy answers. One is that bodyfat and insulin sensitivity tend to correlate: the fatter you get, the more insulin resistant you tend to get and the leaner you are the more insulin sensitive you tend to be.

A second is that, the fatter you are, the more fatty acids you have available for fuel. In general, when fatty acids are available in large amounts, they get used. This spares both glucose and protein. By extension, the leaner you get, the more problems you tend to have; as it gets harder to mobilize fatty acids, the body has less to use. Since there is less glucose available (because you're dieting) this increases the reliance on amino acids (protein) for fuel. The original Ultimate Diet advocated medium chain triglycerides (a special type of fatty acid that is used more easily for fuel than standard fats) and this can be a good strategy under certain circumstances. I'll mention some other options later on in the book.

But that's not all. It turns out that bodyfat percentage is controlling metabolism to a much greater degree than just by providing fatty acids. Research over the past 10 years or so has identified fat cells as an endocrine tissue in their own right, secreting numerous hormones and proteins that have major effects on other tissues. Perhaps the most important, and certainly the one most talked about is leptin, but that's far from the only one. Tumor necrosis factor-alpha, the various interleukins, adiponectin and other compounds released from fat cells are sending signals to other tissues in the body which affect metabolism.

Without getting into all of the nitpicky details (many of which haven't been worked out yet), I just want to talk a little about leptin (if you read my last book, this will all be familiar ground).

Leptin, the short course

Leptin is a protein released primarily from fat cells although other tissues such as muscle also contribute slightly. Leptin levels primarily correlate with bodyfat percentage, the more fat you have the more leptin you tend to have (note: different depots of fat, visceral versus subcutaneous, show different relationships with leptin). At any given bodyfat percentage, women typically produce 2-3 times as much leptin as men.

In addition to being related to the amount of bodyfat you have, leptin levels are also related to how much you're eating. For example, in response to dieting, leptin levels may drop by 50% within a week (or less) although you obviously haven't lost 50% of your bodyfat. After that initial rapid drop, there is a slower decrease in leptin related to the loss of bodyfat that is occurring. In response to overfeeding, leptin tends to rebound equally quickly (much faster than you're gaining bodyfat). In contrast to what you might think, it looks like leptin production by fat cells is mainly determined by **glucose** availability (you'd think it was fat intake). So whenever you start pulling glucose out of the fat cell (dieting), leptin levels go down; when you drive glucose into fat cells, it goes up.

Basically, leptin represents two different variables: how much bodyfat you're carrying and how much you're eating. That is, it acts as a signal to the rest of your body about your energy stores. I'll come back to this in a second.

Like most hormones in the body, leptin has effects on most tissues in the body. Leptin receptors have been found all over the place, in the liver, skeletal muscle, in immune cells; you name a site in the body and there are probably leptin receptors there. There are also leptin receptors in the brain but I'll come back to that below. For now, let's look at a few of the effects that leptin has on other tissues in the body.

In the liver, leptin tends to reduce insulin secretion from the beta-cells. In skeletal muscle, leptin promotes fat burning and tends to spare glucose (and therefore amino acid use). In fat cells, leptin may promote fat oxidation as well as making the fat cell somewhat insulin resistant. Leptin also affects immune cell function, decreasing leptin impairs the body's ability to mount an immune response. Now you know at least part of the reason you tend to get sick more when you diet. On and on it goes. An entire book could and should be written about leptin.

Leptin and the brain

Now, I want you to think back to the first couple of chapters of this book, where I talked about the evolutionary reasons it's so hard to get extremely lean. To your body, becoming too lean is a very real threat to your survival. From a physiological standpoint, that means that your body needs a way to "know" how much energy you have stored.

As you may have guessed, or known from my last book, leptin is one of the primary signals (along with many others including ghrelin, insulin, peptide YY and other as of yet undiscovered compounds) that signals the brain about how much energy you have stored and how much you're eating.

All of these hormones send an integrated signal to a part of the brain called the hypothalamus that "tell" it what's going on elsewhere in your body. Changes in levels of these hormones causes other changes in various neurochemicals such as neuropeptide-Y (NPY), corticotrophin releasing hormone (CRH), pro-opiomelanocortin (POMC) and several others to occur. These neurochemicals regulate metabolic rate, hunger and appetite, hormones and a host of other processes.

So when you restrict calories, causing changes in all of the hormones and neurochemicals mentioned above, and a number of physiological processes change, mostly for the worse. Levels of thyroid stimulating hormone, leutinizing hormone and follicle stimulating hormone (TSH, LH and FSH respectively) go down. This results in lowered levels of thyroid and testosterone. Levels of growth hormone releasing hormone (GHRH) go down meaning GH output can be impaired.

Sympathetic nervous system activity goes down which, along with the drop in thyroid, has a huge impact on metabolic rate. Cortisol levels go up as does hunger and appetite. You get the idea. What you end up seeing is an all purposes systems crash when you try to take bodyfat to low levels. I should note that these processes are occurring to one degree or another during all diets, they simply become more pronounced at the extreme low levels of bodyfat.

Ideally, the opposite effects should occur when you raise calories. However, for reasons I detailed in my last book, the system is asymmetrical: falling leptin (and changes in all of the other hormones) has a much larger impact on the body's metabolism than raising leptin does (unless you're raising it back to normal). So the body ends up fighting weight loss to a far greater degree than weight gain. Generally speaking, people find that it a lot easier to get fat than to get lean. Of course, there are exceptions, folks who seem to resist obesity (or weight gain altogether). Research will probably find that they are extremely sensitive to the effects of leptin (and other hormones), so when calories go up, they simply burn off the excess calories without getting fat.

Most of us aren't that lucky. Rather, like insulin sensitivity discussed above, researchers will probably find that leptin sensitivity is a huge factor influencing how changes in caloric intake affect metabolism. Someone with good leptin sensitivity will tend to stay naturally lean and have an easy time dieting; folks with worse leptin sensitivity (leptin resistance) won't.

You might be thinking that the quick and dirty solution would be leptin injections. As I pointed out in my last book, injectable leptin is a pipe-dream at this point, an effective dose costing nearly $1000/day (not to mention requiring twice daily injections). Using bromocriptine or other dopamine agonists seem to fix at least part of the problem by sending a false signal to the brain by making it think leptin levels are normal.

Recent studies that have given injectable leptin to dieters show that the fall in leptin is one of the primary signals in initiating the adaptation to dieting. However, unlike in rats, injecting leptin into humans doesn't fix all of the problems.

This is because, in humans, there is more of an integrated response to both over and underfeeding. To understand this better, I want to take a snapshot of what happens when you either reduce or increase calories.

Dieting

So you decide to diet, reducing carbs, calories or both. Vary rapidly, blood glucose and insulin levels are going to be reduced. This is good as it releases the "block" on fat mobilization. Additionally, catecholamine release typically goes up (at least initially), further increasing fat mobilization from fat cells. This causes blood fatty acid levels to increase. This is also good, as it tends to promote fat burning in tissues such as liver and muscle. This effect is facilitated if you deplete liver and muscle glycogen, as glycogen depletion tends to increase the use of fatty acids for fuel. The increase in blood fatty acid levels also has the short-term effect of causing insulin resistance. As I mentioned, this is a good thing on a diet since it spares glucose and helps promote fat oxidation. So far, so good, right?

Unfortunately, along with these good effects, a lot of bad things start to happen. I already described many of the central adaptations above: changes in leptin, ghrelin, Peptide YY (and certainly other hormones) "tell" your brain that you're not eating enough. This causes changes in the various neurochemicals stimulating a number of negative adaptations. I want to note that the response is not immediate, there is a lag time between the changes in all of these hormones and the body's response. But that's not all.

There are also many other adaptations which occur when you diet, so let's look at some of those. First and foremost, the drop in leptin directly affects liver, skeletal muscle and fat cell metabolism, mostly for the worse.

While the drop in insulin mentioned above causes better fat mobilization, it causes other problems. One is that testosterone will bind to sex-hormone binding globulin (SHBG) better, lowering free testosterone levels (this is in addition to the drop in total testosterone). As well, insulin is anti-catabolic to muscle, inhibiting muscle breakdown. The increase in cortisol that occurs with dieting enhances protein breakdown as well as stimulating the conversion of protein to glucose in the liver. Additionally, a fall in energy state of the muscle impairs protein synthesis (although it increases fatty acid oxidation). The mechanism behind this is more detail than I want to get into here. But the combined effect of these processes is that protein synthesis is decreased and breakdown is accelerated; this causes muscle loss.

On top of that, high blood fatty acid levels tend to impair the uptake of T4 (inactive thyroid) into the liver. There are also changes in liver metabolism that impair the conversion of T4 to T3 (active thyroid). Both of these processes cause decreased blood levels of T3. There is some evidence that high blood fatty acid levels causes tissues to become resistant to thyroid hormone itself (this is part of why just taking extra thyroid on a diet doesn't fix all of the problems). After the initial increase, there is also a drop in nervous system output (that can occur in as little as 3-4 days after you start a diet). Along with the drop in thyroid, insulin and leptin, this explains a majority of the metabolic slowdown that occurs. The change in liver metabolism (and the reduction in insulin) also impairs the production of IGF-1 from GH.

All of these adaptations serve two main purposes. The first is to slow the rate of fat loss, as this will ensure your survival as long as possible. Related to that, the body tends to shut down calorically costly activities. This includes protein synthesis, reproduction and immune function; there's little point keeping any of these functioning when you're starving to death. The drop in leptin, and the changes in hormones that occur are a huge part of why men tend to lose their sex drive (and ability) and women lose their period when they get lean/diet hard.

The second is to prime your body to put fat back on at an accelerated rate when calories become available again. Decreased metabolic rate and fat burning, along with improved caloric storage all conspire to put the fat back on when you start eating again. As I mentioned earlier, this makes perfect evolutionary sense, even if it presents a huge pain in the ass to us.

I haven't even mentioned the hunger and appetite issue which is a topic worthy of an entire book. The combination signal sent by leptin, ghrelin, insulin, glucose, and a host of other hormones (cholecystokinin, glucagon-like peptide 1 and 2, bombesin and many many others) are all involved in both hunger and appetite. The changes that occur with dieting tend to make both shoot through the roof: you tend to get and stay hungry, thinking about food nearly constantly. Bodybuilders and athletes may have unbelievable food control but it still sucks being hungry constantly when you try to diet.

Ok, enough about dieting, what about overfeeding?

Overfeeding

To a great degree, most of the adaptations that occur with dieting reverse when you overeat. Actually, that depends a lot on the situation. As I mentioned above, the body as a whole tends to defend against underfeeding better than it does against overfeeding which is why it's generally easier to gain weight than to lose it. Studies where leptin has been increased above normal (i.e. to try and cause weight loss in overweight individuals) have generally borne this out: except at massive doses, raising leptin above normal does very little.

There are a couple of theories as to why this might be the case. One theory is that normal leptin levels send essentially a 100% signal, that is they tell the body that all systems are normal. It should seem clear that raising leptin above 100% isn't going to do much. Another possibility is related to something I alluded to above: leptin sensitivity and resistance. It's thought that people have varying degrees of leptin resistance which means, in essence, that they don't respond as well to leptin as they should. On top of this, when leptin levels go up, it appears to stimulate resistance to itself. That is, when leptin gets and stays high, it causes you to become resistant to its effects.

Both explanations for the failure of high leptin levels to defend against weight gain make good evolutionary sense. Your body doesn't want to be lean but it doesn't really mind getting fat. This is because, during our evolution, being fat was never a risk, while being lean was. If anything, getting fat was a benefit which is why our bodies tend to be so good at it. It's only in modern times when people can get and, more importantly, stay fat for extended periods, that being fat is a problem. Ten thousand years from now, perhaps we will evolve defenses against being fat.

Anyhow, if calories are available all the time, it would make little sense for you to get full and/or start burning them off. This is what would happen if you were extremely sensitive to leptin (and does happen in a small percentage of individuals). So high levels of leptin induce resistance to itself, keeping you hungry and eating while the food is available. Leptin can induce resistance to itself in only a few days of overeating.

But we're not really talking about raising leptin above normal here, we're talking about reversing or preventing the drop that occurs with dieting. In that situation, many of the above adaptations to dieting will reverse to one degree or another. What degree will depend on how lean you are, how long you diet, and how long you overeat.

So now you increase your calories and carbs. Let's look at some of what happens when you do so. First there are all of the central adaptations that occurred during dieting, that will reverse to some degree while overfeeding. Leptin will go up (noting again that it goes up more quickly than bodyfat comes on) along with insulin and peptide YY, ghrelin goes down. This signals the hypothalamus that you're eating again signaling that the body can reverse the adaptations that had occurred in the first place.

In addition, the increase in insulin will reverse the binding of testosterone to SHBG; cortisol also goes down. With increased carbohydrates, you increase both liver and muscle glycogen. While this decreases fat oxidation in the muscle, you get improved protein synthesis (the increase in insulin and testosterone and the drop in cortisol have an additional effect).

Of course, with increasing insulin, there is a decrease in blood fatty acid concentrations which improves insulin sensitivity. Skeletal muscle insulin sensitivity is enhanced even more by exercise.

The decrease in blood fatty acids, along with changes in liver metabolism will improve both the uptake and conversion of T4 to T3; along with improvements in nervous system output, this will help to increase metabolism. You get the idea: with overfeeding, the body reverses the basic adaptations that occurred to dieting in the first place.

Summing up for now

Looking at the chart on the next page, you may start to appreciate the problems involved, especially for the genetically normal. Underfeeding is necessary for fat loss but will always have a negative impact on muscle mass. Dieting also induces a number of adaptations that tend to prevent further fat loss. Overfeeding is necessary to gain muscle but will always have a negative impact on fat mass. However, it can reverse many of the adaptations that occur with dieting.

	Overfeeding	Underfeeding
Calories	Up	Down
Protein	Up	No change or Up
Carbs/fat (energy)	Up	Down
Insulin	Up	Down
Total testosterone	Up or no change	Down
Free testosterone	Up	Down
GH	Up	Up
IGF-1	Up	Down
Thyroid	Up	Down
Catecholamines	Down	Up
Cortisol	Down	Up
Leptin	Up	Down
Ghrelin	Down	Up
Cellular energy state	Up	Down
Protein synthesis	Up	Down
Bodyfat levels	Up	Down
Muscle mass	Up	Down
Net effect	Body is systemically anabolic	Body is systemically catabolic

A final note on leptin

Hopefully the above sections have made you realize that there is far more to the adaptations to either dieting or overfeeding than just leptin. Rather, there is an integrated response involving leptin, insulin, ghrelin, fatty acids, liver, fat cell and skeletal muscle adaptations, and probably factors that haven't been discovered yet. This probably explains why injecting leptin into dieting humans reverses only some but not all of the adaptations to dieting.

For example, just injecting leptin would be expected to fix a defect in TSH (and thyroid output) and it does do this. But injectable leptin won't fix the problems with conversion that occur at the liver. Similarly, while injecting leptin would normalize LH and FSH output, it won't correct the problem with increased binding of testosterone to SHBG caused by lowered insulin. Hopefully you get the picture. Now we know the problem. What's the solution?

Cyclical dieting

What I've basically done over the previous pages is make a long-winded argument for cyclical dieting, that is periods where you alternate a low-calorie intake with a high-calorie intake. More specifically I'm describing a diet where you alternate between periods of low calories/carbs with periods of high calories/carbs to alternate between periods of anabolism (tissue building) and catabolism (tissue breakdown). Of course, this is nothing new.

There have been numerous other schemes over the years that alternated periods of low and high calories. DiPasquale's Anabolic Diet, Rob Faigan's NHE and many others have come and gone over the years.

Several years ago, when I first started making some of the connections between leptin and everything else, this really pointed out the need to do periodic refeeds (or cheat days or whatever you want to call them) on a diet. I think it explains part of why people got better results with the Bodyopus diet: it wasn't the ketogenic phase so much as the two day carb-load which refilled muscle glycogen, maybe instilled an anabolic response, and helped to reverse some of the adaptations inherent to dieting.

Since the Bodyopus days, a number of approaches have come and gone. In general, short refeeds, lasting from 5 to 24 hours done anywhere from once per week to every other day (depending on such variables as bodyfat percentage and how hard you're dieting) while dieting have been used. I've tried them all with varying degrees of success.

One of the factors I've been considering lately has to do with the duration of the overfeeding period. While it's true that 5 (or 12 or 24) hours of concentrated overfeeding will raise leptin, the more important question is whether that's sufficient to "tell" the brain that you're fed. While data (especially in humans) is nonexistent, my hunch is that it is not.

My basic reasoning is this: there's a lag time of several days between the drop in leptin and the drop in metabolic rate (nervous system output) for example; I'd be surprised if a mere 12 or 24 hours was sufficient to reverse this. Rather, I'd expect it to take a similar amount of time for the reversal to occur. The more extended logic of my reasoning is beyond what I want to put in this book, email me if you must know.

Now, this isn't to say that short carb-loads/refeeds aren't of benefit. They refill glycogen, turn off catabolism briefly and maybe induce an anabolic response to boot. They also let you eat some of the crap you're really craving which helps psychologically. But I doubt they are sufficient to affect metabolism very much. Instead, a longer refeed is most likely necessary. The drawback, of course, is that longer refeeds have a tendency to put too much bodyfat back on which goes against the entire goal of dieting.

Perhaps the biggest problem with many cyclical dieting approaches is that they don't coordinate training with the diet. Bodyopus was an exception but, for various reasons, I think the workout plan was screwy. If anything, it was backwards, putting tension workouts on low-calorie/low-carb days (where you aren't very anabolic) and glycogen depletion workouts before you are eating a lot. This seemed wrong to me years ago and more wrong to me now that I've delved into it in more detail. This will make more sense as you read the next chapters.

Ultimately, all of this introductory stuff, brings us to the final question: how do we optimize the entire system to maximize fat loss and either muscle maintenance or muscle gain (or, if you're a performance athlete, how do we generate fat loss while maintaining performance)? To understand that, I need to get into a few more details regarding muscle gain and fat loss, which will help you to understand the overall system.

Chapter 5: Fat basics

Ask most people how to lose fat and you'll usually get a pretty generic answer: eat less and exercise. I've made comments along those lines myself. Fundamentally, there's not much wrong with that particular sound bite, at least within a certain range of bodyfat percentages. Basically, if you create a deficit between the energy your body needs and what it has available and at least some fat will be mobilized to be used for energy. How much is used and where it comes from depends on more than just simply reducing calories. That's the partitioning issue I talked about last chapter.

What we're really interested in, more so than just calorie balance, is fat balance. Losing fat requires a negative fat balance. A negative fat balance simply means that you must be burning more fat than you're eating (or producing); we want to try and optimize this process to maximize the rate of fat loss. It is possible, under the right conditions for short periods of time, to be losing fat while eating above maintenance calories, as long as you maintain a negative fat balance.

Now, as I've mentioned in previous chapters, genetic superiors seem to be able to mobilize and utilize fatty acids more effectively than the rest of us. This has a protein sparing effect because, the more you can force your body to rely on fatty acids for fuel, the less it needs to use glucose or amino acids. So is there any way we can mimic this in the non-genetically elite? To answer this question, we need to look at some of the details of fat, both dietary and bodyfat.

Dietary fat

Let's start with dietary fat, the fat that's found in your food. Dietary fat is technically a triglyceride, which is a molecule of glycerol (a sugar) bound to three fatty acids. All fatty acids are not the same, though. They can differ in length from short to medium to long chain, depending on how many carbons are present. They also differ in the number of double bonds which are present. Saturated fats (found mainly in animal foods) have no double bonds, monounsaturated fats (olive oil) have a single double bond, and polyunsaturated fats (fish oils, flax oil) have multiple double bonds. Chemical differences in fat can affect metabolism and physiology.

Unlike proteins and carbohydrates, dietary fats aren't water soluble, they have to be digested in the stomach with the help of bile acids released from the pancreas. After a good bit of processing (the details of which are unimportant here), most dietary fats are packaged as chylomicrons which enter the lymphatic system. In contrast, carbohydrates and protein go to the liver, via the portal system, after digestion and absorption.

After about 3 hours, these chylomicrons will reach the fat cells. There, they are acted upon by an enzyme called lipoprotein lipase (LPL), which liberates the fatty acids from the chylomicron. Those fatty acids can either be stored in the fat cell or taken up into the bloodstream for use by other tissues such as muscle and liver. Whether they are stored or released depends on the metabolic state of the dieter. The main point is that dietary fat isn't going to be readily available by tissues such as muscle and liver, even under the best of circumstances. At best it might become available 3 hours after ingestion.

But remember from above, one of our goals is to make fatty acids as readily available to the body as possible. Is there any way to increase the rate at which dietary fat is available? The answer is yes, having to do with two weird exceptions to the above.

The first exception, and probably the compound most readers have at least heard of, are medium chain triglycerides (MCTs). Unlike longer chain fats, MCTs go the liver and are available for use far more rapidly by other tissue. In addition, MCTs are preferentially used to produce ketone bodies which can be used instead of glucose, amino acids or fatty acids by most tissues of the body. In some studies this has a protein sparing effect and this is especially true in the initial periods on a low-calorie low-carbohydrate diet.

A second potential exception is the newly available diacylglycerol (DAG) oil put out by a company called ENOVA (http://www.enova.com). Unlike triglycerides, DAG oil is made available to the body more rapidly than long-chain fats. Using either MCT or DAG oil should allow calories to be reduced to lower than normal levels without causing as much muscle loss.

So what about body fat?

When people talk about body fat, what they mean are triglycerides which are stored in your fat cells (there is also some stored in your muscles, called intramuscular triglyceride, but it is a tiny amount compared to what's stored on your fat butt or stomach). An average individual may have 30 billion fat cells which are composed of about 90% triglyceride stored as one big droplet. The remaining 10% is water and the enzymatic machinery which controls cellular metabolism.

Now, not all fat cells on your body are the same. Researchers have identified at least 4 different "types" of bodyfat, although we can actually make at least one further distinction.

First is essential bodyfat, which exists in small quantities (about 3% of the total in men, and 9-12% of the total in women) in the brain, spinal cord, etc. You can't lose it, and if you did you'd be dead. The amount of essential bodyfat sets the ultimate lower limit for bodyfat percentage. So that should be 3% for men and 9% for women. I should note that you will occasionally see claims of bodyfat percentages less than 3% for men or less than 9% for women. It's not that the folks are lying, so much as the fact that the measurement methods being used aren't as accurate as they need (or are claimed) to be. We don't need to worry about essential fat in this book. Like I said, you can't lose it and even if you could, you'd be dead.

A second type of fat is brown fat which is a specialized type of fat that actually burns up the other types of fat, producing heat in the process. In contrast to white fat (all the other types), which is primarily triglyceride with a little bit of other stuff, brown fat is made up mainly of mitochondria (the powerhouse of the cell), with very little triglyceride. The high mitochondrial content makes brown fat ideal for burning fatty acids for heat. The problem is, while animals have a lot of brown fat (they need it to keep body temperature up against the cold and such), humans lose most of their brown fat once they move past the baby stage of life. We can pretty much ignore it for the rest of this book. You should also question any supplements that claim to cause fat loss via brown fat activation; it will probably work wonderfully in your pet hamster or mouse but that's about it.

Next up is visceral fat. This is a type of fat that surrounds your internal organs. In excess it gives you a pregnant look because it makes your gut stick out. Visceral fat has a number of different characteristics from subcutaneous fat (which I'll discuss next chapter) which has some consequences for both health and dieting. Men tend to have more visceral fat than women as testosterone and cortisol tends to promote its growth. Women who use anabolic steroids, or who have higher than normal testosterone for whatever reason, tend to accumulate visceral fat as well. By the time men reach the 12-15% bodyfat range, it is unlikely that they will carry much visceral fat unless they have been using androgens.

The type of fat most dieters are concerned with is subcutaneous fat which is found under the skin. In men, subcutaneous fat tends to accumulate around the midsection and low-back; in women, it tends to be on the hips and thighs. This occurs under the influence of the hormones testosterone/cortisol and estrogen/progesterone in men and women respectively. This is why kids before puberty have the same bodyfat patterns and kids after don't.

In fact, when researchers pump sex-change patients up with hormones, they see a shift in bodyfat: men take on female bodyfat patterns and vice versa. Women who don't go on hormone replacement after menopause (meaning they produce no estrogen) tend to lose the fat in their hips and thighs and gain it in their stomach area. As I've mentioned, some lucky individuals have more even fat distributions and still look ok even when they're carrying a lot of fat; they are simply smooth all over. If you're reading this book, odds are you aren't one of them.

We (not the researchers) can subdivide subcutaneous fat into two types: normal and stubborn. Normal fat is just normal subcutaneous fat that comes off without too much effort. A little calorie cutting, a little cardio, and it comes off without too much trouble. Stubborn fat is the other kind, that stuff that goes on first, and comes off last, if it ever comes off at all. Stubborn fat is generally ab and low back fat for men and hip and thigh fat for women. There are reasons that stubborn fat is so stubborn that you'll learn about soon.

Now that you know the basics of both dietary fat metabolism and bodyfat, let's get into the details of how fat is burned off and how we can optimize the process.

Chapter 6: Fat cell metabolism

The ultimate goal of a diet is to lose bodyfat of course so let's look at the processes controlling that. That means examining the steps involved in mobilizing fat from fat cells and burning them off.

First, let me elaborate on what it means to lose or "burn" bodyfat. What this means is that the fat stored in your fat cells is removed from those cells and converted to energy elsewhere in the body. Most tissues in the body (there are a few exceptions such as the brain) can use fatty acids for fuel, but the main ones we are interested in are skeletal muscle and the liver. I want to mention that even though the brain can't use fatty acids directly, it can use ketones which are made from fatty acid metabolism in the liver.

Let's look at the mechanisms underlying the process of fat loss. Although the process can be further subdivided, we are only interested in three major steps of fatty acid metabolism: mobilization, transport, and oxidation (burning).

Step 1: Mobilization

The first step in burning off bodyfat is getting it out of your fat cells. You might even argue that this is the most important step since, if you can't get it out of the fat cell, you can't burn it off.

Recall from last chapter that bodyfat is primarily stored triglyceride, with a small amount of water and some enzymatic and cellular machinery. Mobilizing bodyfat requires that we first break down the stored triglyceride into three fatty acids and a molecule of glycerol. The rate limiting step in this process is an enzyme called hormone sensitive lipase (HSL).

So what regulates HSL? Although a number of hormones such as testosterone, cortisol, estrogen, and growth hormone have modulating effects on HSL activity (mainly increasing or decreasing total **levels** of HSL in the fat cell), the only hormones that we need to be concerned with in terms of HSL **activity** are insulin and the catecholamines.

The primary inactivator of HSL is the hormone insulin and it only takes very tiny amounts (depending on insulin sensitivity) to have an effect. Even fasting insulin levels are sufficient to inactivate HSL by nearly 50%. Small increases in insulin (from either protein or carbohydrate intake) inactivate HSL further. Additionally, the mere presence of triglycerides in the bloodstream (via infusion or by just eating dietary fat by itself) also inhibits HSL activity so this isn't as simple as just blaming insulin. One way or another, any time you eat, HSL is going to be inactivated, either by the increase in insulin from protein or carbs or the presence of fat in the bloodstream from eating fat.

The primary hormones which activate HSL are the catecholamines: adrenaline and noradrenaline. Adrenaline is released from the adrenal cortex, traveling through the bloodstream to affect numerous tissues in the body. This means that blood flow to fat cells has an impact on how much or how little adrenaline will reach fat cells. Noradrenaline is released from nerve terminals which interact directly with the cells.

More technically, both insulin and the catecholamines affect levels of cyclical AMP (cAMP) in the fat cell which is what really determines how active HSL is. When cAMP levels are low, HSL activity is also low and fat breakdown is low. When cAMP levels are high, HSL activity is high and fat breakdown increases.

Insulin lowers levels of cAMP and the catecholamines, in general, raise levels of cAMP (I'll explain this statement in a second). The higher the level of cAMP, the more active HSL is and the more bodyfat that gets broken down and released from the fat cell. It should be clear that, from a fat loss standpoint, we want high levels of cAMP.

A tangent: all about adrenoreceptors

To understand some of the cryptic remarks above, I need to back up a bit and explain how the catecholamines send their signals. All hormones work through specific receptors and the catecholamines are no different, they have their own specific receptors called adrenoreceptors.

There are two major classes of adrenoreceptors: beta and alpha, which are found all over the body. This includes the brain, liver, skeletal muscle, fat cells, heart, blood vessels, etc.; you name it and there are probably adrenoreceptors there.

Now, there are at least 3 (and maybe 4) different beta receptors called, imaginatively: beta-1, beta-2, beta-3, and beta-4 (or the atypical beta-3). Alpha-adrenoreceptors come in at least two flavors, alpha-1 and alpha-2. There are additional subtypes of each adrenoreceptor but this is more detail than we really need. Tangentially, beta-3 receptors (and drugs called beta-3 agonists) became a huge research project when it was found that beta-3 activation caused major fat loss in animals; it was hoped that the drugs would work in humans as well. Unfortunately, beta-3 receptors are found primarily on brown fat cells which, as I said, animals tend to have lots of and humans don't.

The main receptors we need to worry about in human fat cells are alpha-2 receptors and beta-1 and beta-2 receptors, both of which actively bind the catecholamine hormones. When catecholamines bind to beta-1,2 receptors, they increase cAMP levels, which increases fat breakdown. Great. However, when the catecholamines bind alpha-2 receptors, they decrease cAMP levels which decreases fat breakdown. Not great. But it means that catecholamines, which I told you were fat mobilizers, can actually send both fat mobilizing and anti-fat mobilizing signals: by binding to either alpha- or beta-receptors.

So why does this matter? Different areas of bodyfat have different distributions of alpha-2 and beta-2 adrenoreceptors. For example, women's lower bodyfat (hips and thighs) have been found to have 9 times as many alpha-2 receptors as beta-2 receptors. Some research indicates that men's abdominal fat is similar, with more alpha-2 than beta-2 receptors. Now you know part of why its so difficult to reduce these stubborn fat areas; with a greater number of alpha-2 receptors to bind catecholamines, it's that much more difficult to stimulate fat breakdown in those fat cells.

Other factors affect adrenoreceptor function as well. Androgens and thyroid tend to increase the sensitivity of beta-2 receptors to the catecholamines. This may be part of why men (who have higher androgens and higher thyroid, on average) lose fat more easily. The factors controlling alpha-2 adrenoreceptor function aren't as well elucidated.

Back to mobilization: summing up

I should note that insulin pretty much always wins the battle over fat cell metabolism. That is, even in the face of high catecholamine levels, if insulin is elevated, fat mobilization will be impaired. As it turns out, this generally doesn't happen under normal conditions. Typically when insulin is high, the catecholamines are low and vice versa (e.g. during exercise, insulin levels drop as catecholamine levels go up). There are exceptions of course. If you drink a carb drink during aerobic exercise, for example, the slight increase in insulin will decrease fat mobilization despite high levels of catecholamines.

Just remember the following: insulin inhibits fat mobilization and the catecholamines (generally) increase it. Insulin always wins the battle. So when insulin is high and the catecholamines are low, fat tends to be stored. When insulin is low and the catecholamines are high, fat will be mobilized. A bit simplistic? Perhaps. But good enough for the time being. The real take home message is that, from a fat mobilization standpoint, we want low insulin and high catecholamine levels. Both can be readily accomplished by altering diet (lowering carbohydrates and calories) and exercise (which increases catecholamines).

Step 2: Blood flow and transport

So imagine a situation where insulin is low and the catecholamines are high, causing stored triglyceride to be broken down (the technical word is hydrolyzed) to glycerol and free fatty acids (FFAs). Both enter the micro-circulation around the fat cells. The glycerol can be used for a lot of different things, including glucose production in the liver, but we can ignore it for now. The FFAs are what we're interested in.

Some of the FFA will simply get stored back in the fat cell (a process called re-esterification). What doesn't get restored may either sit in the bloodstream as a free fatty acid or bind to albumin (a protein made in the liver). So now we have albumin-bound FFAs sitting in the circulation surrounding the fat cell Since the FFA can't be burned there, it has to be transported away from the fat cell; this depends on blood flow to and from the fat cell.

As with insulin sensitivity and adrenoreceptor ratios, fat depots differ in terms of blood flow. Visceral fat, for example, has an extremely high blood flow relative to other fat depots. This is on top of being extremely sensitive to the catecholamines, and relatively resistant to the effects of insulin. Visceral fat is mobilized fairly easily and, because of this, it generally goes away the fastest (especially with exercise).

Relative to visceral fat, abdominal (and probably low-back) fat has less blood flow, is less sensitive to the fat mobilizing effects of the catecholamines, and more sensitive to insulin. This makes it more stubborn than visceral fat. Hip and thigh fat is, by far, the worst; it has the lowest blood flow, is the least sensitive to the catecholamines and the most sensitive to insulin.

So now we have yet another reason that stubborn fat is stubborn: poor blood flow which makes transporting the mobilized fatty acids away more difficult. Actually, it isn't entirely true that blood flow to stubborn fat cells is always slow. In response to a meal, blood flow to stubborn fat increases readily; at all other times, blood flow to stubborn fat is slow. Basically, it's easier to store calorie in stubborn fat than to get it back out.

Studies show that women tend to have preferential increases in blood flow to their hips and thighs after a meal; the old wives' tale about fatty foods going straight to the hips turns out to be true after all. Men tend to send more to visceral fat (which is actually easy to mobilize) and more of it sits around in their bloodstream; this makes it easier to lose bodyfat but is one reason men are more prone to heart attacks.

But the point is made, poor blood flow to stubborn fat cells is yet another reason dieting to sub-average bodyfat levels is difficult. So how might we improve blood flow to and from fat cells? Blood flow to fat cells improves during fasting and, although we can't fast completely (too much muscle loss), we can mimic the condition with a low-carbohydrate/ketogenic diet. This fits in with our goal of lowering insulin in the first place and turns out to have an extra advantage that I'll discuss in a later chapter.

As it turns out, thyroid levels affect blood flow to fat cells significantly. Low thyroid (which is common among women and, I suspect, among genetically average men) decreases blood flow to fat cells and normal or even high thyroid levels improve it. Short of using thyroid medication (a replacement dose of perhaps 25-100 mcg), there's not much we can do here.

However, aerobic exercise improves blood flow to fat cells in addition to burning calories, so that's a possible solution. Some studies show that exercise can overcome the normally low blood flow. Considering their problems with lower bodyfat, this might explain the observation that female bodybuilders need to do more cardio than men to get ripped.

There are also alpha- and beta-adrenoreceptors on the walls of the circulatory system which control whether or not the blood vessels constrict (slowing blood flow) or dilate (increasing it). As with fat cell metabolism itself, alpha-adrenoreceptors tend to decrease adipose tissue blood flow while beta-adrenoreceptors tend to increase it. Other hormones such as nitric oxide, prostaglandins and adenosine also affect adipose tissue blood flow and it appears that the fat cell regulates its own blood flow to a great degree. In general, if you can get fat breakdown to occur, various hormones which are produced tend to increase blood flow (to get the FFAs away from the fat cell).

Step 3: Uptake and utilization

So now we've mobilized fatty acids into the bloodstream, bound them to albumin and managed to get them away from the fat cell and into the general circulation. What's next? Eventually, the albumin-bound FFA will run into a tissue (such as the liver or muscle) which can use it for fuel. The FFA will be taken up into that tissue (apparently by a specific fatty acid binding protein) at this time for one of a couple of fates. In both liver and muscle, the FFA can either be re-stored as triglyceride (which is unusual under normal dieting conditions but occurs during overfeeding) or burned for energy. We'll focus only on the latter.

To be used for energy, the FFA has to be transported into the mitochondria by an enzyme called carnitine palmityl transferase (CPT). Incidentally, this is the theory behind carnitine supplements; by increasing levels of CPT, you get more fat burning. While this is great in theory, it doesn't really work in practice (if it does, the amounts needed are absurdly high and expensive). CPT activity is controlled by a few different factors, including your aerobic capacity (the more aerobically fit you are, the more fat you burn), as well as glycogen levels.

Glycogen is simply a long carbohydrate chain stored in your muscles or liver. When glycogen is high, CPT activity is low and fat burning is low, and vice versa. This is true for both muscle and liver. By depleting muscle and liver glycogen, we can increase CPT activity, allowing us to burn off the fatty acids at a faster rate. This is readily accomplished with the combination of lowered carbohydrates and intensive training which fits in with our other goals rather nicely anyhow.

Chapter 7: Muscle growth part 1

Everyone reading this knows what muscle is, right? Well, maybe. Technically speaking, your body has three different types of muscle: skeletal or striated muscle, smooth muscle, and cardiac muscle. Smooth muscle is found in your arteries, blood vessels and such, and cardiac muscle is found in your heart. Bodybuilders and athletes are concerned with skeletal or striated muscle and I'll focus on that exclusively.

Skeletal muscle is composed of a number of different components. This includes the actual contractile muscle fibers (made up of protein) as well as a lot of other stuff. The other stuff is basically the support system for the muscle fibers and includes glycogen (stored carbohydrate), water, minerals, creatine phosphate, mitochondria (for energy production), capillaries, a small amount of fat in the form of intramuscular triglycerides and others.

With that said, let's dismiss a common myth which is that muscle is primarily protein. In fact, skeletal muscle is only about 25% protein, and about 70% water. Even the glycogen and the rest only makes up a small percentage of the total weight. In one kilogram (2.2 lbs) of muscle, you have maybe 100 grams (about 0.05 lbs) of protein or so. I'm honestly surprised that no one has ever pushed water as an anabolic since, strictly speaking, it makes up far more of your muscle volume than protein does.

You've probably read somewhere that muscle fibers come in a couple of different types. You may have seen the simple names of slow and fast fibers, or red and white fiber (red is slow, white is fast) or the more technically accurate Type I, Type IIa, Type IIb (or just Type I and Type II). Some researchers delineate even more fiber types such as IIc, IIx, IIcx and even more than that. Ultimately, all of these different nomenclatures refer to the physiological characteristics of the muscle fibers and that's what's most important here.

Type I (or slow twitch or red) fibers fire somewhat more slowly than fast fibers, don't fatigue very quickly, and don't grow very much. They are used mostly for endurance type activities. Type II (or fast twitch or white fiber) contract a little bit more quickly than slow fibers, fatigue quickly, and have a large capacity for growth. They only come into play when high force outputs are necessary, such as during lifting weights, sprinting, or what have you. There are further subdivisions within Type II fibers, which describe variance in force production, fatigueability, growth potential, etc., but that's more detail than we really need.

Now, let's dismiss another pervasive myth: that slow movements only fire slow fibers and fast movements are required to fire fast fibers. Slow and fast are relative terms here, which refer to how quickly the fibers can generate force. To give you an idea, a slow fiber will generate maximum force in 100 milliseconds (that's 0.1 second), a fast fiber in about 25-50 milliseconds (that's 0.05 seconds). Even with the fastest movements, you can't approach those kinds of speeds.

Rather, which types of fibers you recruit during weight training (or other activities) depends on how much force your muscles need to generate. With low force requirements, you recruit Type I fibers, with more and more Type II fibers being recruited as your force requirements go up. A near maximum load, even though it may move slowly, will fire all available muscle fibers. A light weight moved quickly, which may require a high force output, can fire fast twitch fibers as well. As well, if you start with a light weight moved slowly, as some fibers fatigue, you will progressively recruit more fibers throughout the set.

The same holds true for endurance activity, by the way. At low intensities, you use almost exclusively Type I fibers. As intensity (speed) increases, you start recruiting Type IIa fibers, as your approach maximum power outputs, Type IIb fibers will be recruited.

So let's talk about muscle growth since that's what we're really interested in. Muscle growth is most generally referred to as hypertrophy, which refers to an increase in size of your muscle fibers. You may have seen a related term, hyperplasia, which refers to the splitting of the muscle fibers themselves; this causes an increase in the number of fibers. Until its proven that hyperplasia actually plays a significant role in total human muscle growth, you might as well ignore it. We'll focus only on hypertrophy here.

Technically speaking there are two kinds of hypertrophy: sarcoplasmic and myofibrillar. Remember above, we divided muscle into two parts, the muscle fibers themselves and all the other stuff (water, glycogen, etc.)? This is where that division comes back into play.

Myofibrillar hypertrophy refers to an increase in the actual size/protein content of the muscle fibers, that is an increase in the protein content of the fibers themselves. In a sense, this is "real" muscle growth, because it represents an increase in the actual muscle fiber size itself. While myofibrillar hypertrophy is controlled by a complex array of factors (including the hormones I talked about a few chapters back), it also requires something else to get started: a high tension stimulus. That is, high tension in the muscle fibers themselves are the signal which stimulates the cell to increase muscular size (damage also plays a role). This is more or less the rationale behind the old weight training homily, "go heavy or go home". For now just think of this as tension training. We'll talk about that some more in the next chapter.

Sarcoplasmic hypertrophy refers to an increase in size and amounts of everything else in your muscles: glycogen, water, minerals, etc. You might think of this as pump growth. Some coaches also refer to this as energetic growth since it represents an increase in the energy content of the cell. Sarcoplasmic hypertrophy is also controlled by several factors (for example, testosterone increases glycogen storage which is why many steroid users report painful pumps when they train with high reps) but a primary stimulus is depletion of those energy stores (especially glycogen). This stimulates the cell to refill glycogen (and hence water, since every gram of glycogen stores 3-4 grams of water) in the muscle to higher levels than normal, which makes the muscle appear larger. Chronic high-rep training also increases capillary density, mitochondrial density and other non-contractile elements which contribute to increased visual size.

Now I should mention that the idea of myofibrillar and sarcoplasmic hypertrophy as two separate entities is not really supported by American research (or researchers for that matter). But most European texts do make the distinction. Beyond that, you can look at athletes who do different types of training and they simply look different. Yeah, I know, all of the mainstream folks reading this think I'm full of shit now but it's true. Guys who train with heavy intense loads (tension training) have muscles with a different visual quality than guys who only train with high reps and short rest intervals (pump training). That is, powerlifters who almost exclusively use tension training look different than the guys who use only pump training. Even bodybuilders like Dorian Yates, who train with heavy loads, just look denser than the guys who just pump the muscle endlessly. Of course, to maximize total size, you should use both but I'm getting ahead of myself here.

Before we continue, I want to expand on the steps involved in myofibrillar hypertrophy. The first step is the stimulus to grow new contractile tissue, which is a high tension load (along with damage). This activates certain genes in the muscle cell which tell the nucleus to produce messenger RNA (mRNA), which is simply a blueprint for proteins. mRNA comes out of the nucleus where it eventually runs into a cellular machine called a ribosome. With the mRNA as an instruction, the ribosome starts grabbing amino acids out of the intracellular amino acid pool and starts putting them together into new contractile proteins, which are then integrated into the existing fibers. Voilá, bigger muscle fibers. I should note that the processes involved are much more complicated than this but I don't want to get into all of the details in this book.

As I've mentioned before, this is an energy intensive process. Meaning that if cellular energy levels are low (because glycogen is depleted or creatine phosphate levels are low), protein synthesis won't occur very effectively. I should also mention that mRNA doesn't hang around forever, it starts to be degraded fairly quickly. In fact, recent studies show that the increased protein synthesis from a single bout of training is gone within 36 hours after that training bout. So maybe the old dictum of train a muscle every 48 hours wasn't so far out in left field.

It's currently thought that the speed at which ribosomes can synthesize proteins is the rate limiting step for protein synthesis. That is, the number and activity of your ribosomes is the bottleneck for how quickly you can grow. Few and/or slow acting ribosomes and you grow slowly; lots and/or fast acting ribosomes and you can grow more quickly. For the record, I should make mention that Duchaine and Zumpano were years ahead of the curve when they realized this back in 1982, in the original Ultimate Diet. Modern science is validating what they figured out over 20 years ago. I also want to mention that androgens increase ribosome activity which is probably another way they increase muscle mass above normal.

So, you ask, can we increase the number or activity of the ribosomes that are present in the muscle? Well, yes, but only temporarily. Like mRNA, the increased ribosome number is short-lived, on the order of a few days. And how do we do that? Well, in roughly the same way we stimulate growth in general: by applying an unfamiliar stress to the tissue. In response, the cell increases the activity (and maybe the number) of ribosomes. Faster protein synthesis can now occur.

I was originally hoping that my friend Bryan Haycock would have finished his book on Hypertrophy Specific Training, as that would explain all of the details that I skimmed over above. Apparently he hasn't so here's the brief overview. After looking at the research into growth extensively, Bryan concluded that training a muscle more frequently, with progressively increasing loads (weights increase at each workout for 2 week blocks) gives optimal growth. The above physiology gives some of the reason why. You can read more about Bryan's approach to training at http://www.hypertrophy-specific.com.

In any event, with frequent training and increasing tension stimulus, you can keep both ribosomes and mRNA levels elevated and grow more effectively. Training too infrequently doesn't optimize both ribosome and mRNA levels for maximal growth which may explain why many natural athletes don't do well with the typical "train each bodypart once per week" approach. Although we're not going to apply HST in its original form, we're going to use the physiology of muscle growth to our very large advantage. But before we get to that, I want to talk about actual training systems.

Chapter 8: Training systems:
How can they all be right?

Read through any muscle magazines and you'll see a seemingly infinite variety of training styles. Of course, when you look at them a little more generally, you find that they tend to be variations on a theme. To be (very) simplistic, I'm going to divide weight training into three primary disciplines: volume training, tension training and power training. Of course, advocates of each type will argue that their system is the only right way to train but this is obviously incorrect. If there was a single right way to train, then nobody who trained by any other method would be successful and that simply isn't the case.

So rather than looking at each as either right or wrong ways to train, I want to look at the pros, cons and potential application of each. It's not so much that each type of training is inherently right or wrong; rather, each can be "correct" or "incorrect" under a given set of conditions. Part of the goal of the UD2 is to use the proper training at the proper time to get the optimal overall response.

Volume Training

Pump training has been described variously as volume training, depletion training, or even sissy training (since the weights used are typically not as heavy). Poliquin's German Volume Training (10 sets of 10) as well as German Body Composition training also fits this description, as do many other systems. Let's just call it pump training.

Pump training tends to describe a majority of "traditional" bodybuilding training routines which is a high number of sets per bodypart (anywhere from 5 up to 20 or even more) with high reps (10-15 per set or even more) and short rest periods (30-60 seconds or thereabouts). It was definitely the most popular method of training back in the 80's. Modern pro bodybuilders probably tend more towards tension/intensity training (discussed next) although they revert to pump training for contest dieting.

As many will gleefully point out, most successful professional bodybuilders train this way (or at least they claim to, if you believe what the muscle comic books tell you). What is frequently ignored is that most successful professional bodybuilders are on a wide array of drugs that hasten recovery and help to make this type of training productive for them. Most natural bodybuilders, with the occasional exception, don't get much growth out of this type of training without the same drug support as the pros. But that's not to say that it can't be productive or useful under certain situations.

Pump training stresses the sarcoplasmic/energetic elements of the muscle more so than the contractile elements because of the lighter loads and shorter rest periods used. It not only depletes muscle glycogen significantly (due to the large number of sets, high reps, and short rest periods) but also stresses the creatine phosphate stores. It may even deplete intramuscular triglycerides. This results in supercompensation (storage above normal levels) when carbs, calories, creatine (and dietary fat) are made available again. By the end of a pump-training session, in addition to the marginal tension stimulus, there is a major depletion of muscle glycogen and other energy stores.

Unfortunately, as I mentioned in a previous chapter, most people's bodies aren't very good at doing two things at once. Refilling muscle glycogen and growing new muscle tissue counts as two things, and most people can't do both effectively. Since refilling energy stores takes priority (and protein synthesis is energetically costly), most people will refill muscle glycogen first, which may not leave time, energy or fuel for much muscle growth. The people who grow well with pump training are the folks who can do both efficiently; the folks who can't don't. Most folks can't. Trainers who have either naturally high testosterone levels (testosterone improves glycogen storage, yet another advantage of steroid use), high insulin sensitivity (meaning they better push nutrients into muscle cells) or use various drugs grow best on pump training. This doesn't describe your average trainee.

Despite its shortcomings for natural athletes, pump training has a role in this UD2. Although we're not using it primarily for growth, we are going to use it to achieve several specific goals. First and foremost is glycogen depletion which is the first step in setting up for glycogen supercompensation. This occurs for a number of reasons including an increase in insulin sensitivity, glucose uptake, and glycogen synthesis. Under those conditions, when carbs are made available, they are stored at a faster than normal rate. This allows us to overfill the muscles with glycogen.

Glycogen depletion also increases fat utilization by the muscle, which increases how well your body can use fat for fuel. This is important both from the standpoint of fat loss and protein sparing because, the better your body can use fat for fuel, the less it will need to break down protein for energy.

Second, pump training generates a lot of lactic acid (this is what makes your muscles burn but it does not cause soreness, despite what you have read) because of the anaerobic breakdown of glycogen. High levels of lactate are correlated with increase growth hormone (GH) secretion. Whether this GH release is really that relevant or important is debatable, but GH is involved in fat mobilization and raising GH certainly can't hurt.

Third, studies have shown that the hormonal response (mainly the catecholamines) to pump training stimulate fat mobilization. As discussed two chapters back, mobilizing fatty acids is the first step to oxidizing them and getting them the hell off of your gut (or ass).

Fourth, pump training burns quite a few calories, both during the workout and afterwards. Most of the calories burned after the workout come from fat oxidation, an additional benefit.

Finally, constant high-tension training can take its toll on joints and connective tissue. As big as he was, Dorian Yates was also one of the most injured bodybuilders out there; always trying to push heavier loads was probably a contributor. Pump training provides those tissues with a respite from heavy pounding and the higher reps, blood flow, and high lactic acid levels have nothing but a beneficial effect on joint health.

Although it might seem at first glance that pump training would go best with a high-carbohydrate intake, we're going to use pump training during the initial low-carb/low-calorie phase of the diet. This will deplete glycogen, set up for glycogen supercompensation, and enhance fat burning during the diet phase of the UD2. I'll tell you right now that doing pump training on low-carbs is one of the most miserable activities you will ever do.

Tension training

The next style of training I want to discuss is tension training. I should mention for the pedantically inclined that all types of training involve tension and using the terminology to delineate a specific type of training is a bit of a misuse of the word. Too bad, I couldn't think of anything clever to call it.

With pump/volume training, the focus is primarily on volume using a large number of sets and reps to stimulate the muscle. In tension training, heavier weights are used. While tension training can describe a vast number of training programs, it generally refers to any program where the focus is on using heavier weights and longer rest periods than in pump training. The goal, of course, is to stress the muscle with tension (rather than fatigue) in order to stimulate growth.

Although tension training could describe any number of systems, I'm simply going to define tension training as any type of training involving medium rep ranges (6-12 reps) and medium rest periods (1.5-2 minutes or so between sets) with the heaviest weights that can be used within those parameters. The number of sets done per bodypart can vary drastically with this type of training from the extreme low-end of 1 (a staunch HIT interpretation) to maybe 6-8 per bodypart or so.

Generally speaking, advocates of this style of training fixate on training to the point of concentric failure (where you can't lift the bar under you own power no matter how hard you try) on each set. Forced reps and/or negatives are sometimes implemented but this tends to burn out natural lifters very quickly. However, there is no research to substantiate that going to failure has any real benefit in this regards and most of the arguments for failure are logic based, having to do with ensuring you work as hard as possible. Regardless of interpretation, the goal of tension training is basically the same: use a heavy weight to failure (or at least near it) to generate an unusual stress to the muscle and stimulate growth.

Like other training systems, tension training can have drawbacks. This is especially true if failure is (over) emphasized. The first is the potential for injury. Form tends to slip as the lifter gets fatigued and that can cause problems. This also depends on exercise choice. I doubt any of you have ever squatted to failure (well, not deliberately) or seen anybody do it. The risk of a catastrophic injury is simply too high.   For this reason, a lot of intensity advocates use machines of some sort (Hammer machines are very popular and actually quite good).

Another problem with intensity training is that it tends to cause a lot of neural fatigue, which can lead to really extended rest intervals (7 days or more) while the lifter waits for strength to recover. The problem is that strength is a function of both muscle mass and nervous system activity. In the time that the lifter is waiting for the nervous system to recover, the muscle is going untrained. Recall from last chapter that ribosomes and mRNA don't hang around forever and a lot of staunch HIT'ers are reporting growing better by training a little less intensely but more frequently. Bryan Haycock's HST system is based on exactly that concept: maintain an optimal growth "milieu" in the muscle with more frequent training but with a lowered intensity level.

That said, tension training (we're actually going to stop a repetition short of failure to avoid excessive neural fatigue) has its place in this diet. It imposes an unusual stress on the muscle, which will upregulate ribosome activity in preparation for the real growth workout. It also depletes a good bit of muscle glycogen because of the highish reps and short rest periods. It also fulfills many lifter's psychological needs to go all out (or nearly so) effort wise.

Tension training will be used to start the carb-up towards all of the above purposes and one more. For optimal glycogen supercompensation, you not only need glycogen depletion, you also need training in general. As it turns out, you can only achieve glycogen supercompensation in the muscles trained. This means that, for optimal results, we need to train the entire body in a single workout. This is probably a little unusual for some readers, but tough. Since we only have 7 days to fit the cycle into in the first place, a full body workout is the only way to achieve what we want to achieve.

Power training

Power training (more accurately termed strength training) is for those lifters who love to impress their buddies with the amount of weights they can move. Trainees who don't like to lift heavy weights won't enjoy this type of workout very much. Too bad, but suffering builds character and the power training workout is a necessary evil.

Power training is typified by low reps (usually 3-5), multiple sets (varies from 3 to 10), and long rest periods (3-5 minutes) so that the heaviest weights possible can be used. Additionally, exercises are chosen that work the most muscles at a time, so that the heaviest weights can be used, placing the most tension possible on those muscles. Squats or deadlifts for the legs, bench, incline and shoulder press for pecs and delts, chins and bent over rows for back, close grip bench for triceps, barbell curl for biceps. Those are the exercises that folks typically use for power training. It's not that you can't use other exercises for power training but most isolation exercises are difficult or dangerous (especially to joints) to do with those kinds of low reps.

As stated above, power training puts a large tension load on muscle, as well as on joints and connective tissue, but it doesn't really stress energy stores that much, because the sets are short and the rest periods are long. You use mainly ATP and creatine phosphate stored in the muscle; very little glycogen is used.

The near maximum tension stimulus from power training sends a very definite myofibrillar growth signal to the cell nucleus, producing the mRNA to synthesize new tissue from the increased ribosomes generated from the HIT workout. And since energy stores aren't depleted much, if at all, synthesis of new contractile proteins can be the main priority of the cell (rather than energy repletion).

Tangentially, I suspect this is why lower volume (and/or lower repetition) training tends to be more effective for genetically average trainees. Without the capacity to replenish energy stores readily, your genetically average trainee simply can't get much growth out of higher volume and/or higher repetition types of training. By limiting the amount of work done, and the amount of energy depletion, natural trainees can actually get their bodies to apply energy to growth.

In order to make power training most productive, we want to be both glycogen and creatine loaded. Glycogen supercompensation, along with the water it brings into the muscle, increases strength through purely mechanical means. The physical stretching of the cell appears to act as an anabolic signal itself. Creatine phosphate provides the energy for high tension lifting. Ideally, you want ribosome activity upregulated so that the tension stimulus from the power training will generate real muscle growth.

So the power training workout is most effectively placed after carb and creatine loading and after the tension workout. This ensures that the cell's energy level, along with ribosome number and activity, is high so that protein synthesis can proceed as quickly as possible. Maybe you're starting to see how this system fits together now. If not, don't worry, all will be explained in the next few chapters.

Ch 9: Ultimate Diet overview

So now you have a lot of information floating around, details of partitioning, fat metabolism, and training systems. In this chapter, I want to look generally at how it all starts to fit together. In the next three chapters, I'll look at the day to day details of the cycle.

The UD2, fundamentally, has two parts: a catabolic/low-carbohydrate phase to maximize fat loss and an anabolic/high-carbohydrate phase to rebuild and gain muscle. Although this is a lot to go through, it aims to eke the best performance out of a sub-optimal metabolism. As I mentioned earlier, this is nothing new and the UD2 stands on all of the diets before. The UD2 is simply more up to date and more refined, taking advantage of the most current research. Before getting into the nitty gritty of the cycle, let's look at each phase individually.

Fat loss

First let's sum up what we want to do in terms of maximizing bodyfat loss during a diet. In the most general sense, we want to accelerate the rate of fat utilization by the body. Not only does this increase fat loss, it will exert a protein sparing effect. Let's look at how we might accomplish this dietarily.

By using MCT or DAG, we can provide dietary fatty acids to tissues like the liver and muscle at a more rapid rate. But that's only part of the picture and the low-carbohydrate phase is not very high in fat as you'll soon see. Quite in fact, you'll only be allowed a relatively small amount of fat during the diet phase but, if possible, that fat should come from either MCTs or DAGs. Coconut oil, which is half MCT can also be used.

We also want to accelerate the processes of mobilization from the fat cell, transport (via increased blood flow), and utilization in the muscle. Let's look at each.

First we want to lower insulin levels, which is easily accomplished by reducing dietary carbohydrate intake. This will have a natural effect of increasing catecholamine levels (especially in the first few days of carb restriction), but we can do more. Exercise is a natural choice, of course, and both weight training and aerobics (or interval training) will enhance catecholamine output (this effect is further enhanced on low-carbohydrates). Compounds such as ephedrine/caffeine or clenbuterol also enhance nervous system output, increasing fat mobilization (and possibly utilization in skeletal muscle). Both have anti-catabolic (protein sparing) effects.

So now we have enhanced nervous system output, both adrenaline and noradrenaline. Depending on the ratio of beta-1,2 to alpha-2 receptors in the fat cell, we might get a potent fat mobilization signal (high beta-1,2/low alpha-2) or a poor fat mobilization signal (if there are a lot of alpha-2 receptors and not very many beta-1,2 receptors). As mentioned, stubborn fat is known to have more alpha-2 receptors; this is especially true for women's hip and thigh fat and somewhat true for men's abdominal and low-back fat. As it turns out, beta- and alpha- receptors also affect blood flow to the fat cell; beta-receptors increase blood flow and alpha-receptors inhibit it. Though I'm not aware of any direct data, I suspect that the circulation to women's hips and thighs (and perhaps men's ab fat) has a preponderance of alpha-2 receptors which decrease blood flow.

As mentioned before, thyroid has a profound effect on adipose tissue blood flow but, short of using thyroid medication, there's not much we can do here. Fasting increases blood flow to fat cells and low-carbohydrate/ketogenic diets effectively mimic fasting. Aerobic exercise also increases blood flow to adipose tissue in addition to its other effects on calorie burning.

So, you ask, is there any way to inhibit alpha-2 receptors? Several years ago, Dan Duchaine introduced the world to yohimbe, a natural compound that inhibits alpha-2 receptors. Although not perfect orally, regular use (0.2 mg/kg of bodyweight) seems to help. Unfortunately, yohimbe (especially the bark) tends to cause side effects: sweating, a racing heart, and other problems. Yohimbine HCL (the drug form) eliminates many of these problems but poor blood flow to fat cells still limits its usefulness.

Given enough time, yohimbe will build up in the tissues and exert a greater effect (it's also likely that yohimbe improves blood flow to the fat cell). Recently, there have been a slew of topical yohimbe creams that purport to isolate the alpha-2 inhibition to the fat cell; you rub them on your stubborn fat areas and then go do cardio.

As it turns out, there is a dietary way to inhibit alpha-2 fat cells: raising blood fatty acids. When you lower carbohydrates to 20% of total calories or below, blood fatty acids increase (as a side-benefit, the catecholamine response to exercise is also increased when carbs are lowered this much).

After 3-4 days of exposure to fatty acids, alpha-2 receptors become inhibited naturally (without the side effects of oral yohimbe). Combined with aerobics, this is a potent way to get rid of stubborn fat. It also explains the previous observation that low-carbohydrate diets make mobilization/loss of women's stubborn fat easier: between the lowering of insulin and the fatty acid mediated inhibition of alpha-2 receptors, those fat areas were easier to get rid of. Of course, decreasing carbohydrates fits in nicely with our goal of lowering insulin in the first place.

So now we have large amounts of fatty acids floating around in the bloodstream. While this will, in general, increase fatty acid utilization by the body, we can do more. By depleting muscle glycogen with weight training (or even intensive endurance or interval training), we can upregulate CPT activity and enhance fat oxidation in the muscle. Depletion of liver glycogen (via carbohydrate restriction and exercise) will do this in the liver as well. As I mentioned previously, glycogen depletion also sets us up for compensation (and an anabolic rebound) later in the cycle.

Fundamentally, all I have described is a low-carbohydrate/ketogenic diet coupled with training which happens to address most, if not all, of the processes we are trying to optimize for fat loss. Which makes this as good a place as any to discuss ketosis.

What about ketosis?

If you're familiar with Bodyopus or any of the other cyclical diets of this sort, you're probably wondering about the importance (or not) of ketosis, which I haven't mentioned at all. Ketosis is a metabolic state that occurs when fatty acid oxidation is ramped up to a very high degree, such that the liver is unable to use them all for energy. This occurs under conditions of total starvation, low-carbohydrate diets, and even extensive endurance exercise. Under those circumstances, fatty acids are only partially oxidized. This results in excess acetyl-CoA which is then converted to ketones which are released into the bloodstream. When ketones accumulate beyond a certain concentration, the body is said to be in ketosis. When ketosis occurs and blood glucose is low (as in the case of a low-carbohydrate/ketogenic diet), ketones become the preferred fuel in many tissues.

Now, ketones have a very important role in human physiology: to provide the brain with fuel when glucose is available in only low amounts. Recall from previous chapters that the brain can't use fatty acids for fuel; its primary fuel is glucose. When glucose becomes unavailable, the body needs an alternate energy source; that source is ketones. In fact, after 3 weeks of ketosis, the brain will derive nearly 75% of its total energy requirements from ketones; the remaining comes from glucose which is made in the body from amino acids, pyruvate, lactate and glycerol (from fat metabolism).

Depending on who you talk to, ketosis is either thought to be wonderfully beneficial or deadly. As with most extremist stances, the truth is somewhere in the middle. It's true that diabetic ketoacidosis, which only occurs in Type I (insulin dependent diabetics) can be lethal but this level of ketosis will never develop in non-diabetics. There are various feedback loops that prevent it. And, under certain conditions, ketosis has potential benefits.

One is protein sparing. Arguably the main reason the brain shifts to using ketones during starvation is to reduce its reliance on glucose; this means less body protein needs to be broken down to make glucose. In studies where protein intake is too low, ketosis may also be protein sparing. For the most part, if protein intake is adequate to begin with, I haven't seen any convincing data that ketosis has much of an additional benefit.

One problem may be that lean individuals can't make enough ketones to exert a protein sparing effect; this is a consequence of the difficulties in mobilizing fatty acids in the first place. Even during total starvation, when you'd expect ketosis to have the greatest impact, ketones aren't protein sparing in lean individuals (<15% bodyfat or so). Perhaps this is the shining moment for MCTs, by producing ketones in larger amounts, we can exert a protein sparing effect beyond simply providing quick fat energy. Assuming protein intake is sufficient in the first place, I still tend to doubt ketosis has any huge advantages in this regards. If it does, it simply hasn't shown up in real world experience. Bodyopus did generate better fat loss and less muscle loss for some people, but I suspect this had more to do with the carb-load.

A second argued advantage is that ketones are inefficient, that you'll lose more fat for a given calorie deficit in ketosis than without. The mechanism given is that one pound of fat converted to ketones doesn't provide the same energy as one pound of fat burned directly. This may be somewhat true but the difference is minor, amounting to a few percentage points at most. As well, except in very obese individuals, most tissue of the body aren't using ketones past the first couple of weeks; they are using fatty acids. There is also a small loss of ketones in the urine but this also tends to amount to very little. As with the protein sparing effect, increased fat loss from being in ketosis just hasn't shown up in the real world with lean athletes and bodybuilders.

For the most part, I simply see ketosis as a "side-effect" of fat loss (burning to be more accurate), more than something to be explicitly sought out. That is, when you accelerate fat oxidation with the methods above, you tend to enter ketosis. Ketosis in and of itself isn't any big deal. For that reason, I won't talk about monitoring ketone levels with Ketostix or anything like that. Frankly, using a low-carbohydrate/ketogenic diet for the fat loss phase of the UD2 has more to do with lowering insulin, raising catecholamines, and ramping up fat oxidation; ketosis is simply a tangential effect. A low-carbohydrate diet is also the only way to reduce calories as low as I'm going to suggest; there simply isn't caloric room for many carbohydrates in the fat loss phase of the diet.

Transition

Now, the fact is that no matter what we do, dieting is going to be inherently catabolic, for the reasons I expounded upon in the chapter on partitioning. Even if we train, eat lots of protein, provide fat energy as rapidly as possible, dieting still causes muscle loss. Duchaine said it best in Bodyopus when he said "You will always lose muscle on a diet." It's depressing but it's true.

That ignores the myriad adjustments that occur to metabolic rate in response to dieting. In some studies, metabolic rate can start to drop by the fourth day of dieting. While Dan pinned the drop on thyroid, hopefully you understand from previous chapters that it's more complicated than this: it's an integrated response to decreased thyroid, insulin, nervous system output, leptin and everything else.

So although I've tried to optimize all of the processes involved in fat loss, mobilization, transport, and burning, we still have a problem: muscle loss. Generally, at best you can slow muscle catabolism (breakdown) on a diet; generating an anabolic response is almost (if not completely impossible). An ideal fat loss diet **must** rebuild lost muscle.

I suppose I should mention psychological issues, too. Dieting is no fun, long stretches of caloric or food deprivation are hard to stick to. Yes, bodybuilders and athletes will generally suck it up and stick it out but this is usually neither necessary nor productive. Allowing forbidden foods (even junk food) and relaxed eating has psychological benefits above and beyond the physiological benefits I've discussed already.

The anabolic rebound

In Bodyopus, Dan used simple glycogen supercompensation as an anabolic driver. In short, by driving glucose and water into the muscle cell at an accelerated rate (by preceding a carb-load period with a full-body glycogen depletion workout), he attempted to generate an anabolic response. As I've mentioned before, I don't think this was ideal. Glycogen compensation probably does work as an anabolic driver for muscle: heavy tension workouts and muscle damage works better. In my book on ketogenic diets, I suggested doing a heavy tension workout prior to the carb-load; in the UD2 I'm going to expand on that.

We're going to take the approach of the original UD, using both a tension workout and a power workout, except that we're going to do in 7 days what it took the original UD 10 days to accomplish. I should probably mention one of the primary reasons I moved to a 7 day cycle: convenience. A 10 day cycle is, frankly, a pain in the ass. Most people work on a 7 day work week, and training and diet programs that don't take that into account tend to have poor adherence. Admittedly, it's complicated to cram all of what we need to do in a 7 day cycle, I spent a great deal of thought and time doing it. But the UD2 will allow you to eat relatively normally on Friday and Saturday, which is when most people want to go out with friends, eat out at restaurants and generally cavort and carry on.

Anyhow, by depleting muscle glycogen during the low-carbohydrate phase of the diet with training and carbohydrate restriction, we set ourselves up for glycogen supercompensation which is an anabolic driver in its own right. But while the UD did it in 3 days and Bodyopus did it in 2, we're going to get it done in just over 24 hours.

We're going to couple that with a fairly standard full-body tension workout to upregulate ribosome activity and number. While this is anabolic in its own right, its main purpose is to set us up for the second part of the one-two growth punch. With the advantage of increased energy stores, increased mechanical leverage from carbohydrate loading, and the increased ribosome number and activity from the tension workout, the power workout presents a powerful growth stimulus without depleting muscular energy stores. This is followed by a day or two of relatively "normal" eating to allow growth and recovery.

In addition to the muscle growth effect we're going to achieve, the 2-3 days of high(er) calorie overfeeding will help to reverse many (if not all) of the metabolic adaptations which took place during the low calorie phase. I'll talk about calorie levels in the next several chapters but you get to eat relatively freely on weekends which, as I mentioned above, has profound psychological benefits in addition to all of the physiological benefits.

We won't get fat again because we will be controlling where those calories are going (some caloric restriction on Sat or Sun may be required). By transiently increasing insulin sensitivity (with glycogen depletion and training), we can direct incoming calories towards muscle, instead of fat cells. By making specific macronutrient choices (emphasizing carbohydrates over dietary fat), we can further enhance this. The growth stimulus from the tension and power workouts further enhances the shuttling of calories into muscle mass (meaning there aren't as many to go to fat cells). We also get to take advantage of a neat metabolic trick which I'll describe later.

And then the cycle repeats. Coming out of the 3 days of overfeeding, metabolism will be somewhat recovered, making fat loss more efficient during the next 4 day dieting cycle. This will all make more sense in the next chapters.

Four last things before starting

Before you jump into the UD2, I need to make four final comments. If you've been dieting up until this point, I strongly suggest that you take 7-14 days off your diet and eat at maintenance. This will allow metabolic rate to recover so that you can start fresh on the UD2. Because of the 3 days of overfeeding in each UD2 cycle, metabolic slowdown is mostly attenuated, but you don't want to go into the diet with an impaired metabolism to begin with.

Second, you're going to need to know your current mixed diet maintenance calorie level since you'll be using that to set your calories during most phases of the diet. If you don't know what that value is, shame on you. But you can use a rough estimate. The average person will have a maintenance caloric requirement somewhere between 14 and 16 calories per pound of current bodyweight or so. If you feel that you have a slow metabolism, pick the lower value. If you feel that you have a high metabolism, pick the higher value. If you think you're in the middle, use the middle value. Women are typically at the lower end of the range and you'll have to play with the calorie levels a little bit anyhow.

This, and perhaps most importantly, if you have been using a traditional high-carbohydrate/low-fat diet, jumping into the UD2 is a recipe for disaster. You'll feel like shit and probably give up after two days of carbohydrate restriction. In this case, I **strongly** suggest you move to a moderate carb/moderate fat diet containing something like 30% protein, 40% carbs and 30% fat and stay there at least two weeks (if you've been dieting, you can use this as your 2 weeks off diet to save time). Then you can move into the UD2.

Finally, like any diet the UD2 should be used in phases. Most people diet for too long at a stretch and this causes more problems than it solves. From a starting point of 12-15% bodyfat, most readers shouldn't need more than 6-8 weekly cycles to reach their goals. If you need longer, I strongly suggest you take a 2 week break every 6-8 cycles of the diet, eat normally and at maintenance. Then you can return to dieting. Otherwise, you'll probably end up overtraining (this is true of any diet). The same goes for athletes using the UD2 for mass gains, after 6-8 weeks of constant use, take 2 weeks, eat and train normally before returning.

Chapter 10: The low-carbohydrate phase

Over the next three chapters, I'm going to walk you through a week's cycle of the UD2. For each day of the cycle, I'll look at training, diet, and any supplement or drug options you may wish to consider. I'll also talk about setting calories for the different goals I discussed way back in the earlier chapters. I'll also give you an idea of what to expect on each day. I'll address some other issues, such as UD2 variations for different goals in Chapter 12. Don't get worried if I don't address all of your concerns here.

As with the original Ultimate Diet or even Bodyopus, don't expect to nail the UD2 perfectly the first time through. You should figure on at least two cycles before you get everything dialed in just right. The biggest issues will revolve around calorie levels on Saturday and Sunday since there seems to be the most variability there in how people respond.

I'm going to assume you're starting the cycle on Monday, since that makes your workouts Monday, Tuesday, Thursday and Saturday, leaving Friday night off if you want to go out. But there's no law that says you have to do it that way and there's actually one reason you may want to shift things forward a day.

Day 1/2: Monday/Tuesday

Day 1 and 2 are both low-carbohydrate days, coupled with high rep, short rest period workouts. I should mention right now that high reps and short rest periods on low-carbs is a miserable, miserable experience. Nausea, lightheadedness and a general desire to track me down and kick my ass are common reactions. All I can say is to suck it up; if you want superior results, you're going to have to work for them.

Where you set calories depends on your goals. If your goal is maximal fat loss, these are the days to really cut calories. As I mentioned last chapter, you should know your normal mixed diet maintenance calorie level. Cutting this value by 50% is the goal (so if your maintenance is normally 3000 calories/day, you'd cut that to 1500 cal/day). Normally I wouldn't use such a severe deficit but since you're only dieting for 4 days (more or less), this is necessary.

In general, nobody should go below 1200 calories/day as it becomes simply impossible to get enough protein or micronutrients and stay full. If you have a low maintenance calorie level (i.e. the average woman may have a maintenance level of about 1700 calories/day) and want to create a larger deficit, you'll have to add some aerobics. Men with low activity levels or slower metabolisms may need to do aerobics as well.

If your goal is more attuned towards muscle gain, don't cut calories as hard on the low-carb days. A 10-25% reduction is sufficient. You might even try eating at maintenance or higher if you want to make the entire cycle as anabolic as possible. Just realize that you probably won't lose much fat if you lose any at all.

The key to these days is also reducing carbohydrates. As a general rule, you should eat no more than 20% of your total caloric intake as carbohydrates. This will put most people in the 65-70 gram range or so. You can reduce this further to 50 grams if you wish.

Why not zero as in Bodyopus? Because, outside of letting us cut calories further, there doesn't seem to be any major benefit to going lower. Even a minimal 50 grams of carbs per day will help to limit the need to produce glucose from muscle protein. Remember, Bodyopus was concerned with rapidly establishing ketosis which requires as few carbs as possible to get it done quickly. Since I don't care about ketosis, I don't see any real need to reduce carbs below 50 grams per day. If you simply want to reduce carbs further (to cut calories), you will want to increase protein slightly (see below).

Be forewarned, if you've never done a very low-carbohydrate diet, you're in for a shock. Don't be surprised if you feel mentally fuzzy or a little bit brain dead (this is also why I suggest at least 2 weeks on a moderate carb/moderate fat diet before you jump into the UD2). If this is the case, you may need to increase carbohydrates to 100 grams/day. This usually goes away after a few cycles.

With a carbohydrate intake this low, the source won't matter hugely. The reduction in carbs alone will be sufficient to cause all of the effects I described in previous chapters: lowered insulin, increased catecholamines, increased blood fatty acid levels, etc. However, choosing lower glycemic index, high-fiber carbs will keep you fuller because they take longer to digest and sit in your stomach longer. Veggies in your morning omelette or a salad at lunch and dinner add considerable bulk to the diet without adding large amounts of digestible carbohydrate calories. The fiber will help you poop as well. Small amounts of starches can be added up to your daily limit and you could even have one of the low GI fruits if you really wished. Just keep track of the total amounts and keep the value to 20% or less of your total calories.

Protein should bet set at 1-1.5 gram per pound of lean body mass depending on how many calories you have available to eat each day. If you decide to cut carbs to less than 50 grams/day, I'd suggest using 1.5 g/lb of protein. More than this is neither necessary nor beneficial. Pick from varied (food) protein sources including egg, fish, chicken and lean red meat. Many dairy sources are relatively carb-free as well and recent research has implicated dairy calcium as being beneficial for fat loss.

Red meat tends to keep people fuller for a variety of reasons but you do have to watch the fat content. If you must use a protein powder, stick with a casein or casein/whey blend. Casein has recently been shown to be anti-catabolic compared to whey protein, which means it will be more useful in helping to prevent any muscle loss during the diet days. I think using a low-fat cheese on top of your other proteins is an excellent way to get casein protein without resorting to liquids. It tastes better too. Liquid proteins don't tend to control hunger very well which is why I don't recommend them on the diet days.

So now you've set your calorie, carbohydrate and protein levels. The rest of your caloric allotment, whatever it is, should come from dietary fat. How much fat will depend entirely on where you set calories. Between protein and carbohydrates, you should be consuming in the 800-1000 calorie/day range (I'm figuring 150-200 grams of protein and 50-75 grams of carbohydrate per day).

If you were only eating 1200 calories/day, that'd leave between 200 and 400 calories from fat. At 9 calories/gram, that's just over 20-40 grams of fat. This isn't much, but should be enough to keep you full and happy. Don't forget that most protein sources have a small amount of tag-along fats so 20-40 grams leaves you with about 2-3 additional tablespoons of fat at most. Obviously, if calories are higher, fat intake will be higher as well.

As far as fat source, the only real requirement is that you get 6X1 gram capsules of fish oil per day. If you absolute can't stand them (some people get nasty fish burps), flax oil is an alternative source. As I mentioned previously, it would probably be ideal to get the rest of your fats from MCTs or the new DAG oil. Even if they aren't protein sparing, they will tend to make you feel more energetic. You can also use coconut oil which contains 50% or more MCTs. Failing that, you can use something fairly neutral such as monounsaturated oil (olive oil or one of the high oleic safflower oils; both go well on salad).

Oh yeah, one more thing. Because of the low calories, the typical 6 meals per day approach isn't really feasible (nor necessary). Well, not unless you find a meal of 200 calories to be particularly satisfying, and I doubt many of you do. Three to four meals tends to work best, spread throughout the day. That might mean breakfast at 8am, lunch around 12:30, supper at 5pm and dinner at 9 or 9:30. Various non-caloric snacks can help you get through the day if hunger is a real problem. If your sweet tooth just won't give up, sugar free jello or something similar can be helpful.

I hate meal plans but here's a sample daily menu at 1200 calories. Breakfast would be scrambled eggs (1 whole egg, 5 egg whites) with a glass of milk or piece of low-calorie, high-fiber bread as toast (this provides only 12 grams of digestible carbohydrate). Lunch would be a can of tuna fish with 2 tablespoons of fat free ranch dressing, a teaspoon of olive oil, and two slices of low-calorie/low-carbohydrate bread (about 25 grams of digestible carbohydrate). Or replace the bread with a small sweet potato. Or replace the tuna with turkey or chicken breast for a sandwich. You could repeat this meal later in the day or have a couple of fast food hamburger patties without the bun with a side-salad. Dinner might be a chicken Ceaser salad or something similar; basically lean meats with vegetables and fairly small amounts of dietary fat. It's not a terribly exciting or varied diet, but you only have to follow it for four days out of seven so suck it up. Most dieters tend to rely on the same foods day-in and day-out so I don't think this will be a huge problem anyhow.

Other than fish oils and basic vitamin and mineral supplements, nothing is truly required on these days. The ephedrine/caffeine stack, if it's still available by the time this book sees print, is an excellent adjunct to any diet. It increases fat mobilization and burning, blunts appetite, and helps to limit part of the drop in metabolic rate. It may be one of the most useful OTC compounds out there, although it's certainly not required. The combination of 20 mg ephedrine and 200 mg of caffeine taken at least three times during the day (but no later than 4 hours before bedtime if you like to sleep) has demonstrable results. Synephrine, an alpha-1 agonist, may have benefits as well. Various and sundry appetite suppressants can be useful to help blunt nighttime hunger if it's a problem.

A good multi-vitamin along with some Vitamin E (800 IU of d-tocopherol) and zinc (50 mg zinc orotate) are another good addition. Additional calcium (600 mg in the morning and 1200 mg at bedtime) has been shown to modulate fat cell metabolism and increase fat loss on a diet. Some, but not all, studies have shown dairy calcium to be superior but pills are an easy way to bump up calcium intake without adding calories. At bedtime, two grams of the amino acid glutamine will increase GH output, which may help with fat mobilization. Larger amounts of glutamine (5-10 grams) appears to improve sleep quality so this is a consideration if you have trouble sleeping on low-carbohydrates.

If you prefer stronger compounds, clenbuterol (60-160 mcg/day) or thyroid (T3, 50-75 mcg/day) medications would also be a possibility here. Injectable growth hormone is ungodly expensive but profoundly fat mobilizing. Although anabolics don't work terribly well on lowered calories, they probably have muscle sparing effects that would be useful on a low-calorie diet.

Although training in the morning is probably ideal because you'll have more energy then, most people's work schedules may prevent it. I'll assume an evening workout session. In the big scheme of things, it won't matter that much. The goal over these two days is to deplete muscle glycogen in all of your major muscle groups, for all the reasons I've discussed in earlier chapters. At the same time, you don't want to deplete glycogen so far that you increase muscle loss. It's a fine line to walk. The original Ultimate Diet gave a recommendation of 20 sets per bodypart, 15-20 reps per set which was the standard depletion advice. Bodyopus recommended 2 to 20 sets which wasn't terribly helpful.

Recent research into glycogen depletion rates during weight training can let us be a bit more specific in determining how much work to do. Assuming each of your sets lasts about 45-60 seconds (which is 15-20 reps at a fairly quick tempo or 10-12 reps at a slower tempo), you'll need about 10-12 sets per bodypart to deplete glycogen to about the right level.

You should be using a weight that is roughly 60% of 1 rep max, tough to complete the set but without causing failure. That will generally allow about 15-20 reps or so. Take about a minute or a minute and a half between sets. This gives you enough rest to avoid puking, but still lets you elevate lactate/growth hormone levels. Be warned, if you're not used to this type of training, be prepared for some serious discomfort. Nausea, intense muscular burning and fatigue are going to be your new friends on these workouts.

In fact, if you don't feel all those sensations and more, you're not working hard enough. More concretely, if you're not feeling a burning sensation in the muscle you're trying to train, you're not depleting muscle glycogen. Either your form sucks and you're not using the muscle you think you're using or you're going too fast during your sets.

A pre-workout stimulant such as caffeine, or ephedrine/caffeine (if you're not training too late at night), or the amino acid l-tyrosine (1-3 grams) with caffeine (200 mg) will help you get through the workout. Two grams of the amino acid glutamine prior to this workout will enhance the GH output if you're convinced that it matters. It won't hurt but I'm not convinced it will help either.

You've got a few options as far as training. The most miserable way to do it would be to do a full 2 hour depletion workout on Monday but this is workable if you've got a high pain tolerance. Personally, I get bored after about an hour so I prefer to do the carb depletion across two days.

Alternately, you can split the depletion across two days. There are several possible splits to use. Chest/back/shoulders on Day 1 and legs/arms/abs on day 2 is one good option. This puts all the work for the shoulder girdle on one day, and pairs the exhausting leg workout (high rep/short rest legs is just brutal) with the less energy intensive arms on day 2. The leg workout also comes before the day of rest. You can also alternate chest and back exercises and biceps and triceps exercises to get out of the gym faster. Also, since arms get worked indirectly from the other exercises, you can cut the number of sets down.

Another option would be a more traditional chest/shoulders/triceps on Day 1 and legs/back/biceps on day 2. I'll warn you that legs, back and biceps is a brutal combination and I personally prefer/recommend the first option over this one.

Exercise selection for these workouts isn't really that critical. Make sure for large bodyparts like chest and back that you select exercises which hit different angles. So flat and incline bench for chest; rows and pulldowns for back, that sort of thing. This is to ensure glycogen depletion of as many muscle fibers as possible. Generally I think that machines are a better choice since weight changes are faster and you don't have to hassle with loading and unloading plates. This is especially true if you do the workouts circuit fashion jumping from exercise to exercise.

My personal preference is actually to do two full body workouts on both days with 5-6 sets per bodypart done at each workout. I'll only do 2-3 sets for shoulders and arms. I'll pick one exercise per bodypart and do 3 sets of 12-15 with a 1 minute rest, before moving to the next bodypart. After working through the entire body, I'll rest a few minutes and then do it all over again. I do this workout on both Monday and Tuesday. The workout appears on the next page.

After each workout, moderate amounts of cardio (30-60 minutes) is permissible but don't be surprised if you're not terribly motivated and/or don't have the energy for it. Smaller individuals (and most women) with a low metabolic rate who need to increase their caloric deficit will need to do cardio on these days. Alternately, you could do cardio first thing in the morning and lift in the evening or vice versa. Just realize that the cardio will tend to make you even weaker than you'd already be when you lift. Two more grams of glutamine at bedtime can help with GH release.

Sample Monday Workout

Monday	Tuesday
Leg press: 3X15	Leg press: 3X15
Leg curl: 3X15	Leg curl: 3X15
Chest press: 3X15	Incline bench: 3X15
Row: 3X15	Pulldown: 3X15
Lateral raise: 2-3X15	Lateral raise: 2-3X15
Calf raise: 3X15	Calf raise: 3X15
Biceps curl: 2X15	Biceps curl: 2X15
Triceps pushdown: 2X15	Triceps Pushdown: 2X15
Repeat twice	Repeat twice

Day 3: Wednesday

Day 3 is nutritionally a repeat of Days 1 and 2; low-carbohydrate with the same foods as on day 1 and calories set depending on goals. Its also a day off from weight training which you'll probably want by this point. If you're not used to the type of training you did on day 1 and 2, don't be surprised if you're more sore than you expect to be. It's a consequence of the unusual stress of high reps/short rest periods and the lack of carbs to help with recovery.

If you want to do cardio today, that's fine but don't be surprised if your legs are very tired from the day before. While I'm not convinced that doing it first thing in the morning will have any real benefit, especially on lowered carbohydrates (where you've got lots of fatty acids floating around all day long), you can do it this way if you so desire. Forty-five to 60 minutes of moderate intensity cardio is probably about right for most people. Women especially should include the cardio to improve blood flow to stubborn fat areas. Oral (0.2 mg/kg with caffeine but no food) or a topical yohimbe cream could be used before cardio to help with stubborn fat mobilization.

Ephedrine and caffeine during waking hours will keep your metabolism humming and help keep appetite at bay. If nighttime hunger is a problem, try eating more veggies, or fattier cuts of meat so that the food stays in your stomach longer. You could also move cardio to the evening, as exercise tends to blunt hunger a little bit. If you can find it, norephedrine (aka Dexatrim) is an excellent non-stimulant appetite suppressant. Once again, if you want to boost GH at bedtime, 2 grams of glutamine works fine.

I should mention that mentally this is probably the hardest day for most people. You don't get to train but you do get to be hungry all day. Even though the high rep workouts suck, at least the training keeps your mind off of the hunger for a little while. Don't worry, you're almost to the fun part of the diet.

Day 4: Thursday AM

If Wednesday is the toughest day physically, Thursday is the toughest day psychologically. Between glycogen depletion and dehydration, most lifters feel flat, stringy and weak. They look like shit in the mirror and can't get or hold a pump. Considering the psychological issues present in these folks, this does not a happy athlete make. Well, live with it, you're almost there. Thursday is also difficult psychologically because the carb-load is so close to starting, but you still have to suffer through another day of low-carbs.

Thursday is also the oddest of all of the days of the cycle. We're going to wind down the caloric restriction/carbohydrate depletion/fat loss phase of the diet, and get ready to move into the recarb/growth phase. Meaning that you have both low and high-carb meals today. This causes some confusion when it comes to caloric intake and meal planning. I'll offer an alternate option for people who really dislike the combinatory nature of this day in a later chapter.

Another moderate cardio session in the morning (or lunchtime at the very latest) is an excellent idea here. As I mentioned in a previous chapter, something nifty happens when you've been on low-carbohydrates for 3-4 days, the high free fatty acid concentrations inhibit those pesky alpha-2 receptors, making stubborn fat mobilization that much easier. For maximum fat loss (especially stubborn bodyfat), you should take advantage of it. Just don't tire yourself out too much before tonight's workout; try to get at least 4 hours between cardio and your lifting session.

During the day, you're going to stay with your normal low-carb/low-calorie fare but you only get to consume 75% of the total calories that you were eating on days 1-3. Basically, you only get to eat your first 3 meals: breakfast, early lunch, late lunch. So if you were on 1200 calories/day during day 1-3, you would eat 900 calories during the day. Your last meal should be about 3-4 pm, assuming a 7pm workout.

Chapter 11: The carb-load

Now it's early Thursday evening and you've finished the final low-carbohydrate/low-calorie day of the cycle. Now it's time to turn things around and rebound into an anabolic/growth phase. We pick things up on Thursday evening.

Day 4: Thursday PM: The workout

To prepare for today's weight workout, you want to consume about 25-30 grams of carbs, with about 15 grams of whey protein 30-60 minutes before the workout. This accomplishes a few things. First, it starts to shift you out of a ketogenic state, so you can resume anabolism and start the carb-load. The increase in insulin will also lower free fatty acids, helping to increase insulin sensitivity. Second, it will make you much stronger in the gym by raising blood glucose from low-normal levels. Third, it will provide amino acids for growth. If you decide to creatine load over this and the next day, I'd also advise adding 5 grams of creatine to this "meal".

As far as food choices, sucrose or fructose are both okay here since you want to start refilling liver glycogen to shift your body back to an anabolic state. A protein drink and one or two pieces of fruit are a possibility, I frequently just use a protein bar that contains roughly the amounts of nutrients I want. Some caffeine and 1-3 grams of the amino acid l-tyrosine will give you a nice pre-workout stimulant effect (to make you strong in the gym) without keeping you awake like ephedrine would.

Thursday's workout is the heavy duty/high intensity workout. As I talked about previously, this workout has several important goals, so let's recap them here. The first goal of the workout is to deplete the last little bit of glycogen to prepare you for the carb-load. Because you're already mostly glycogen depleted, you only need 2-4 sets per bodypart or so to prepare your body for carb-loading. Training the full body also upregulates enzymes of glycogen storage and synthesis and increases insulin sensitivity. The second goal is to start setting your muscles up for growth over the next few days. The stress of this workout will upregulate mRNA levels and ribosome activity so that you'll get maximal growth from the upcoming power workout.

First, let me mention that you'll be training your entire body today. I know full body workouts are out of vogue in bodybuilding these days, but that's what you're going to do. Hopefully, by this point, Bryan Haycock's HST book is out, where he gives some good reasons for full body workouts. If not, I'll outline a few of those reasons, especially as they pertain to this diet.

One of the main reasons is that training upregulates the enzymes responsible for glycogen synthesis and storage to higher levels than simple carbohydrate depletion can accomplish. Training also improves insulin sensitivity and glucose transport but, as with enzyme activity, this only occurs in the muscles that get trained. Training the entire body ensures that the incoming carbs will be stored at a maximal rate in all muscle groups. A split routine would not accomplish this.

This was the logic behind the full-body depletion workout in the original Bodyopus diet but we're using a tension workout instead of a high-rep workout. From a more practical, and less physiological standpoint, it's also part of the price we pay for squeezing the old 10 day cycle of the Ultimate Diet into 7 days. Since we're cramming the 3 day carb-load of the original Ultimate Diet into 30 hours or so now, we have to train the entire body to ensure maximal glycogen storage. There is also the issue of increased ribosome activity and number which will also only occur in the muscles that are trained. To prepare all muscle groups for the growth inducing power workout in 2 days, a full body workout is required.

Your goal during this workout is sets of 6-12 reps which should be somewhere between 70 and 85% of your 1 repetition maximum. Rather than go to full failure, you should stop one repetition short. This is to avoid excessive neural fatigue that would hamper Saturday's power workout. Pick at most two exercises for legs, chest, back and shoulders, and only one exercise for arms (or two exercises worked for one set each). Not including warmup sets, you'll be doing 4 sets for legs, chest, back, and shoulders and 2 sets for arms. A little calf work and you're out of there. So this should be about a 20 set total workout. You should take 1-2 minutes between sets and be done within an hour. It may not sound like much, but, trust me, you'll be trashed by the time it's over. A sample workout appears below.

Sample Thursday Workout

Leg press: 2X6-12
Leg curl: 2X6-12
Leg extension: 1-2X6-12
Seated leg curl: 1-2X6-12
Calf raise: 3-4X6-12
Bench press or chest press machine: 2X6-12
Cable or machine row: 2X6-12
Incline bench press: 1-2X6-12
Pulldown or chin: 1-2X6-12
Lateral raise: 2-3X6-12
Biceps curl: 2X6-12
Triceps pushdown: 2X6-12

I should mention that some people report intense nausea during this workout. This most likely has to do with the high amount of lactic acid generated. To avoid problems with this, you can take a little longer between sets or alternate bodyparts in superset fashion. Putting leg work at the end of the workout also seems to help.

Day 4: Thursday PM: The carb-load

Carb-loading has been intensely studied since the 60's when it was found that depleting muscle glycogen (with a low-carbohydrate diet and intensive exercise) followed by a high-carb intake could increase muscle glycogen levels above normal enhancing endurance performance. Since that time, research has continued looking at issues of type, timing and amounts of carbohydrate as well as other factors.

As I mentioned before, the original UD took 3 days to carb-load. It started with relatively reasonable amounts of carbs and increased over the span of 3 days ending in an all you can eat junk food fest. If anything, this order was backwards: the high GI carbs should come first and taper down to lower GI carbs. The Bodyopus diet compressed the carb-loading period into 48 hours and was meticulous about the amounts, types and timing of carbohydrate intake. You started with large amounts of high GI carbohydrates and, over the length of the carb-load, you switched to smaller amounts of low GI carbs.

The UD2 manages to compress the carb-load into just under 30 hours. Admittedly, we might achieve slightly higher glycogen levels by extending the carb-load (actually, you'll be doing just that over Saturday and Sunday) but there is a rapidly reached point of diminishing returns. Recent research has shown that 100% glycogen repletion (not quite supercompensation) can be achieved within 24 hours as long as two requirements are met. While you might achieve small percentile increases in glycogen levels with longer carb-loads, they are out of proportion with the time and energy invested. Since we only have 7 days, we're going to hit 100% compensation in 24-30 hours and do the rest of the carb-load over the weekend.

So what are the requirements to reach glycogen compensation within 24 hours? The first is a high intensity workout, as this upregulates glucose transport and enzymes of glycogen storage and synthesis. The second is sufficient carbohydrate intake. The Thursday high intensity workout meets the first criteria, as described above. Now we need to talk about the carb-load itself. The main issues are total intake, type, and timing of carbohydrates. Let's look at each.

Amount of carbohydrate

For the most part, total carbohydrate intake is the key aspect, so let's look at that first. Assuming full glycogen depletion, which you should have achieved if you followed the recommendations, somewhere between 12 and 16 g/kg of lean body mass is the magic number here. That works out to approximately 7-8 grams of carbs/lb of lean body mass for the metric impaired. A lighter lifter with 70kg (154 lbs) of LBM will be eating 1000-1200 grams of carbohydrates over this 24 hour span from Thursday night to Friday bedtime. Larger lifters consume more and lighter lifters consume less.

In addition to all of those carbohydrates, don't forget protein at 1 gram per pound and low to moderate amounts of dietary fat; meaning about 15% of total calories or about 50 grams or so. Unsaturated fats such as olive oil seem to give a better carb-up but saturated fats let you eat more garbage (donuts and pizza anyone?).

Now, if you work out the calories amounts involved, you'll realize that they are extremely high. Even our lighter lifter might be consuming 4000-4800 calories from carbs alone, with an additional 600 calories from protein and another 500 or so from fat. That's 5000-6000 calories and probably double his maintenance calorie requirements. Larger individuals may be consuming significantly more. You may be asking yourself what keeps him from getting fat. The short answer, of course, is partitioning. With all of these machinations, we're controlling where all of those incoming calories are going to go. With full glycogen depletion, the body's first priority is glycogen repletion, calorie storage in fat cells is purely secondary. As I mentioned two chapters back, the two workouts further ensure that incoming calories are shuttled primarily to muscle, leaving less to go to fat stores.

During the Friday period, we also get to take advantage of another neat metabolic trick. Normally when you're eating lots of carbs, they get used for energy and fat gets stored. However, when glycogen is depleted, as it will be going into Friday, carbs go to glycogen synthesis first, and energy production second. This effect lasts for about 24 hours (or until glycogen is restored to normal levels) before it's gone.

This means that, for short periods, you can actually overeat carbs, and continue using fat for fuel. Back when people were playing with the Bodyopus diet, I remember folks eating literally 7,000-10,000 calories during the first day of their carb-load and still losing bodyfat. I don't recommend you start with something that radical but you should see how far you can push up the calories/carbs today without putting any fat back on. One of the keys to avoiding fat gain during this day is avoiding a high fat intake. It's not as fun, mind you, but it works better.

Type of carbohydrate

In terms of types of carbohydrates, most research suggests that as long as total amounts are sufficient, it just doesn't matter much. Sure, if you're looking at short-time periods (6 hours between twice daily workouts), it matters hugely what types of carbs you eat. In general, you probably want to start with high glycemic index liquids such as glucose and glucose polymers and move more towards starches as time passes. But, again, over 24 hours, as long as you meet total intake requirements, it won't matter as much.

However, people sometimes do notice subjective differences in carb-load quality depending on the types of carbs eaten. Generally, carb-loads based around large amounts of fructose or sucrose (which is half fructose) give inferior results. Fructose is used preferentially by the liver and tends not to be as good at refilling muscle glycogen. Unfortunately, this limits a lot of junk foods which are either high in sucrose or fructose (usually as high fructose corn syrup). You can eat some, but don't make them the entirety of your carb-load.

At the same time, small amounts of fructose (perhaps 50 grams over a 24 hour period) or sucrose (100 grams over a 24 hour period) seem to improve the carb-load. I have personally had my best (qualitatively) carb-loads eating primarily starches (bagels, milk, pasta) with small amounts of sucrose (usually some type of sherbet or high sugar cereal). The key is to keep starches dominant with small to moderate amounts of fructose or sucrose. This should let you satisfy any nagging cravings you have without ruining the quality of your carb-load.

Timing of carbohydrate intake

The third issue to consider is timing. Bodyopus required dieters to eat every 2 hours; this included waking up in the middle of the night. For the most part, assuming people got sufficient total carbohydrates, this didn't seem to have a huge impact on results; whether people woke up in the middle of the night or not, they got about the same level of glycogen storage. At the same time, there is a limit to how quickly glycogen can be synthesized and spreading out your carbohydrate intake over the 24 hour period just makes sense.

So say we have our lifter above, who needs to consume 1000 grams of carbohydrates over a 24 hour period. There should be time for 2-3 meals after the Thursday workout and at least 6 or 7 more the following Friday. So 8-10 total meals containing 100-150 grams of carbohydrates/meal would be sufficient. If our lifter wanted to wake up in the middle of the night to eat, he could increase the number of meals to 12 over a 24 hour period. 75-100 grams of carbs/meal would be sufficient.

What I personally find works best (since I hate setting an alarm) is to include some type of liquid with each meal. For example, I might have two large bagels (50 grams of carbs each) with a protein shake. This usually ensures that I wake up 2-3 hours later to pee at which time I'll repeat the meal. This wakes me up again 2-3 hours later to pee and I'll eat again. Then it's time to wake up on Friday and I start my normal eating schedule.

A sample carb-load

So let's look at a sample carb-load. Immediately following the Thursday workout, you would want to have a post-workout shake containing about 1 g/lb of carbohydrates and 1/3rd as much protein. For our 150 lb lifter, that's 150 grams of carbs and 50 grams of protein. The carb sources should be from glucose or glucose polymers with some fructose. Twinlab UltraFuel or Unipro Carboplex are some examples. Whey protein would be the best choice here since it will get amino acids into your system the most quickly. It will also increase insulin secretion. If you're going to creatine load, go ahead and put 5 grams of creatine in this drink.

2 hours later, either repeat the drink or have a normal meal of fairly high carbs (again, about 1 gram per pound of lean body mass), moderate protein and low fat. If it doesn't interfere with your bedtime, a third meal (or shake) at bedtime would be effective. As above, if you wake up to use the bathroom anyhow, you can go ahead and eat again in the middle of the night. If not, make sure to have a large carb-based breakfast first thing in the morning. Every 2-2.5 hours during this day (Friday), plan to eat again. By the end of Friday night (or whenever you end your carb-load), just make sure you've gotten the necessary 12-16 g/kg of carbohydrate.

You should be prepared for some pretty large scale energy and blood glucose swings during the day. Coming off of low-carbs, most people get really tired and fatigued during carb-loading, because their blood glucose is swinging up and down wildly (the increased carbs also raise serotonin which tends to make people drowsy). This might be one reason for you to shift the entire cycle forward one day, so that the carb-load is on Saturday, where your work performance may not be compromised.

You don't want to use thermogenics today although small amounts of caffeine may help to keep you awake. Thermogenics like ephedrine and clen impair insulin sensitivity which will limit the effectiveness of your carb-load. High doses of caffeine do as well so try to limit it if at all possible. Finally, make sure and drink lots of water today. Although you should be doing that every day anyhow, it’s even more critical today. Each gram of glycogen you store stores 3-4 grams of water along with it, so ensuring proper water intake will make sure your muscles are as full and hydrated as they can be. This will make you stronger for Saturday's power workout for reasons I've described before. Increased cellular hydration may also be anabolic in its own right.

Other additions today would be creatine loading (20 grams spread out through the day), which has been shown to increase glycogen storage by 20-25%. In fact, considering how cheap bulk creatine is, I highly recommend creatine loading during this phase. Even if it doesn't increase glycogen storage, it will make you stronger for the Saturday workout. One guinea pig (who used the UD2 to prep for a powerlifting contest) found that carbohydrate + creatine loading for 1 day made him as strong as just carb-loading for 2 days.

An insulin sensitizer such as alpha lipoic acid (200-600 mg with each meal, which gets very pricey fast) would be useful here too. Compounds which increase insulin output could conceivably help as well. Recent research has implicated vinegar in helping with glycogen storage. Taking a "shot" of it with each carb meal is an extremely cheap (albeit not very tasty) approach for the natural lifter to try.

For bodybuilders who want something a little stronger, and don't care about the high risk, injectable insulin can be used along with much higher amounts of carbohydrates to get even greater glycogen compensation. Humalin R or the new Humalog would be taken 10-15 minutes before a meal at a dose of 1 IU for every 10 grams of carbs to be consumed. Alternately, one of the biguanide drugs (a diabetic drug which increases insulin output from the pancreas) could be used to enhance insulin secretion. Note that they are difficult to predict in terms of their effect on blood glucose and insulin levels. A fast acting testosterone would also increase glycogen storage as well as helping your body to re-establish optimal anabolism. Think testosterone suspension at 100-150 mg/day.

"Natural" lifters could try to achieve a similar effect with low-dose prohormones taken frequently throughout the day to achieve a similar effect. Men should use one of the diols at 200-300 mg taken every three hours during the day, women the diones at maybe 100 mg every three hours during the day. One of the topical prohormones might be workable although they tend to take longer to achieve steady blood concentrations.

To be honest, if you get enough carbohydrates in within 24 hours, most of the above seems to be fairly irrelevant. I've had testers carb-up with and without insulin, vinegar, creatine and the rest and none of it seems to impact on glycogen compensation to a great degree. Getting the necessary carbs (12-16 g/kg lean body mass) is the most crucial aspect.

Finally, no workout today. If you worked out hard enough last night, you shouldn't want to train anyhow. Don't be surprised if you're sore from the intensity workout from yesterday. Just rest and eat. And eat. And eat. And enjoy the looks people give you when you tell them that you're eating Captain Crunch on your diet.

Chapter 12: The weekend

Before I talk about Saturday's diet and training, I want to mention something else: Saturday morning can be used to gauge how well (or poorly) your carb-load went. Ideally, you should wake up looking full and lean, with no bloatedness or water retention underneath the skin. If not, you may have eaten too many total carbs (once glycogen storage maxes out at 16 g/kg, more are neither necessary nor beneficial) or too many of the wrong types of carbs (too much sucrose or fructose).

There is also some individuality in what foods people best carb-load on. One friend of mine bloats when she eats rice, but carb-loads just fine on other sources. As I mentioned earlier after one or two trial runs, you should have your optimal food choices for the carb-load dialed in. If you're tracking weight, you should find that your bodyweight has rebounded to just about what it was at the start of the cycle (weight can fluctuate a good 6-7 lbs between carb-depletion and compensation). Yet you should look and be leaner. If you don't accomplish this, look at the quality of your carb-load. Perhaps you ate too much sucrose or fructose or too much dietary fat. If so, make adjustments until you nail it.

Day 6: Saturday

So now it's Saturday. You've upregulated ribosome activity and number so your muscles are prepared for a high tension, growth promoting workout. You are both carb and creatine loaded and have had a day of rest, you should feel very strong and your muscle should be full.

Which is good because today is the day for all the macho lifters who like to move heavy weights around. That's because Saturday is the power workout. As with Thursday, you'll be training your full body today again. If the idea really offends you or you can't give maximal output with the full body approach, you can train half the body in the morning, and the other half in the evening. I don't think this is ideal but it is workable. Put priority bodyparts in the morning, and less important bodyparts in the evening. Or do legs/chest/back in the morning and shoulders and arms in the evening. Or go old school/Hardgainer style and just train legs, chest, and back and screw direct delt and arm work. As long as it all gets trained, it won't matter. As with day 4, the 7 day schedule precludes you from using a split routine across two different days.

You'll want to get at least one meal in you, with moderate amounts of carbs and protein about 2-3 hours before this workout. This will top off muscle and liver glycogen and make sure that blood glucose levels are optimal for the workout. You'll feel stronger if you do this. 30 minutes before, take 30 grams carbs with 15 grams of whey protein and some creatine (just like on Thursday night) as this may help increase anabolism.

A stimulant such as ephedrine/caffeine, or caffeine/tyrosine an hour before the workout will also make you stronger and able to inflict a greater tension stimulus to your muscles. Sipping on a dilute carb drink (think Gatorade, just get 30-60 grams of carbs in 32 oz. of water) during the workout will help get you through it, as well as helping keep you in an optimal anabolic state.

Now, power training can take many forms. On average, you may be looking at anywhere from 3-6 (up to 10) sets of anywhere from 3-6 reps on average. Since we are working the entire body, and don't want to spend all day in the gym, we have to condense this a bit. Even then, with the long rest periods involved, the workout will probably take an hour and a half or so. Maybe two hours if you dawdle or are using extremely heavy weights and need a lot of warmup sets.

Somewhere between 3-6 sets of 3-6 reps per bodypart is probably about right. Since shoulders and arms get hit with all heavy pressing and pulling exercises, you can do relatively fewer sets (or skip them altogether as I mentioned above). Or work them with slightly higher repetitions. So you might pick 2 exercises per bodypart (i.e. flat and incline bench for chest, row and pulldowns for back) and work each for 3 sets of 3-6 reps. Or you could pick a single exercise and work it for 6 sets of 3 or perhaps 5 sets of 5. Just get in that range and you should be fine.

A sufficient warmups is important to work this heavily safely, but don't dawdle or waste energy. Several low-rep (5 reps or less) warmup sets pyramiding up is your best bet. So if you were squatting 315X5, you might do 135X5, 185X3, 225X3, 275X1, and then move into your work sets.

During the sets, you want to lower the weight under control (2-4 seconds down) and then try to lift it as quickly as possible. It won't move that quickly because it's too heavy, but this guarantees that as many muscle fibers as possible are recruited and subjected to the heavy tension. On that note, don't just heave the weights around; you still want to make sure that you're putting tension on the target muscles. Using so much weight on your barbell curls that you turn it into a low-back exercise won't do you any good in terms of training your biceps. Neither will doing a heave-ho, bounce off the sternum, bench press. Move as quickly as you can during the concentric while keeping tension on the target muscles.

You also want to take fairly long rest periods between work sets, 3-5 minutes if possible, but without making the workout too long. If your gym allows it, alternate supersetting exercises is an excellent way to work efficiently.

By alternate supersetting, I mean this: set up your workout so that you've paired exercises that are antagonistic to one another. Flat bench and rows would be one example. So after a heavy set of bench, rest 2 minutes, then do a heavy set of rows, rest 2 minutes, back to bench, rest 2 minutes, back to rows, until you've completed all of your sets. The move to the next exercise.

This gives you a full 5 minutes between sets of bench without you having to just sit around picking your nose waiting for your nervous system to recover. This is also the workout where you should try to increase the weight each week during the cycle, so that you continue to exert a novel tension stimulus to your muscles to stimulate growth. You should be strong enough after the carb/creatine load that this isn't too much of a problem. At the very least, you should be able to maintain your strength without loss during your diet cycle.

For joint safety, you should pick basic, compound exercises such as squats (or front squats or deadlifts), bench and incline presses, rows (bent over or machine) and pulldowns or chins for back, shoulder presses or upright rows for shoulders, and barbell curls and close grip bench presses for arms. At most two exercises per bodypart is sufficient, although arms should only need one. This should give you 4-6 heavy sets for legs, chest, back and shoulders, and 2-3 for arms. Using Charles Poliquin's notation (exercises with the same letter are alternate supersetted as described above), a sample workout might be:

Sample Saturday Workout

	Exercise	Sets	Reps	Rest period
A1.	Squat or deadlift:	2-3	3-6	2 minutes
A2.	Calf raise	2-3	3-6	2 minutes
B1.	Flat bench	2-3	3-6	2 minutes
B2.	Bent over row	2-3	3-6	2 minutes
C1.	Incline bench	1-3	3-6	2 minutes
C2.	Pulldown or chin	1-3	3-6	2 minutes
D.	Front squat or leg press	1-3	3-6	3 minutes
E1.	Shoulder press	2-3	3-6	2 minutes
E2.	Rear lateral	2-3	3-6	2 minutes
F1.	Barbell curl	1-2	3-6	2 minutes
F2.	Close grip bench	1-2	3-6	2 minutes

So you'd alternate squats with calf raises, flat bench with row, incline bench with pulldowns/chin, shoulder presses with rear laterals, and barbell curls with close grips. Front squats or leg press would be worked by itself. Or superset it with seated calf raises or something.

After the workout, go ahead with your normal post-workout drink, carbs plus protein and some creatine or whatever else you want to throw in there. You can expect less muscular fatigue than from the other workouts but don't be surprised if you're simply exhausted. It's simply a different kind of fatigue than from higher rep training.

As far as diet on Saturday, a lot of the specifics (especially caloric intake) will come down to goals. The tradeoff basically comes between maximal growth and the potential of regaining bodyfat (or slowing its loss). On average, I'd say a carb intake of 4-5 g/kg (about 2-2.5 g/lb) lean body mass is appropriate with most, if not all, of the carbohydrates coming from complex sources. Of course, this goes along with the standard 1 g/lb of protein and fairly low fat (40-50 grams from predominantly monounsaturated sources). This should be roughly maintenance calories for most dieters and works out to roughly a 60% carb, 25% protein and 15% fat diet or so. Just a typical bodybuilding/athletic diet day. I should add that dieters who want to accelerate fat loss can cut calories by 10-20% (the cut should come from carbohydrates) on this day. This will limit growth potential but may be necessary if maximal fat loss is the goal.

Day 7: Sunday

Sunday is another day of rest, before the start of the next cycle, but you have a couple of choices regarding diet. As with Saturday, if your goal is maximal growth/anabolism, it would be best to eat at roughly maintenance calories. However, it would probably be best to cut carbs back to about 2-3 g/kg (1-1.5 g/lb) lean body mass with protein at 1 g/lb and dietary fat making up the rest. This will put you at roughly Zone or Isocaloric proportions. Early in the day, you can eat small amounts of complex carbohydrates but this should be shifts towards lower GI veggies as the day progresses.

For maximal fat loss, calories can be reduced by 10-20% (or more) on this day with a return to low-carbohydrates in the evening. In practice, what seems to work best is to eat small amounts of carbohydrates for the first half of the day (breakfast and lunch) and go back to dieting low-carbs (vegetables only) at night.

And although it will impair recovery, a cardio session late Sunday evening will help to deplete liver glycogen, lower insulin and start setting up the next fat loss cycle. As always, use your own recovery capabilities and goals as your guide. It should be obvious that any of the supplements or drugs used during the other low-carbohydrate days could be used here (including EC or clen in the afternoon, glutamine for GH release at bedtime, etc). Then, of course, you're back to day 1.

Chapter 13: UD2 Variations

Although I feel that the UD2 is close to optimal the way it's currently set up, there are a few situations where you may want to make some slight adjustments to it.

Modifications to the UD2 for fat loss

There are times that the main UD2 schedule may need to be adjusted even for fat loss. I mentioned one example in the last chapter: folks who find that the carb-load wipes them out (by causing fluctuating energy levels) may wish to move the entire cycle forward to put the carb-load on Saturday. That would make the schedule look like this:

	Training	Diet
Tuesday/Wednesday:	Depletion	Low-carb/low-calorie
Thursday/Friday AM:	Cardio	Low-carb/low-calorie
Friday PM:	High intensity	Begin carb-load
Saturday:	No training	Carb-load
Sunday:	Power workout	Diet variable
Monday:	No training/cardio	Diet variable

A second option is for dieters who just don't like the way Thursday is set up. Psychologically, many dieters seem to find a partial day of dieting to be a waste. They would rather have a day that is either just dieting or just refeeding. Whether valid or not, this is a real occurrence and I'm going to give you a solution.

This variant also applies to folks whose schedules require them to workout in the morning, making a Thursday night workout impractical. In this case, I suggest making Thursday a normal low-carbohydrate/low-calorie dieting day with cardio. The high-intensity workout gets moved to Friday morning and the carb-load is scheduled for Friday to Saturday morning. This also means that there are only 24 hours between the high-intensity workout and the power workout, but this is fine; you'll be stronger than you think and you'll still get a growth response. Bodybuilders have a block against training a muscle two days in a row, but other athletes do it all the time. The full schedule would be:

	Training	Diet
Monday/Tuesday:	Depletion	Low-carb/low-calorie
Wednesday/Thursday:	Cardio	Low-carb/low-calorie
Friday:	High intensity in AM	Begin carb-load
Saturday:	Power workout	Diet variable
Sunday:	No training/cardio	Diet variable

A related option, for folks who can't train on Saturday morning would be to move the power workout to Friday night, 24 hours after the Thursday night high-intensity workout. This would leave Saturday and Sunday off with the diets described as per the last chapter.

Using the UD2 for mass gains

Although the UD2 is primarily aimed at fat loss with muscle maintenance (or even gain), it can also be used with slight modifications for muscle gain while minimizing bodyfat gain. Recall from the chapters on partitioning that the same mechanisms that make fat loss without muscle loss a problem for the genetically average also conspire to cause more fat gain when muscle is gained. The same processes that the UD2 temporarily improves (notably calorie partitioning) can be used for muscle gain.

The main change, of course, would be in calorie levels. Although I didn't talk about it, the original Ultimate Diet was borne from Michael Zumpano's Rebound Training. Rebound training was essentially the same plan but with higher calorie levels on all of the days.

For the UD2, I think a slightly different approach is better. For mass gains, I'd suggest several changes. The first would be both an increase in calories (to maintenance or maybe 10% below) AND carbohydrates (to 100 grams/day) on the low-carbohydrate days. This will avoid ketosis more or less completely while still allowing glycogen depletion. Along with that, changing the Monday and Tuesday workouts to more of high-rep tension workouts is also an option. Still use sets of 12-15, but with heavier weights and slightly fewer sets. This will still cause glycogen depletion (although not as much) while instilling more of an anabolic response. In this case, I'd also suggest using more of a traditional two way bodybuilding split routine (see example below). Small amounts of cardio could be done on Wednesday and Thursday morning to keep fat gain to a minimum.

Thursday and Friday would stay the same as would the Saturday workout. Additionally, you would eat at maintenance or slightly above on the weekend. The overall plan would look like this:

	Training	Diet
Monday:	Chest: 6-8 sets of 12-15	Maintenance calories
	Shoulders: 4-6 sets of 12-15	or 10-25% below
	Triceps: 4 sets of 12-15	100 g carbs/day
Tuesday:	Quads: 6-8 sets of 12-15	Maintenance calories
	Hamstrings: 4-6 sets of 12-15	or 10-25% below
	Calves: 6-8 sets of 12-15	100 g carbs/day
	Back: 6-8 sets of 12-15	
	Biceps: 4 sets of 12-15	
Wednesday:	Cardio optional; 30-40 minutes max.	Maintenance calories
Thursday AM:	Cardio optional: 30-40 minutes max.	Maintenance calories
Thursday PM:	High intensity workout	Begin carb-load
Friday:	No training	Carb-load
Saturday:	Power workout	Maintenance+10%
Sunday:	No training	Maintenance+10%

Using the UD2 for endurance competition

There are times when endurance athletes need to drop bodyfat while trying to maintain performance, just like other athletes. While most tend to stick with reduced calorie/carbohydrate based diets, this can cause the same sets of problems that occur in natural bodybuilders of average genetic disposition. The UD2 can solve those problems. Additionally, current research has shown that following a 5 day low-carbohydrate period with a 1 day carb-load can actually **increase** performance in some endurance athletes. The low-carb days increase fat utilization while the carb-loading period increases muscle glycogen for high-intensity efforts. The UD2 fits that basic schedule.

Now, a fairly recent approach to endurance training (especially among cyclists) is called block training (see "Performance Cycling" by Dan Morris for more information). Rather than the standard hard day/easy day approach, block training advocates that athletes do two or three high intensity workouts followed by several low-intensity workouts. This fits the schedule of the UD2 quite well. A possible schedule (assuming no weight training, just endurance sports) would be as follows:

Monday: Long-duration, exhaustive endurance workout such as 1-2 hours at 75-80% of maximum heart rate. You could finish with several medium duration (2-3 minute) intervals to deplete fast twitch muscle fibers. This actually represents the third day of the training "block" since you'll still be carb-loaded; it will be used for glycogen depletion. Think 1-2 hours at a reasonably high training intensity. Diet is low-carbohydrate and low-calorie.

Tuesday/Wednesday/Thursday AM: low intensity aerobic training, 30-60 minutes at 65% of maximum heart rate. Diet is low-carbohydrate and low-calorie. This represents your three easy days of recovery following the hard training block.

Thursday PM: warmups followed by 5X1 minute intervals at 95% of max effort. This finishes glycogen depletion and sets up for the carb-load.

Friday: No training, carb-load

Saturday: High intensity training sessions. Could be intervals, or time trialing or even extensive endurance training. This is the first day of the training block. Diet as described in the previous chapter. You would eat at maintenance for optimal performance or 10-20% below maintenance for maximal fat loss.

Sunday: Another high intensity training session. This is the second day of the training block. Diet as described in the previous chapter. You would eat at maintenance for optimal performance or 10-20% below maintenance for maximal fat loss.

Obviously, if as an endurance athlete you are also performing weight training, you can use a more standard version of the UD2: using high repetitions on Monday and Tuesday for glycogen depletion, a tension workout on Thursday, and a heavier workout (or just endurance training) on Saturday.

The UD2 for other athletes

Other athletes can probably utilize the block training approach as in the endurance athlete example above. The key is to put the hardest weekly workouts on Saturday, Sunday and Monday (when you have plenty of carbs and calories to train) and lower intensity workouts on Tuesday through Thursday. A short-high intensity workout should precede the carb-load as in the example above.

I'm sure creative readers can come up with even more variations than what I've touched on above. Frankly, I'd be happier with an 8 day cycle, 4 days of low-carbohydrates and low-calories followed by 4 days of overfeeding with training. It would look more or less like this.

	Training	Diet
Day 1/2:	Depletion	Low-carb/low-calorie
Day 3/4:	Cardio	Low-carb/low-calorie
Day 5:	High intensity in AM	Begin carb-load
Day 6:	No training	Continue carb-load
Day 7:	Power workout in AM	Diet variable
Day 8:	No training	Diet variable

It's basically just like the normal 7 day cycle but the oddity of day 4 (the combo day) is remoed: you have 4 strict diet days, 2 days of carb-loading and 2 days of relatively normal eating. You also get a full 48 hours of rest between the high intensity workout and the power workout. Of course, the cycle precesses through the week each cycle, meaning you don't get to eat normally on weekends. But for lifters with no life, who can schedule eating and training on any day, this may be a better way to go. For everyone else, the compromise of fitting the cycle into 7 days mandates the current setup.

Chapter 14: Final commentary

Like Bodyopus and other cyclical diets before it, the UD2 is a particularly energy and effort intensive approach. It requires more attention to detail than your typical diet but it also happens to produce results. The feedback I've gotten from testers has surpassed even my original hopes, consistent fat loss of 1.5 pounds per week with no muscle loss (or even gain); one athlete even prepped for a powerlifting meet, leaning out to make his weight class and getting stronger all the while. It seems to work best for the individuals who have poor results with more traditional approaches.

However, also like Bodyopus before it, the UD2 is a sledgehammer approach; it uses nutritional tricks to avoid the problems with partitioning I discussed in previous chapters. It doesn't fundamentally solve them, it just lets the genetically average side step them for a brief period to achieve a higher level of development. Integrated diet and training programs are a step in the right direction but they can only go so far.

In Bodyopus, Dan mentioned that future approaches to the problem should pinpoint the specific defects in nutrient partitioning at the cellular level so that they could be corrected. In my Bromocriptine book, in focusing on the central (brain) mechanisms involved in calorie partitioning, I predicted that the future of body recomposition would be in modifying neurochemistry. Both would require either supraphysiological doses of nutrients, drugs, or gene therapy.

Products are starting to appear and research is always moving forward. As we learn more about the specific pathways involved in partitioning at the cellular level we may come closer to being able to control or modify them. Drugs, of course, are still the easiest (and frequently the cheapest) option but modern morality and laws prevent their use by the majority. Other approaches are required.

My friends at Avant Labs (warning: shameless plug coming up) have developed a product called Leptigen that they claim will partition calories away from fat cells and towards muscle cells. I'm sure other companies will jump on the bandwagon as well. While I always remain skeptical, I hope that such products do what they claim. Until that time, extreme approaches such as the UD2 may offer the only body salvation for the genetically average individual; in the future, less extreme approaches will be required. Or so we can hope.