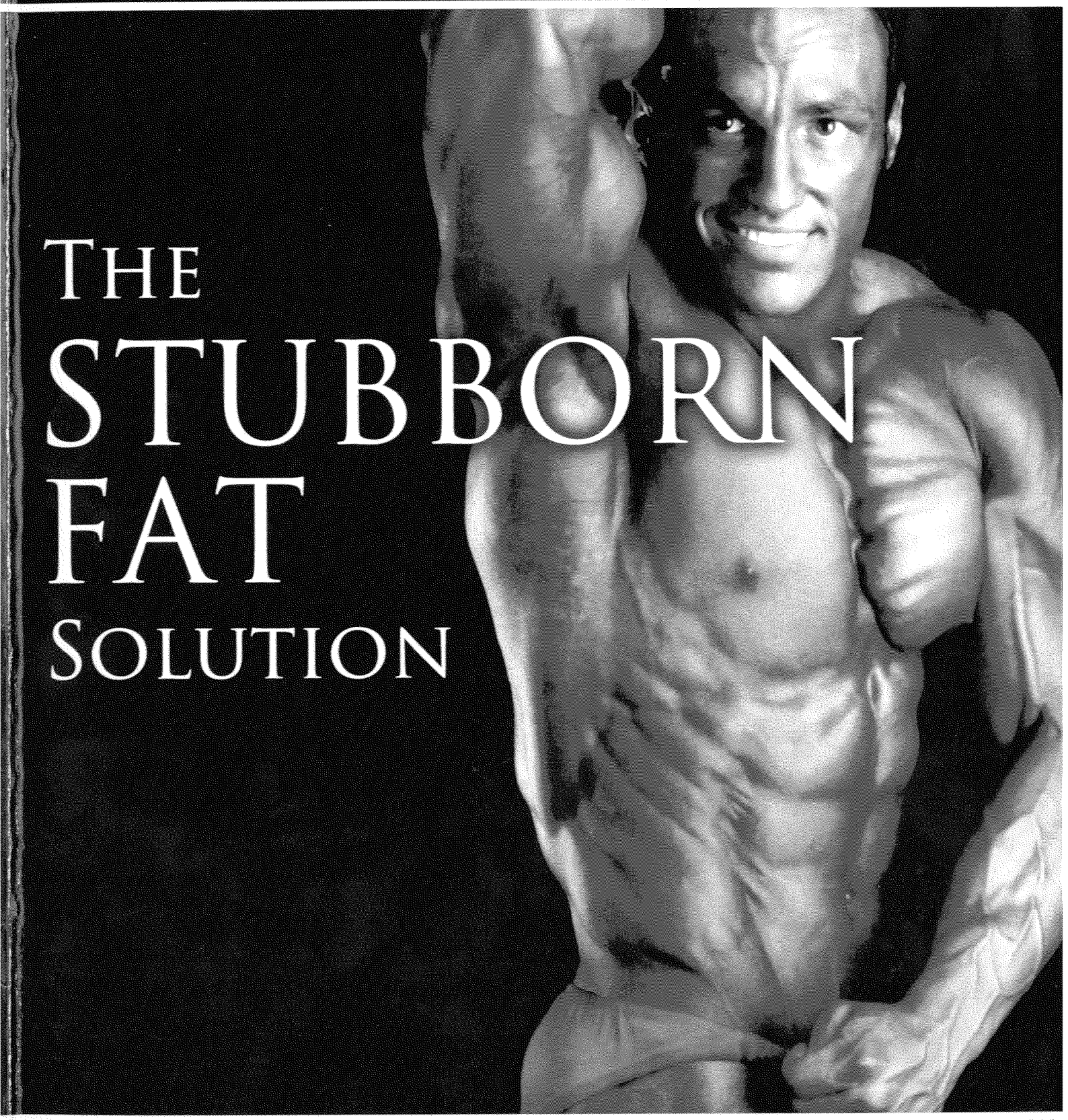
Lyle McDonald



The Stubborn Fat Solution

BODY RECOMPOSITION
www.bodyrecomposition.com

The Stubborn Fat Solution

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. Use of the guidelines herein is at the sole choice and risk of the reader.

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Introduction

For decades (if not longer), bodybuilders and those seeking what they consider an ideal physique have struggled with the issue of stubborn body fat. Females have typically had the biggest problems leaning out their legs. As Dan Duchaine commented years ago in **Bodyopus**, it's not at all uncommon to see a female bodybuilder who looks like she's had two different half-bodies stapled together. She'll be ripped up top with smooth (a polite way of saying 'too damn fat') legs. Even non-bodybuilding females who only want to get athletically lean often end up losing all of the fat from their upper bodies, while their lower bodies don't change at all.

Men, under a variety of circumstances, can run into the same issues. Abdominal and low-back fat along with the obliques are often a problem. While not nearly as stubborn as female lower body fat, men's stubborn fat can still be tough to get rid of. Some men have fat deposition patterns more like a woman's to begin with; losing lower body fat is just as difficult for them as it is for females.

As with so many aspects of physique enhancement, there is a lot of lore and outright bullshit when it comes to the topic of stubborn body fat and what to do about it. Estrogen is all too commonly blamed for female stubborn fat problems, cortisol gets blamed for ab fat. If things were that simple, all it would take is an anti-estrogen or an anti-cortisol compound to get rid of it. In the real world, while this may help a bit, it doesn't cause magic to happen. Ergo, those are not the real problems.

Rather, stubborn body fat is stubborn for a myriad of interconnected physiological reasons which, over the past several years, research has helped to delineate. Stubborn fat not only stores calories more effectively, but it is hellishly resistant to giving it up. That's why it's stubborn.

To understand how to get rid of stubborn body fat, first we have to understand what causes it and why it's there in the first place. This means that, as usual, I'm going to bore you with a lot of background physiology about body fat and stubborn body fat specifically. If you've already read my **Ultimate Diet 2.0**, some of it will sound familiar but I'm going to go into much more detail here.

If you learn nothing else from my physiological ramblings, you'll at least learn why one of the single most common approaches to stubborn lower body fat is also one of the most ineffective. Put differently, what often works at least adequately for men's stubborn abdominal fat is generally utterly worthless for targeting lower body fat. I don't usually feel that there are huge differences in how men and women should train or diet, but getting rid of lower body fat is one area where there are differences that must be appreciated for optimal results.

After I dissect the physiology of stubborn body fat in my typical obsessive way, I'm going to present four separate approaches to dealing with stubborn body fat. This simply reflects the fact that not every solution is appropriate for every individual. How you're training, your dietary and supplement preferences, and other factors have to be taken into consideration to determine which of the four approaches is going to be best.

Many of you may have come across mention of the Stubborn Fat Protocol (SFP) on the Internet. Sometimes folks are even decent enough to credit me and call it Lyle McDonald's SFP. That, along with two other approaches are already somewhat commonly known. However, in this book I'll introduce the newly developed Stubborn Fat Protocol 2.0 which no one outside of my test subjects knows about. I'll tell you up front that the SFP2.0 is not for everyone and not everyone can do it. For those who can handle it, there's nothing more effective at stripping stubborn body fat.

In any case, by the end of the book, you'll have all of the tools you need (training, diet, supplements, etc.) to get rid of stubborn body fat once and for all.

Defining the Problem

Several of my books have started with a chapter defining the problem that I'm going to address, and this one is no different. Assuming you're reading this at home, I want you to go get naked in front of the mirror, you might even want to jump up and down a couple of times (for God's sake, don't take video and upload it to YouTube).

What jiggled?

If you're a male with typical body fat patterning, the fat covering your abs (upper and lower), love handles and lower back did. You might have amazingly striated legs but the abs look smooth (again, a polite way of saying 'fat'). Odds are that when you gain any fat at all, it accumulates around your midsection. When you diet, everything else gets leaner but the abs just never quite come in.

If you have skinfold data, it probably looks something like

Pectoral: 3 mm
Thigh: 5 mm
Abdominal: 22 mm (holy shit!)

If you're a female with typical female fat patterning, it was the stuff on your legs that moved. If you're semi-lean, you might have striations in your delts and even have a visible 6-pack (or at least abs). Yet the thighs remain smooth and uncut with no definition or separation. When you gain fat, it's mostly in the lower body; when you diet, everything else gets ripped up but the legs just don't drop fat. Sometimes it even seems like your upper body is leaning out while your legs get fatter (I'll explain this later on). You may or may not have as extreme a variance between sites as a man; typically, the thigh is the biggest, triceps is next while suprailiac (above the hipbone) and abdominal are both fairly small.

Now, if you're an atypical male, you might have lower body fat problems (if you're very unlucky you might have problems with both ab fat *and* thigh fat). Some females show atypical fat patterns as well, either they have abdominal fat in addition to the normal hip/thigh fat or they have abdominal fat instead of the normal hip/thigh fat.

Extremely overfat females can often start to develop male fat patterning (with the health risks that go along with it). As a random tangent: am I the only one noticing that younger girls are starting to have more ab fat these days? Something odd seems to be going on hormonally there but I'm not sure what. Anyhow.

Now, if you're supremely lucky, you don't have any particular trouble spots. Rather you carry your fat fairly evenly. When you gain weight, it doesn't seem to accumulate in any one place; you can carry tremendous amounts of body fat without looking particularly fat. The advantage is that folks in this situation don't typically have trouble spots that are resistant to loss. The drawback is that it takes absolutely forever for any real appearance changes to occur. Except that when it happens, it seems to happen overnight. Smooth. Smooth. Smooth. Holy shit, I'm cut. The genetically elite are often in this category.

If you're in that last category, with very even fat distribution and no real trouble spots, I have two things to say to you. First, screw you. The majority of us with stubborn fat problems hate you, deal with it. Second, you don't need this book since you don't have stubborn fat problems in the first place. Not that the information won't help you or that you won't get something useful out of this book, but fundamentally any old approach will probably work just fine for you. Which is a nice segue into...

Who is this book for?

Like my **Ultimate Diet 2.0** (hereafter UD2), the information in this book is not meant for everyone. This is only for fairly lean individuals looking to get super lean. In fact, my requirements for who needs this book are even stricter than in the UD2 (which was for men of roughly 12-15% body fat and women around 21-24% body fat).

How lean do you need to be to need this book? For men, this will usually mean being 10% body fat or lower before needing to be concerned with this book's approach; for women, a body fat in the mid-teens (15-17% body fat) is probably about right. These aren't absolutes but are generally when the problems with removing stubborn body fat become particularly apparent.

If you're fatter than that, you simply don't need to worry about stubborn fat or the protocols in this book. As you'll learn shortly, the body always takes fat from the least stubborn places first; until you get rid of the easy stuff, there's no point in targeting the stubborn stuff.

I'd note that I don't expect the above paragraph to dissuade people. I still get folks asking if they can do UD2 when they are far above my body fat cutoffs; often they want to know if the information will still 'work'. The answer, generally, is that, yes the information will still work. It's simply that it's not necessary.

I always recommend that folks use the simplest, easiest, least labor intensive approaches to accomplishing a goal; the stubborn fat protocols are not in the category of simple, easy or non-labor intensive. Don't bother with them if you don't need them; that way you'll have them in your arsenal when you do.

Am I talking about spot reduction?

Spot reduction refers to the idea that, with some combination of specific exercise or what have you, the body will take fat from a specific area of the body. For example, everyone toiling away in those hour-long ab or hip/thigh classes hopes that by doing an hour of abdominal work, they can remove fat from the midsection. Women will often perform hundreds of repetitions on the inner/outer thigh (aka 'good girl/bad girl' machine) hoping it will trim their saddlebags. If only it were that simple.

While I'm on this topic, I should mention a recent study that a lot of people have been trotting out as proof of the concept of spot reduction. In it, researchers found that exercising a given muscle increased blood flow to the fat cells adjacent to that muscle. So working the legs increased blood flow to the thigh fat. As you'll learn later in this book, blood flow is a huge area of importance for getting rid of stubborn body fat. So why doesn't this study matter? The researchers estimated that 30 minutes of exercise might mobilize an extra 1 milligram (one thousandth of a gram) of extra fat from the adjacent fat cells. Unless you have 80 years to get lean, this simply isn't relevant.

So am I describing spot reduction in this book?

The short answer, of course, is no. Spot reduction is an absurd myth that refuses to die and is, fundamentally, a physiological impossibility. The protocols in this book aren't magically going to reduce abdominal or hip/thigh fat preferentially; that's not what I'm talking about.

Recall from above how I said that the body will pull fat from the easiest, non-stubborn places first; recall also how I said that only very lean individuals need worry about the information in this book and how to get rid of stubborn fat. This always holds and the protocols in this book can't change that pattern of loss. Rather, this book will facilitate getting rid of stubborn fat more easily and readily once a dieter is to the point where it has become an issue.

At this point you might be wondering why we even need to consider an approach to stubborn body fat in the first place? There are two primary reasons which are inter-related. The first and most obvious is that individuals who need to be super lean (for whatever reason) won't reach their appearance goals if they can't get rid of the fat. Traditional diet and training often fail in this regard and there are legions of dieters who did everything 'right' yet who failed to reach their fat loss goals.

But there's a more insidious reason that stubborn fat removal becomes important. When you're in a calorie deficit, your body needs to mobilize fuel. Anybody who has been super lean knows that muscle loss often accelerates near the end of the diet. There are a number of reasons for this (see my UD2 for a detailed discussion) but an inability to mobilize fat for fuel is one of them. Because the body can't easily get stubborn fat to use for fuel (and there's no other body fat left to use), in a deficit situation it will turn to muscle mass. You don't get leaner, you just get smaller.

By forcing the body to mobilize stubborn body fat for fuel, not only do you get leaner faster at the end of your diet, you spare muscle loss. This book is a targeted approach to

eliminate body fat (after everything else is gone) that is notoriously resistant to removal. **The Stubborn Fat Solution** accomplishes this by directly addressing the physiological reasons that makes it so difficult to remove in the first place. However, the protocols in this book will not let you side step how your body normally removes body fat.

It's not spot reduction in any sense.

So who are you?

So who is this book for? The ideal candidate for the information in this book is a male at 10% body fat or lower and women around 15-17% body fat or lower. Obviously, you should have your general diet and training under control and understand the basics of each. Your goal, of course is that you're trying to get to extreme levels of leanness and get rid of stubborn body fat.

Usually this means folks dieting down for a bodybuilding, fitness or figure contest but this isn't always the case. You might be a lean female or male model who needs to get rid of some stubborn fat. Maybe you just want to see your abs for once or are a female who wants to see cut legs just once. Please note: getting this lean even once will screw you up for the rest of your life; once you've been uber-lean anything above that feels fat.

Odds are you've tried other approaches, magic pills, potions and powders. Hour long ab, hip and thigh classes, wraps, creams, etc.; you name it and people have tried it. It didn't work because these approaches weren't approaching the problem in the right way. You're reading this because the other stuff didn't work. It goes without saying that my approach does.

What can you expect?

As I go through this topic in my normal way, I'm going to address a variety of topics related to diet, training and supplementation that can impact on stubborn body fat. However, while my UD2 was very, very specific and structured, you'll find that the information in this book is a bit more flexible.

As I mentioned, I'll be covering four *different* approaches to targeting stubborn body fat. Which you choose will depend on how you prefer to diet or train and which best fits your needs. You will, of course, have to change some aspect of your training or diet to achieve the goal of stubborn body fat loss but I imagine you've assumed that as par for the course.

If you've read through this chapter and think it describes you and your situation, read on. If not, read on anyway; as with all of my books, even if you don't apply a single bit of information in this book, you'll end up knowing more about the topic than most people on the planet.

This, of course, is primarily important in allowing you to win Internet arguments. And, at the end of the day, isn't that what it's really all about?

What is Body Fat

Most people think they know all they need to know about body fat; I'm here to tell you that that isn't the case. If you've read my articles, you may have seen some of this before but I want to make sure everyone is on the same page before I deluge you with the technical bits.

What it is?

The more technical term for body fat is adipose tissue, with individual cells being called adipocytes ('adipo' = fat; 'cyte' = cell). In humans, the primary type of fat cell is called white adipose tissue, or WAT, so named because of its color (it's actually sort of a milky yellow). While there is another type of fat, called brown adipose tissue or BAT (which is actually more of a reddish orange), it's generally been thought that humans didn't have much BAT and hence it could be ignored. As I'll discuss later, this fact has been brought into question by recent research. I'll come back to BAT in the next chapter.

I'd note that there is also fat stored within skeletal muscle, called intramuscular triglycerides (IMTG) that can be used by skeletal muscle for energy. Somewhat unfortunately, the body often burns IMTG before it will start using fatty acids from other sources (e.g. your fat butt) for energy.

More unfortunately, the loss of IMTG is part of why dieting makes dieters look muscularly flat as IMTG contribute to overall muscular volume. Within the bodybuilding subculture, many contest gurus are now using something called 'junk loading' (combining high fat and carbohydrate intake immediately before a contest); what they are trying to do is refill both muscle glycogen AND IMTG levels for maximum fullness.

Additionally, under certain conditions, the body can start storing fat in places it doesn't belong such as the pancreas, liver, etc. and this causes bodily damage. This generally only occurs under conditions of out of control obesity and weight gain. I'm not going to talk about IMTG or the fat that gets stored in the wrong places any more in this book since my focus is on removing WAT stored in the stubborn spots.

In humans, fat cell number can vary drastically. Lean individuals may have anywhere from 41 to 65 billion fat cells while obese individuals can have upwards of 200 billion fat cells; fat cell number can also vary based on genetics, race and the area of fat you're looking at. Contrary to popular belief, fat cell number can change up or down. Fat cells can range in diameter from 70 to 120 micrometers (one millionth of a meter) and it turns out that the size of the fat cell can affect its physiology.

WAT in humans is composed primarily (anywhere from 80 to 95%) of lipid. By lipid, I mean stored triglycerides (TG) which are simply a glycerol molecule bound to three free fatty acid (FFA) chains. The remaining part of the fat cell is comprised of a little bit of water as well as all of the cellular machinery needed to produce the various enzymes, proteins, and products that fat cells need to do their duty. As it's turning out, fat cells produce quite a bit of stuff, some good, some bad, that affects your overall metabolism.

For the record, one pound of fat is 454 grams and let's assume 90% lipid on average. So about 400 or so grams are actual stored TG. When burned by the body, one gram of fat provides 9 calories so 400 grams of fat contains about 3600 calories of stored energy. Now you know where the old axiom of ~3,500 calories to lose a pound of fat comes from.

What's it for?

Other than being considered unattractive in modern society, you may be wondering what role fat cells play in the body. The main role is as an energy storage dump. In fact, up until about 1994, the predominant view of fat cells was that this is all they were: a passive place for the body to store energy for times when there wasn't enough food available. This turns out to be drastically incorrect but, before we go on, let's look at that one aspect of fat cells.

Fat cells are truly exceptional in their capacity to store energy. As mentioned above, a single pound of fat contains roughly 3,500 calories of stored energy. Assuming you could burn 100% fat as fuel (you can't for reasons that aren't important now), this is enough energy for a 150-pound person to walk roughly 35 miles or so before using that energy. That's ONE pound of fat. Even at an extremely low 5 lbs of body fat, which is near the very low-end of how much a human might carry, you're looking at ~15,000 calories of stored energy. That's 150 miles of straight walking or so.

Here are some more numbers to give you a little better perspective. The average American male who may weigh 160 lbs with 15% body fat has 24 pounds of body fat which is about 84,000 calories or so of stored energy. He'll have a metabolic rate close to 2,400 calories/day. The amount of energy stored in his fat would get him through nearly 35 days without any food, assuming he was using 100% fat.

Extremely obese individuals may have upwards of 50-100 lbs of stored fat to the tune of 175,000-350,000 calories of stored energy. That'll keep them alive for several *months* at least without emptying their fat stores. Some very obese individuals have been fasted for up to a year without problems.

In contrast, your other major energy source, which is stored carbohydrate in your muscles and liver, only amounts to about 500 grams at the maximum. Each of those grams of carbs gives your body 4 calories so that's 2000 calories or so as stored glycogen. Not even enough to meet even a single's day's caloric requirements.

So at the low end, fat has a good 7-times as much energy as all of the carbohydrate in your body. At the high end, it can be several hundred times more. Although you don't really want to use body protein for energy, assuming you used it all, it'd still only provide about 10,000 calories, still far fewer than your body fat. And you'd die long before you could use it all up anyhow.

The reason for the difference in energy storage capacity is water. Carbohydrate (stored in your muscles and liver as glycogen) is accompanied by a good bit of water. For every gram of glycogen stored, you store anywhere from 3-4 grams of water with it. In contrast, triglycerides only require about 1 gram of water for every gram of fat stored.

To store even 10,000 calories of energy as carbohydrate (2,500 grams of carbohydrate) would require 7,500-10,000 grams of water. Your cells would explode. Since it doesn't have a lot of water associated with it, fat is a very space efficient way to store energy. Relatively speaking anyhow.

On top of that, a gram of fat provides more useable energy than either a gram of carbohydrate or protein. As above, the commonly given values are 9 calories/gram for fat and only 4 calories/gram for carbohydrates and proteins. So on top of being able to store more grams of fat total, each of those grams provides more useable energy to the body. Body fat is truly an ideal storage form of energy.

From an evolutionary standpoint, the exceptional energy storage capacity of fat cells provided an excellent advantage to humans. Being able to store an effectively unlimited amount of energy in a relatively limited amount of space made it easier to survive through the time periods when food wasn't available. Now it's just another evolutionary leftover that makes it a bitch to get into shape.

Speaking of evolutionary explanations, you might be wondering why men and women show such different fat patterning and I don't mean why as in 'What causes it to occur' (which I'll discuss in a later chapter). Rather, what's the reason for it in terms of 'Why did men and women evolve this way.'

To be honest, I don't recall any good explanation for men's propensity to store fat viscerally and around the abdomen. Perhaps more visceral fat was helpful in mobilizing fuel quickly when men were hunting; maybe they needed more ab fat to protect their organs when they were beating the shit out of each other to try and get a cavewoman's attention. One researcher thinks that male abdominal fat patterning has no actual role, and is simply an effect of gaining fat generally.

Women are actually easier to explain in this context. Women typically store fat in both the hip/thigh area as well as the breasts; this turns out to make a lot of logical sense from an evolutionary standpoint.

Hip and thigh fat, as it turns out, exists to provide energy for breast-feeding after pregnancy. Interestingly, when women are lactating, the normally impossibly stubborn hip/thigh fat becomes the easiest to mobilize. I've wondered for a while if this couldn't be mimicked somehow (e.g. with drugs) but could never come up with any practical way of doing it.

In addition, women's fat patterning probably also evolved for sexual selection reasons. Studies have clearly shown that men have a preference for an ideal waist/hip ratio (which suggests fertility and health) in women. A woman with a narrow waist and curvy hips/thighs is more likely to have this optimal waist/hip ratio than a female who carries more visceral/abdominal fat.

Quite in fact, many of the situations that cause females to accumulate visceral fat (such as polycystic ovary syndrome or PCOS) are associated with decreased fertility. This indicates how strong of a signal the waist/hip ratio (reinforced by fat on the hips/thighs) is from an evolutionary standpoint.

Why women have fatty breasts (something not seen in any other mammal) is an area of much debate. It's been suggested that they are meant to act as a 'buttocks on the front of the body' due to the fact that humans mate face to face (well, sometimes). It's also been suggested that full breasts may act to 'trick' men so that they can't tell when the woman is fertile or not. Entire books have addressed this issue but I'll sum up why women have breasts, at least from a sexual selection standpoint, by pointing out the rather simple fact that 'Men like boobies.'

In any case, I have one final comment before moving on: I can't explain why women put fat on the backs of their arms. I've thought about it for years and simply can't begin to come up with a reason women put fat there. If you know, or think you know, please email me.

But wait, there's more

There's an old (and incorrect) idea that adult humans don't make new fat cells. That is, and I'll discuss this more in a bit, you get born with a certain number of fat cells and you may develop more at puberty or during pregnancy but that's it; your body doesn't make new fat cells. Everything in that sentence is true except the last statement; even non-pregnant adults can make new fat cells.

Usually this happens when the fat cells you have reach a certain size; that is, they are as full as they can physically be. When this occurs, the actual stretching of the fat cell stimulates the release of factors, such as Angiotensin II, prostacylin and others, which 'tell' the body to make new fat cells from something called preadipocytes. Preadipocytes are dormant cells, sort of soldiers who wait for the signals to get called into action. When the right growth factors are released, preadipocytes mature into normal adipocytes. Voila! New fat cells.

Those newly formed fat cells can now be filled with yet more fat and glucose. In fact, a new class of diabetic drugs (called TZD drugs) appear to work by stimulating the production of new fat cells, which gives the blood glucose and fatty acids another place to go. Oh yeah, if the new fat cells get too large, your body will keep making more.

I'd note that, in certain situations, the body doesn't make more fat cells and this causes a ton of health problems because, without anywhere to put the incoming calories, the body starts storing fat in inappropriate places like the liver, pancreas and other tissues. I'd also note that this is only an issue for the extremely obese as a general rule of thumb. There is a weird medical condition (called partial lipodystrophy) where lean adult humans don't have enough fat cells and they can get some of these health problems too. I can say with great certainty that you're not one of these people.

Unfortunately, getting rid of fat cells is nearly (but not completely) impossible. Sure, liposuction is always available but, beyond that, eliminating fat cells is very difficult and only occurs under extremely severe conditions. I'll talk about fat cell apoptosis (death) later on.

If there is a single reason for athletes not to get too fat in the first place, this is probably it: if your fat cells get too big, your body will make new ones. And it's nearly impossible to get rid of the new ones. Obviously, if you're already very fat, there's little you can do: you're pretty much stuck with your fat cells short of liposuction or something along those lines. But if you're a lean athlete looking to gain weight (and realizing that you must gain some body fat to do it effectively), you should keep a lid on that fat gain. You don't want to stimulate your body to make new fat cells.

Dismissing a myth: Fat cells aren't all bad

If you asked most people about body fat, they'd say it was bad, they want to get rid of it, etc. Even researchers tend to promote this view, that body fat is a negative. The main focus tends to be on the negative health consequences of excess body fat (i.e. obesity) and there is certainly much truth to this. Excess adiposity is associated with a number of health consequences, most of them bad. From insulin resistance to the maintenance of a low-level chronic inflammation, getting and staying fat in the long-term is generally not healthy.

But simply saying that 'fat is bad' is a simplistic and incorrect view. The existence of body fat has important roles in human health and survival beyond just energy although that is one of its primary roles. Basically, while too much body fat is definitely a health risk, too little can be just as problematic.

One of the roles of body fat is as a physical cushion for your internal organs. The physical nature of body fat allows it to dissipate force more effectively than muscle. Athletes involved in combative sports (football, etc.) may benefit from carrying a little extra body fat, to protect their internal organs when someone rams into them. Of course, in modern society, most people don't really have to worry about such things.

Fat cells also act as insulation, keeping folks warm when it gets cold. Of course, most modern people can put on more clothes or turn up the heat, something that our ancestors couldn't do.

A less well-recognized aspect of fat cells is their critical role in immune and inflammatory responses. Pre-adipocytes, which I mentioned above, act like macrophages, cells that are critical for a proper immune system response. Of course, this certainly doesn't provide an excuse or reason for carrying excess body fat. But people who diet to extremely lean levels frequently report getting sick more, feeling more inflammation. While this certainly can't be completely attributed to the loss of body fat, that may be part of the puzzle. As I noted above, the opposite also holds true: the obese mount a chronic low-level inflammatory response that causes other health problems.

As the final important role of body fat for this section, I want to talk about the role of body fat stores in glucose tolerance. For optimal health, the body needs to maintain blood glucose between fairly narrow limits. Anyone familiar with diabetes (either Type I or Type II) knows that a big part of the health consequences of those diseases has to do with the chronic high blood glucose (hyperglycemia) that occurs. For reference, normal blood glucose is 80-120 mg/dl or so. Type I diabetics can run blood glucose in the 300-400's or higher, Type II diabetics in the high 180's or worse. Simply put, running chronically high blood glucose causes a lot of damage to the body.

Body fat is one of the places, along with muscle and liver, that your body stores glucose. Studies of rats, or humans who have no fat cells (the lipodystrophy I mentioned above) show chronic high blood glucose, just like diabetics. This is because muscle tissue has a limit to how much glucose it can store as glycogen. Diabetics can't get blood glucose into the cells because they are insulin deficient (Type I) or insulin resistant (Type II); rats and humans without any body fat don't have any fat to store the glucose in. So the mechanism is different but the end result is the same. It's also crucial that the body be able to buffer incoming dietary fat by acutely storing it in fat cells; without this capacity, bad things happen.

Of course, having no fat cells is rare and generally causes death at a very early age. So, if you're reading this book, you don't have it. My point is simply that all of the functions of body fat aren't inherently negative. Fat exists for a reason, even if most of us are carrying more of it than we need or want.

But wait, there's even more

Hopefully you get the idea that your body fat is an amazingly efficient place to store energy, in addition to having other roles mentioned above. Not only does it store incredible amounts of energy as it is, it can increase its energy storage capacity if needed by making new fat cells. It'd be great, and I could end this chapter, if that's all there was to it. As usual, it's not and things are much more complicated.

Since the mid-90's (1994 to be specific), the image of the fat cell as nothing more than a passive player in the body has gone the way of the dodo. On top of its major role in energy storage, your fat cells do so much more in terms of modulating your overall

metabolism. Body fat is turning out to be an endocrine organ in its own right. That simply means that fat cells are releasing hormones and compounds that are acting on other tissues in the body (such as brain, the liver, and skeletal muscle).

On top of its obvious role in disposing of dietary fat, fat cells have been shown to play roles in overall glucose metabolism, blood pressure, appetite, fuel utilization, and hormone production to name just a few. And with each week, it seems as if yet another role for the humble fat cell in modulating human metabolism is found. I'm not going to get into massive detail in this book (beyond what's applicable to the stubborn fat issue) but want to make you aware of some of what's going on in fat cells.

On top of storing and releasing triglycerides, your fat cells also produce hormones themselves. A partial list of the compounds released from your fat cells would include leptin, which is involved in appetite, hormone levels, fat burning and muscle loss, not to mention dozens of other systems; Angiotensin II, which is involved in blood pressure regulation and even controls blood flow to the fat cell itself; Tumor necrosis factor-alpha, which has many varied functions including fat burning, immune functioning, and cell death; IGF-1, which is an anabolic hormone; inflammatory cytokines like Interleukin-6, which is involved in immune function among other things; various prostaglandins, nitric oxide, acylation stimulating protein, resistin, adiponectin and I could probably list a half dozen more if I wanted to.

Fat cells are also one of the major sites of hormone metabolism. Testosterone is converted to estrogen (via the enzyme aromatase) in fat cells in both men and women. In fact, most of the estrogen in males (and in post-menopausal women) comes from the conversion of testosterone in fat cells. Carrying more body fat for males means not only more estrogen, but also less testosterone (yet another reason for athletes to avoid getting too fat). Athletes who use anabolic steroids (or even prohormones) without using an anti-aromatase will get more conversion to estrogen if they are carrying more body fat. The metabolism of other hormones such as DHEA and androstenedione also occurs in fat cells.

Cortisol is also metabolized in fat cells, via an enzyme 11-beta-steroid dehydrogenase (11-beta-HSD) and there is some indication that differences in the activity of this enzyme may be related to how the body handles/produces cortisol, and to obesity.

And that's just a quick look at some of the things that your fat cells are doing in your body. With each week, researchers seem to turn up more. As a quick tangent, the discovery that fat cells were more than a passive place to store energy revolutionized the study of obesity. So instead of dozens of studies indicating why fiber is important for weight loss, there are now hundreds/thousands of studies dealing with all of the myriad hormones that are released from fat cells, and how they affect metabolism.

Although many of these factors can't be controlled at this point, their discovery has opened up an entirely new area of possibilities for manipulating body composition and body fat levels. At the very least, we know more about why our bodies are reacting a certain way. Knowing the why gives us at least some possibility of being able to do something about it.

The main point for you to get from this chapter is that fat cells are far more than just a passive storage site for excess energy. Yes, they are amazingly well adapted to their role in energy storage but they do far more. Basically, this is just background for the next chapter. So turn the page already.

Types of Body Fat

Now that you know more about fat cells (in general) than you ever wanted to know, I want to move to the next topic of discussion which is the different 'types' of body fat.

In the last chapter, I sort of made it sound like all fat cells are basically the same; nothing could be further from the truth. One of the more interesting areas of fat cell research has to do with the heterogeneity of fat cells and fat cell metabolism. Heterogeneity is just a nerd way of saying that the fat cells aren't all the same.

Fat cells differ in how they handle dietary fat, how they respond to insulin and other hormones, which of the myriad hormones they release and in what amounts, what their blood flow is like and, as it turns out how easy or stubborn they are to get rid of.

The human body has at least 4 different depots of stored fat, although I'll actually distinguish a fifth that isn't really used in the research. Let's look at each since this provides the major background for the rest of this book.

The first type of fat: Essential body fat

Essential body fat refers to the fat found around your organs (where it serves an important cushioning role), in your nervous system (nerves are surrounded by sheaths of fat), and especially in your brain. On which note: low fat diets can impair neural development in developing babies because of the high fat content found there. Without enough dietary fat (specifically DHA, one of the fish oils), baby brains don't develop well. As well, deficiencies of certain type of fatty acids in the brain seem to be related to both depression and psychosis. Whether this is a function of diet, or of bad development as a child is unknown.

For the most part, we don't really need to worry that much about essential fat. As the name implies, essential body fat is exactly that: essential. You can't lose it and, even if you could, you wouldn't want to because you'd be dead.

In men, essential fat typically makes up about 3% of total weight or so. So a male with 10% body fat has about 1/3rd of that as essential fat that can't be removed. This means that anytime you hear someone claiming a body fat percentage below 3%, you should be fairly suspicious. Either they're lying or, more likely, the methodology they used to measure body fat was inaccurate (some methods of estimation will give athletes a negative body fat percentage, for example). Physiologically, a body fat percentage of 1% is impossible, as it would require getting rid of the essential fat.

In females, essential fat is higher, and usually estimated at 9-12% or so. That difference in essential fat (3% vs. 9-12%) is a big part of the difference in total body fat levels in men and women. That is, a man at 10% body fat is roughly equivalent to a female at 16-19% body fat; they are both 7% above their essential body fat levels. Again, you'll hear of women (mainly bodybuilders) reporting values below 9%, which has more to do with the problems in the estimation equations than anything else.

I don't have much else to say about essential body fat and won't mention it again in this book. Even if you're obsessive about getting rid of *all* of your body fat, you don't want to lose essential fat and probably couldn't do it in the first place. It's there for a reason, enough said.

The second type of fat: Brown adipose tissue (BAT)

I mentioned BAT in the last chapter and want to discuss it briefly again here. In comparison to normal white adipose tissue (WAT), BAT is very different. Whereas a primary role of WAT is energy storage (and of course, the other roles I described in the last chapter) the main role of BAT is energy utilization, primarily to provide heat.

As mentioned last chapter, WAT is primarily made up of stored triglyceride with a small amount of enzymes and other stuff. What I didn't mention last chapter is that WAT also has very few mitochondria (the powerhouse of the cell). Mitochondria make energy by burning free fatty acids.

In contrast, BAT has very little lipid but quite a lot of mitochondria, which is why it's so good at energy (and heat) production. On top of that, BAT appears geared to burn fat almost exclusively. And when it does so, the energy produced is lost as heat; it's not used to fuel any other chemical reactions.

The discovery of BAT, and its functions, opened up an entirely new area of obesity research, as researchers thought that BAT activation might help to burn off excess calories and limit or help to cure obesity. It was also thought that defects in BAT activity might be a contributing factor to obesity.

And while drug companies became briefly interested in drugs to activate BAT, they pretty much failed completely (for reasons I'm not going to get into here). To my knowledge, drug companies have essentially given up on them. Of course, supplement companies continue to sell compounds aimed at activating BAT; but having products that actually work has never been the goal of that industry.

For the most part, worrying about BAT is a losing proposition since humans don't have very much (what little we do have is found between the shoulder blades and in a few other places). New research suggests that (some) humans might have relatively more BAT than originally thought but it's unclear how relevant this is to fat loss or obesity. Babies have lots of BAT but most of it is lost when folks reach adulthood. A certain type of tumor increase BAT levels and, oddly enough, lumberjacks (who spend their lives in the cold) have more BAT than usual. But, practically speaking BAT is basically a dead-end at this point.

Of more relevance to this book, worrying about BAT simply isn't important in terms of getting rid of stubborn body fat and I'm discussing it mainly for completeness. With new drugs or what have you, finding ways to turn WAT into BAT may become available. For now it's a dead end.

The third type of fat: Visceral fat

Of all the types of fat in the body, visceral fat is probably the one that has gotten the most interest, especially in terms of its effects on health. But let's back up a step and look at what visceral fat actually *is* first. Visceral fat is found around your internal organs and can be thought of as gut fat. But it's not the fat that you can see on top of your stomach; it's actually underneath your abdominal muscles surrounding your organs.

People who carry a lot of visceral fat are referred to as having central obesity and their tummy pooches out quite a bit from their body. You can go to any mall food court if you want to see examples of this. While this typically happens in males, it can also happen in females under certain conditions, mainly in extreme obesity and after menopause. Women who suffer from PCOS also have problems with central obesity due to higher testosterone levels. I'll talk about hormones and how they impact body fat (and stubborn fat specifically) in a later chapter.

Although carrying excess fat generally increases health risks, a great deal of research suggests that visceral fat is even worse in this regard. At the very least, carrying around a lot of visceral fat is associated with insulin resistance (meaning that insulin can't do its job well) although it's questionable whether high visceral fat causes insulin resistance or insulin resistance causes visceral fat accumulation. Early theories argued the former, current research suggests that the latter is true.

Visceral fat is different than subcutaneous fat (discussed next) in many ways, which is why I'm discussing it separately. The biggest difference is that visceral fat is more metabolically active than subcutaneous fat. Meaning that it responds more effectively to fat mobilizing/burning stimuli than other types of body fat. It's also less affected by insulin, which tends to shut down fat burning; when insulin goes up, visceral fat doesn't stop releasing fatty acids like other fat cells. This is a problem because high blood fatty acids and high insulin levels tends to be a bad thing from a health perspective.

Visceral fat also has better blood flow compared to other body fat, meaning it's easier to get the fat out of the fat cell. All of this adds up to a type of body fat that is mobilized more easily than the others.

Visceral fat also appears to undergo something called apoptosis (which just means cell death) more readily than other types of fat. There is also some research showing that visceral fat responds differently to diet and exercise than other types of fat. It appears that aerobic exercise gets rid of visceral fat more effectively than just reducing calories, probably because of the hormonal response involved: aerobics raise catecholamine levels while diets often reduce them, and visceral fat is *very* responsive to catecholamines.

For the most part, by the time you need the information in this book, visceral fat should be a non-issue. Some males at 15% body fat can still have some but anybody much leaner than that, unless they're using massive amounts of androgens, should have gotten rid of it already.

The fourth type of fat: Subcutaneous fat

Subcutaneous (sub-q for short) just means 'under the skin' ('sub'=under, 'cutaneous' = skin) which is where this fat is found. This is the type of fat that most folks are focused on getting rid of (health professionals worry more about the impact of visceral fat). For the rest of this book, I'll be focusing exclusively on subcutaneous body fat since that is all that is relevant to the topic of stubborn body fat.

Of your total body fat (including all of the above 'types' of fat), sub-q fat is the most prominent. Anywhere from 40% to 60% of your total body fat is found under the skin, which is what allows you to estimate body fat percentage with methods such as skinfolds.

As you're probably well aware, people differ quite a bit in how their fat is distributed, I mentioned this briefly in Chapter 1 but want to get into more detail here. Typically, on top of their greater amounts of visceral fat, males carry more of their fat on their midsection and upper back, with much less on their hips and glutes. It's not uncommon to find men who have an extreme amount of body fat on their abdominal area while still having very lean legs. This is sometimes called an android body fat deposition pattern. It's also described as having an apple shape. This turns out to be associated with many of the health risks I briefly talked about above, mainly due to the increased visceral fat.

Women, in contrast, typically carry more fat on their hips and thighs with less on their abs. Breast size can vary quite a bit but is another place where women store body fat. It's not uncommon for women to be extremely lean in their upper bodies (frequently having visible abdominals) while having fairly fat hips and thighs. This is frequently called a gynoid body fat pattern. It's also described as having a pear shape. Women are protected against many diseases that men get (such as heart attacks) because they store less fat viscerally and more fat subcutaneously.

There are, of course exceptions. Little boys and girls display similar body fat patterns before puberty suggesting that the causes of male and female fat patterning is due to the changes in hormones that occur during puberty. I'll discuss this in a later chapter.

Women who go through menopause without going on hormone replacement therapy can switch to a more male-like body fat pattern (including gaining visceral fat). As well, many

men will display more gynoid body fat patterning and women can display more android body fat patterning (e.g. carrying more of their fat centrally around their stomach). When I talk about hormones later, you'll see why a lot of this occurs.

Now, it turns out that even sub-q fat isn't all the same. Hip and thigh fat is metabolically different than abdominal fat, and there may be differences between different 'parts' of abdominal fat. Recent research has identified three different areas of abdominal fat. Deep abdominal fat is a lot like visceral fat and is relatively easy to get rid of. Superficial abdominal fat can be further subdivided into upper and lower pieces with the upper fat being easier to mobilize than the lower bit. Now you know why you get your upper 4 pack before the lower two abs ever come in.

Of course, hip and thigh fat is the hardest of all to get rid of for reasons I'll discuss next and in subsequent chapters. Women have the worst problems, as do men with female fat patterns.

A little bit tangentially, I should at least mention breast fat and what (can) happen during the course of dieting to extremely low body fat levels. As expected, breast fat is yet another area that the body can draw energy from. Many female dieters find that their breasts basically collapse during the course of an extended diet, especially when they reach very low levels of body fat.

Worse yet, when they regain body fat, the breasts often don't go back to normal (although the hips and thighs certainly do). Some research has suggested that breast cells can undergo apoptosis (cell death), similar to visceral fat, and this probably explains the collapse followed by the lack of regain in the breast area.

However, this isn't universal by any stretch, some women's breasts don't change at all. Empirically, it seems that women who have had a child are more likely to have their breasts collapse. There is probably also a hormonal effect. Females with more male like body fat patterning or those who carry their fat more evenly, seem less likely to have their breasts shrink during a diet.

The fifth type of fat: Stubborn fat

Now, although most researchers would be apoplectic (what a great word) at what I'm going to say next, I'm going to differentiate subcutaneous body fat into two different types: regular fat and stubborn fat. You can probably guess what those mean. Regular fat is the stuff that comes off fairly easily. Just adjust diet and exercise a bit and it comes off without too much trouble. Just about any non-retarded diet and exercise program will work fine for that stuff.

Stubborn fat is the other kind, the fat that just doesn't seem to want to come off without a nearly superhuman effort. Even then it doesn't always come off. There are a number of physiological reasons why this is the case. You'll learn about them very shortly. The severe muscle loss that frequently occurs in extreme dieters usually occurs when they are trying to chase down the last of the stubborn fat.

As I mentioned in Chapter 1, when your body can't get fat to burn, but needs energy because you're cutting calories, it starts going after muscle at a quicker rate. This is one of several reasons that muscle loss accelerates when people get super lean; for more details see my **Ultimate Diet 2.0** which talks about the other reasons behind this.

Of course, most of this book is aimed at stubborn fat. In men, who generally don't store much fat in their lower bodies, ab and low back fat is usually the most stubborn stuff, although not for the reason you probably think. Men's lower body fat is actually just as stubborn as women's (when researchers biopsy it, men and women's lower body fat is physiologically identical). Since men don't generally store fat there, however, it's a non-issue. So ab/low back fat becomes stubborn by default. I'd note that, for the most part, losing ab/low back fat is far less an issue of major diet and training manipulations and more an issue of patience (which most men lack). No matter how you cut it, men's ab fat is simply not as hard to get rid of as lower body fat. EVER.

Women, of course, tend to have the worst problems with getting rid of the last of their body fat, as I've mentioned already. The upper body will often be completely shredded while the legs remain smooth. You'll see why as you continue reading.

This isn't to say that the methods in this book can't help male dieters. I'm simply trying to point out that they often aren't necessary. Many men can and do get plenty lean (at least to the level of 6-pack abdominals) with nothing but the standard diet and exercise advice. But this frequently fails for females.

When I discuss some gender differences, you'll see why this is actually a huge problem; the same strategies that work fine for men's 'stubborn' ab fat often doesn't cut it for women's lower body fat. A lot of male coaches forget this and assume that what works for them will work for their female clients. But it simply doesn't get the job done.

Summing up

So that's an overview of different types of body fat with a bit of detail on how they do or don't respond to various stimuli; you'll get many more details in upcoming chapters.

In general, fat will come off the body from least to most stubborn and there is a clear hierarchy in how fat comes off the human body.

With few exceptions, usually involving heavy androgen use, visceral fat will come off fairly quickly and easily. Women don't generally carry much visceral fat so this tends to be a non-issue for them. Men usually do carry some visceral fat and the fact that it comes off so quickly is one reason that men always lose fat faster on a diet/exercise program than women: the visceral fat is so easy to get rid of and there's usually plenty of it. I'd note that losing visceral fat often makes people 'feel' leaner even though they don't look it. This is hard to describe if you haven't experienced it. You feel leaner, you can suck your stomach in and it may be flatter. But visually your abs are just as jiggly as ever. That's due to visceral fat loss.

After visceral fat comes off, the next place to lean out is the non-stubborn subcutaneous fat. Delts, upper back, arms, etc. these areas always lean out far before the others. Few have problems with these areas specifically, which is why I didn't mention them.

When folks start getting leaner, the body starts dealing with abdominal fat. The deep abdominal fat, similar to visceral fat, comes off first before the body attacks the superficial abdominal fat. Even there, the upper portion comes off before the lower portion. For most men, this would occur before the love handles or low back lean out but there is a great deal of inconsistency here.

With few exceptions, the last to come off (if it comes off at all) is hip and thigh fat. As I noted above, since men don't typically carry a lot of hip and thigh fat, ab fat (superficial ab fat) is their stubborn fat.

For women, unless they have atypical fat patterns, hip and thigh fat is always the last to come off. For men with female fat patterns, the same comments apply: if it comes off at all, it'll be after every other part of the body looks like a living anatomy chart.

You may be wondering what makes stubborn fat so stubborn and it's really the key question of this book; figure out why stubborn fat is stubborn and we can start addressing how to get rid of it.

But, before I can address that issue, I need to give a little more background information on fat cells and how fat is stored and burned. Don't worry; reading through my endless exposition is always worth it in the long run.

Basic Fat Cell Metabolism

Now that you know what body fat is and a little bit more about the different 'types' of fat, I want to briefly discuss fat cell metabolism and what can occur in those fat cells.

Strictly speaking, there are four distinct metabolic events that can occur in fat cells. They are: fat cell hyperplasia, apoptosis, lipogenesis and lipolysis. Don't freak out at the nerd-speak, I'll explain the processes one by one.

Hyperplasia: Fat cell hyperplasia refers to an increase in fat cell number (normally, fat cells increase by growing in size via hypertrophy); this is also called adipogenesis. I've mentioned fat cells hyperplasia in other chapters and pointed out that this is generally only relevant for people who are extremely fat and continuing to gain fat. This certainly isn't relevant to readers of this book and I won't discuss it further.

Apoptosis: Apoptosis is a term referring to the death and removal of cells. With a few exceptions (notably visceral fat along with breast fat under certain conditions), fat cells don't generally undergo apoptosis except under very extreme conditions. Since it generally won't occur during the course of a normal diet, I won't discuss apoptosis further.

For the majority of our purposes, only lipogenesis (fat storage) and lipolysis (fat mobilization) are important and this chapter will focus on both. Towards that goal, let me further explain lipogenesis and lipolysis.

Lipogenesis: Lipogenesis ('lipo' = fat, 'genesis' = making) simply means the formation of new fat in fat cells from glycerol and three free fatty acids. Theoretically, the body can convert carbohydrates to triglyceride (a process called de novo lipogenesis) but most studies indicate that this contributes only minimally to total fat storage under most normal dietary conditions. Usually it only occurs with long-term massive carbohydrate overfeeding or high calorie glucose infusion or things like that; there is also some evidence that it can occur to a relatively greater degree when individuals are hyperinsulinemic but, again, this generally only occurs under conditions of obesity and isn't relevant to this book.

Lipolysis: Lipolysis ('lipo' = fat, 'lysis' = breakdown) refers to the breakdown of the fat in fat cells, producing glycerol and free fatty acids. Lipolysis is the single most important issue to the topic of this book and I'll discuss it in much greater detail in the next chapter.

I should note that fat storage and breakdown are typically going on at the same time, in what's termed a futile cycle. That is, the body is both storing fat and breaking it down simultaneously; the combination of fat breakdown and resynthesis is referred to generally as turnover (the same process occurs for protein as I detailed in my **Protein Book**). While this seems wasteful, it allows the body to tailor its biological response to rapidly changing needs.

As it turns out, there are site-specific differences in the rate of turnover. As I talked about a chapter or two ago, visceral fat tends to be more metabolically active; it has a higher rate of turnover than sub-q fat. There are also differences between different subcutaneous depots that are gender specific. I touched on this last chapter and will discuss it in more detail later.

In any case, what ultimately happens to fat mass depends on the balance between the two processes. If the body is storing more fat than it's releasing, you will be gaining body fat in the long-term. If the body is releasing more fat than it's storing, you will be losing fat over time.

Of some interest, and I'll come back to this in a later chapter, recent research suggests that the body can release fat from one fat cell and have it be stored in a different fat cell. Even more strangely, that same study found that women's bodies may actually release small amounts of fat from their upper bodies to be subsequently stored in their lower bodies. This may explain a previous claim (that I had always dismissed) whereby women seem to get leaner in the upper body while their lower bodies actually get fatter. I'll come back to this later.

What is fat redux

In a previous chapter, I explained that body fat is actually made up of stored triglyceride (TG). I want to mention that the majority of dietary fat that you eat is also in the form of TG's. Chemically, TG's are made up of a glycerol (a sugar) backbone attached to three fatty acid chains.

The chemical structure of the fatty acid chain is actually what people are referring to when they talk about saturated, unsaturated, or polyunsaturated fats. More accurately, they are referring to the structure of the specific fatty acid chain (meaning that any given TG can actually contain some mixture of saturated, unsaturated and polyunsaturated fatty acid chains). I don't want to get into the details beyond that, just keep in mind that a TG is a molecule of glycerol bound to three fatty acid chains. There are also di-glycerides(DGs, I'll tell you about one in a second) and monoglycerides (MG) which are a molecule of glycerol bound to two and one fatty acid chain(s) respectively.

So you already know that your fat cells are mostly stored TG. The other source of TG is dietary fat and I want to talk briefly about what happens when you eat fat.

Dietary fat metabolism in a nutshell

After consumption, dietary fat is broken down, repackaged into something called a chylomicron, absorbed into the lymphatic system, and appears in the bloodstream about 3 hours after you eat. While a certain percentage of ingested dietary fat will be used for energy or go to the liver or skeletal muscle for either storage or burning, some proportion will always make it to the fat cells where it can potentially be stored. This is unavoidable.

Of some interest, research has shown clear gender differences in how ingested dietary fat is handled. In men, a greater proportion of ingested fat tends to either be used for energy immediately, absorbed by visceral fat, or simply sits in the bloodstream. The benefit of this is that men can more easily burn off ingested dietary fat since less tends to get stored in the sub-q fat. The drawback is that having lots of fatty acids sitting in the bloodstream is one reason that men are more prone to heart disease and heart attacks.

In contrast, women tend to store more ingested dietary fat in subcutaneous fat; while this protects women from heart attacks it also means more ingested fat gets stored that eventually has to be mobilized and burned off. It actually gets worse than that; research has found that, when women eat, their bodies preferentially increase blood flow to the lower body, storing calories preferentially in the hips and thighs. Remember how your mom said that eating cake went straight to the thighs. She was right.

I would comment that this preferential fat storage appears to be more pronounced when women are over-eating, whether it occurs significantly with calories at maintenance or while dieting is debatable. The benefit of this extra fat storage in sub-q depots is that women are protected from heart attacks (at least before menopause). The negative is that now you have to get it out of the fat cells to get lean.

I'd note that a couple of odd fats, notably medium chain triglycerides (MCT's) and diglycerides (DGs, two fatty acid chains attached to glycerol, available commercially as Enova oil) are handled differently by the body compared to normal dietary fat.

Rather than going into the lymphatic system for storage, MCT's and DG's go through the liver and there is evidence that the body uses them for energy preferentially over storing them in fat cells. MCT's have a slight thermic effect (15 grams per day can raise metabolic rate by about 5% and consuming DG on a diet has been shown to increase fat loss slightly (the impact is not massive, possibly a few tenths of a pound of fat per week). They may have utility when folks are trying to get rid of stubborn body fat.

Fat storage

While fat storage and mobilization is unbelievably complex, I'm going to skip a lot of the tiny details and focus on the bigger picture here. The tiny sub-pathways that are out of our control and only of importance to researchers nerdier than me simply aren't relevant or interesting.

In a general sense, fat storage is a process whereby the fat cells takes three fatty acids and a molecule of glycerol and puts them together, storing them as TG in a lipid droplet. Since it's less important to this discussion, let me get glycerol out of the way first.

Now, when stored TG is mobilized, it produces glycerol and three fatty acids which move into the bloodstream. For complex reasons, the fat cell can not reuse the glycerol, although it will happily re-absorb the fatty acids. Glycerol is used elsewhere in the body and can be converted back to glucose in the liver. Fat cells can only absorb glucose from the bloodstream, which is then converted to glycerol within the fat cell before being attached to three fatty acid chains.

So where does the glucose come from? Well, on a carb-based diet, it comes from ingested dietary carbohydrates. Under low-carbohydrate conditions, the body will convert other fuels to glucose. I already mentioned glycerol which is converted to glucose in the liver. Pyruvate, lactate and some amino acids (leucine, alanine) are also converted to glucose. These all provide glucose for the fat cell to absorb, convert to glycerol and bind to fatty acids. That's really all I have to say about glycerol.

Now, the fatty acids being stored will generally be coming from the diet, from the breakdown of ingested TG. As I mentioned above, recent research has also identified a new pathway whereby fatty acids released from one fat cell can be restored in another fat cell. My point being that even on a zero-fat diet, there are always plenty of fatty acids floating around in the bloodstream for storage.

So how do the fatty acids (either from dietary TG or from other fat cells) get stored in the fat cell? As with all processes in the body, this process is driven by enzymes. Although there are lots of other pathways that play a role, the two enzymes of major importance for this discussion are lipoprotein lipase (LPL) and acylation stimulating protein (ASP). Pretty much everyone in the field focuses on the first, few seem to even be aware of the second. In doing this, they miss much of the big picture.

Lipoprotein lipase

Lipoprotein lipase, or LPL, is one of the primary enzymes involved in fat storage. For many years, in fact, it was thought to be the most important enzyme or possibly the only one that mattered. Of course, as these things go, it turns out to be not quite that simple. Another protein called acylation stimulating protein (ASP), discussed next, turns out to be as, if not more, important than LPL.

LPL is produced within the fat cell, moves through the cell membrane, and attaches itself to the outside of the fat cell. The only molecule that LPL seems to bind to are chylomicrons which, as you'll recall, are a way that the body packages triglycerides.

When LPL interacts with a chylomicron, it breaks down the triglycerides in the chylomicron, releasing free fatty acids (FFA) into the microcirculation around the fat cell. Some of those FFA move into the fat cell while others are carried away via the bloodstream so they can be used for fuel elsewhere. The FFA which move into the fat cell are combined with glycerol to produce triglycerides, as I discussed above.

One of the primary regulators of LPL is insulin which affects both the amount and activity of LPL. Insulin is released when you eat, primarily in response to carbohydrate and protein ingestion; in contrast, consuming dietary fat by itself does not raise insulin (as you'll see, it still affects fat cell metabolism). The effect of insulin on LPL activity is a lot of where the idea that only insulin stores fat comes from (this is the source of a lot of dietary silliness as well). Unfortunately, that's simplistic and incorrect, as you'll soon see.

LPL is also found on the cell membrane of muscle tissue and cardiac tissue, where it plays a slightly different role. In both, LPL produces free fatty acids to be burned for energy within the muscle or heart cell. The point being that you shouldn't think of LPL as a 'bad' enzyme per se. Whether LPL has good or bad effects, in terms of fat storage, depends on where it's doing its job.

Overactivity of LPL on the fat cell isn't a good thing by any means as it means that fat cells are being exposed to higher levels of FFA for potential storage. In contrast, when LPL activity in muscle or heart cells increases, those tissues can uptake more fatty acids for either burning or storage.

In addition to the effect of insulin, LPL activity is also increased by the presence of chylomicrons in the bloodstream which occurs after a meal. Its activity is decreased when chylomicron levels decrease (such as during the period between meals). However, there are other factors that affect the overall amount of LPL in the cell. These include both hormones and habitual diet; there is assuredly a genetic factor.

However, of some interest, research has found that animals bred without LPL can still store fat just fine. That single data point, ignored or unknown by most in the industry is a huge indicator that there is more to fat storage than just LPL. Since people will incorrectly go from *'LPL stores fat'* and *'Insulin activates LPL'* to *'Insulin stores fat'*, it's important to talk about the other factors involved in fat storage. The most important in this case is acylation-stimulating protein or ASP.

Acylation-stimulating protein (ASP)

For many years, it was thought that LPL was the key enzyme involved in fat storage. In fact, if you look in most current physiology books, it is still the only enzyme that gets much discussion. As tends to happen, this is utterly simplistic and completely incorrect. This brings us to the discussion of acylation stimulating protein or ASP for short.

Discovered back in the 80's (and yet ignored by most), ASP appears to be even more critical for fat storage than LPL. One researcher has stated that ASP is the single most potent stimulator of triglyceride synthesis in the fat cell. This is important.

Of importance, it's been shown that rats who are bred without LPL but who have ASP can still store fat in their fat cells. But this doesn't work the other way around; rats without ASP but which have LPL can't store fat.

That tells us that ASP is the critical substance, not LPL, at least in rats (the data on ASP in humans is less well developed). And while strategies to block ASP are being researched,

not all is good; if the body can't store nutrients in fat cells, they can accumulate in the bloodstream causing health problems. My point being that LPL doesn't appear to be required for fat storage, but ASP most definitely is. So what is ASP and what does it do?

ASP is also made within the fat cell out of three different proteins, the names of which aren't important here. When ASP is activated, it promotes the synthesis of TG's from FFA and glycerol. LPL is only important to break FFA out of the chylomicron, ASP is the key factor for actual TG synthesis.

So what activates ASP? Well, insulin can activate it for sure. However, the mere presence of chlyomicrons in the bloodstream also activates ASP, even without any increase in insulin. Studies have clearly shown that eating or infusing fat, even in the complete absence of an increase in insulin will affect fat storage (increasing it) and breakdown (inhibiting it). Insulin isn't required for any of this to occur.

Even there, ASP itself does other things such as stimulating the pancreas to raise insulin levels. Any time you eat dietary fat, there is potential for it to be stored, insulin or no insulin. Avoiding eating carbohydrates with fat or whatever other silliness that is so often proposed won't change this. You could mainline dietary fat by itself and still store fat. The body isn't stupid and wouldn't let you magically avoid fat gain if you only ate dietary fat without ever raising insulin. Eat fat and the chylomicron will activate ASP; along with storing fat, ASP will increase insulin levels itself.

Of course, if you're on a diet and in a caloric deficit, this doesn't matter massively as the overall effect (fat storage minus release) should still have a net result in decreasing body fat mass. That is, even if you store some fat after a meal (insulin or not), as long as you're losing more over the course of a day (because you're in a caloric deficit), you still get leaner.

With one weird exception that has only recently been brought to light. Female dieters have occasionally claimed to be gaining fat in their legs while leaning out in their upper bodies. I used to dismiss this as some weird perception thing but there may be truth to it. As I noted above, research has identified a new pathway by which fatty acids mobilized from one place (e.g. the upper body) can be stored in others (e.g. the legs). This could potentially occur even in a caloric deficit, fatty acids are getting pulled out of the upper body and restored as fat in the lower body.

In any case, having looked at the processes of fat storage, I want to move onto a more interesting topic which is how fat is burned off the body. That's the topic of the next chapter.

5

Burning Body Fat

Having looked at fat storage in the previous chapter, I now want to move on to how fat is 'burned' or 'lost', both in general and specifically in terms of the issue of stubborn body fat. First I want to define what 'burning' body fat even means, before looking at the three key steps involved in getting rid of body fat.

What exactly does it mean to burn body fat?

People usually talk about 'burning' body fat without ever really being clear on what it means. In physiological terms, 'burning' is better described as oxidizing. This simply means that the body reacts some fuel with oxygen, ultimately producing adenosine triphosphate (ATP). ATP is the only fuel that cells can use directly and you can consider it to be the basic energy currency of all cells in the body; other fuels are only valuable in that they can be oxidized to produce ATP. Any time a cell burns fuel for energy, it produces ATP, how much is actually produced depends on which fuel you're talking about (I don't want to get into details beyond that).

What's relevant here is what's happening to the triglycerides (TGs) stored in your fat cells, which is where the fatty acids which are 'burned' are coming from. Most tissues in the body can use fatty acids for fuel but the main ones we are interested in are skeletal muscle and the liver. A few tissues, such as the brain, can't use fatty acids directly, however they often can use ketones which are made from fatty acids (in the liver).

So let's look at the mechanisms underlying the process of fat burning. Fundamentally, there are three primary steps: breakdown, transport, and oxidation (burning). There are some other steps that are often discussed in the literature, however they are not practically relevant. The three below are the key ones of interest.

Step 1: Breakdown

The first step in burning off body fat 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 the fat out of the fat cell, you can't burn it off (various forms of liposuction being the only way around this).

Recall that body fat is primarily stored triglyceride with a small amount of water and some enzymatic and cellular machinery. Mobilizing body fat requires that we first break down the stored triglyceride into three fatty acids and a molecule of glycerol. The single step that limits how quickly or slowly fat is mobilized (scientists call this the 'rate-limiting' step) is the activity of an enzyme called hormone sensitive lipase or HSL.

What regulates HSL? A number of hormones such as testosterone, cortisol, estrogen, and growth hormone have modulating effects on HSL (mainly increasing or decreasing the total **levels** of HSL in the fat cell). However, for this chapter's purposes, the only hormones that we need to be concerned with in terms of affecting HSL **activity** are the catecholamines and insulin.

The catecholamines are adrenaline and noradrenaline, which are also called epinephrine and norepinephrine depending on where you live. In the US, people use adrenaline/noradrenaline; in the rest of the free world, they call the hormones epinephrine/norepinephrine. Do I really need to tell readers of this book what insulin is?

I really want to drive this point home that, in humans, only insulin and the catecholamines are relevant in terms of acute fat mobilization. The other hormones have either a modulating effect, at best, or no effect at all.

The primary **inactivator** of HSL is the hormone insulin, which blunts HSL activity at even low concentrations. In fact, even fasting insulin levels are sufficient to inactivate HSL by nearly 50%. Even small increases in insulin (from either protein or carbohydrate intake) inactivate HSL almost completely. As it turns out, the mere presence of triglycerides in the bloodstream (via infusion or by just eating fat by itself) also inhibits HSL activity so you can't just blame insulin. Any time you eat, HSL will be inhibited.

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 various tissues. 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. As you'll see in a subsequent chapter, this is important because how someone exercises can drastically affect how adrenaline and noradrenaline are released.

The key regulator of HSL is a compound called cyclical adenosine monophospahte (cAMP). When cAMP levels are low, HSL activity is low and fat mobilization will also be low. When cAMP levels are high, HSL activity is high and fat breakdown increases. For optimal fat mobilization, therefore, we want high levels of cellular cAMP.

I'd note that cAMP is all over the body and this has been a source of problems with previous approaches to stubborn fat loss. Anything consumed orally (e.g. the supplement forskolin) that affects cAMP is going to affect cAMP levels all over the body. This can have a variety of effects, some good, and some bad. Ideally we want to try and isolate cAMP changes to fat cells although, depending on what you're talking about (diet, activity, etc), this is usually impossible. At best you try to ensure that raising cAMP in fat cells doesn't do something nasty elsewhere in the body, like jacking heart rate and blood pressure sky high.

Insulin lowers levels of cAMP, inhibiting fat mobilization. The catecholamines are little bit more complex. Depending on the type of fat cell, and the levels of catecholamines, the end result can be increased or decreased levels of cAMP. To understand that, I need to explain adrenoceptors.

All about adrenoceptors

To understand some of the cryptic remarks above, I need to back up a bit and explain how the catecholamines send their signals. So you know, all hormones work through specific receptors and the catecholamines are no different, they have their own specific receptors called adrenoreceptors or simply adrenoceptors. I'll use both interchangeably throughout the book.

Now, 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. Case in point, the penis has adrenoceptors, so does female genitalia. One of the compounds I'll talk about later (yohimbe or yohimbine), by affecting the adrenoceptors in the genitals increases blood flow and sexual stimulation. It's always fun when you get a boner in the gym after taking oral yohimbe, but I digress.

There are at least 3 (and maybe 4) different beta-receptors called, imaginatively: beta-1, beta-2, beta-3, and beta-4 (aka the atypical beta-3 adrenoceptor). Alpha-adrenoreceptors come in at least two flavors, alpha-1 and alpha-2. Each has a whole bunch of subtypes although this isn't that important for this book. In animals, beta-3 receptors are found mostly in BAT (discussed previously); I already told you that most humans don't have much BAT. Alpha-1 receptors do play a role in fat cell metabolism but it has more to do with glucose than fat metabolism per se, so I won't address them here.

The main receptors we need to worry about in human fat cells are alpha-2 receptors and beta-2 receptors (beta-1 may play a role in fat mobilization as well), both of which actively bind the catecholamine hormones. When catecholamines bind to beta-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. Which signal is sent depends on several factors including the levels of each hormone, as well as the relative proportion of adrenoceptors in a given tissue.

As it turns out, different areas (or depots) of fat have different levels of alpha and beta-adrenoceptors and this controls, to a massive degree, whether any given exercise stimulus has a net lipolytic or anti-lipolytic effect. I'll come back to this in two chapters when I finally answer the question of "What makes stubborn fat stubborn." Various factors control adrenoceptor number, I'll detail some of them later in this book.

For now, all you need to remember is that adrenoceptors ultimately determine whether the catecholamines end up stimulating fat breakdown or not.

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, high insulin levels in the face of high catecholamine levels 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 may decrease fat mobilization despite increased levels of catecholamines.

Tangentially, whether or not ingested carbohydrate impairs lipolysis or fat oxidation depends on the intensity of the exercise. At low intensities, ingested carbohydrates clearly impair fat oxidation, especially in untrained individuals. At higher intensities, in trained individuals, carbohydrate ingestion does not negatively affect fat utilization during aerobic exercise.

Ultimately, you just need to remember the following: insulin inhibits fat mobilization and the catecholamines can either stimulate or inhibit lipolysis depending on a few variables that I'll address in detail in a later chapter. With few exceptions (the main one being severe insulin resistance of the fat cell, meaning that insulin can't exert its normal effects), insulin wins the battle.

Step 2: Blood flow and transport

Unfortunately, just mobilizing fatty acids out of the fat cell isn't all that is required for fat loss to occur. The next, and critically important step, is to get the fatty acids away from the fat cell, to other tissues where they can actually be burned. In fact, if the fatty acids aren't moved out of the adipose tissue in this fashion, the body will happily re-store them (a process called re-esterification). So after mobilization, the next key step is transport out of the fat cell to other tissues. This process is dependent on blood flow through the adipose tissue.

Adipose tissue blood flow (ATBF) is tough to measure and there's a lot less research than I'd like on the topic; but it's become clear that ABTF is not only differentially regulated but can be potentially manipulated. For example, I already mentioned that following a meal, females appear to preferentially increase blood flow to the lower body, increasing nutrient storage there. This appears to be especially the case when excess calories are being consumed.

Other factors such as hormones (discussed next chapter) can also affect adipose tissue blood flow. Aerobic exercise tends to increase adipose tissue blood flow with the effect becoming greater as the duration increases. Temperature appears to play a role too, the body closes off blood flow when it's cold and increasing temperature increases blood flow; perhaps the tummy and thigh wraps worn during exercise do more than just cause local water loss. Interestingly, blood flow to fat cells increases with long-term fasting, probably to help the body mobilize fatty acids for fuel.

Adipose tissue blood flow is also profoundly controlled by adrenoreceptor levels with the same basic scheme I discussed above about fat cell metabolism holding true. Beta-adrenoceptor activity increases adipose tissue blood flow; alpha-adrenoreceptor activation inhibits it. Which means that, conceivably, exercise will impact on adipose tissue blood flow in the same way that it does on adipose tissue metabolism as a whole. That is, depending on the relative concentration of each hormone, along with the ratio of alpha- and beta-adrenoceptors, ATBF may be controlled by exercise, diet, etc. in the same way that fat mobilization is.

Interestingly, insulin also affects blood flow but not in the way that you think. Insulin, via stimulation of something called nitric oxide (NO, yes the same stuff that all of the new supplements claim to affect) increases blood flow; this is part of why folks get vascular and pumped when they consume carbs before training. So raising insulin should help with fat loss, right? Wrong. Remember that insulin is profoundly anti-lipolytic, as discussed above. As it turns out, many of the compounds that increase ATBF end up inhibiting lipolysis.

For example, nitric oxide is anti-lipolytic so don't think you can trick the system by using a NO supplement before aerobics. It might increase blood flow to adipose tissue (maybe) but at the consequence of inhibiting fat breakdown.

In any case, let's assume that everything has worked right and stored triglyceride has been broken down into fatty acids and glycerol. Both enter the microcirculation around the fat cells. As mentioned, glycerol can't re-enter the fat cell so let's forget about it.

Some of the released FFA bind to a protein in the blood called albumin. If blood flow is particularly sluggish, fatty acids just get re-esterified into the fat cell, accomplishing nothing. If blood flow is good or increased, the albumin bound FFA will get carried far far away from the fat cell. Let's assume this latter event has occurred and see what happens to the fatty acids bound to albumin now being carried through the bloodstream.

Step 3: Uptake and utilization

So now we have albumin bound FFA floating around in the bloodstream; this makes the FFA generally available to other tissues in the body. Eventually, the albumin bound FFA will come across a tissue (such as the liver or muscle) which can use it for fuel. The FFA will be taken up into that tissue (there is a specific fatty acid binding protein which transports the fatty acid into the cell) at this time for one of a couple of potential 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. I'll only focus 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, that by increasing levels of CPT, you get more fat burning. Looks great on paper although it's never really panned out (the reasons why are discussed in detail in my **Protein Book**). 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 muscle glycogen levels.

Glycogen is a long carbohydrate chain stored in your muscles or liver. When glycogen is high, CPT activity is low and fat oxidation is low, and vice versa. This is true for both muscle and liver. By depleting muscle glycogen, dieters can increase CPT activity, allowing them to better burn off the fatty acids that we mobilized from the fat cell.

I suspect that this is part of why certain types of training such as high rep short rest periods work the way they do. While the usual explanation has to do with the post-exercise energy expenditure, this turns out to be pretty small and fairly irrelevant. However, by depleting full body glycogen stores, fat oxidation is ramped up ensuring that mobilized fatty acids will get burned for energy (note: this is why I included a depletion phase in the **Ultimate Diet 2.0**). On a caloric deficit this is clearly a nice thing.

Summary

When people refer to fat burning, or just to fat loss in general, what they mean is that stored fat (triglyceride) is first broken down in the fat cell, released into the bloodstream, and subsequently used to produce energy in another tissue such as the liver or muscle.

Fat breakdown is arguably the crucial step as you can't lose fat if you can't get it out of the cell (liposuction excepted). While other hormones clearly play a role, the two most important hormones controlling fat breakdown are insulin and the catecholamines. The impact of insulin is distinctly negative, insulin inhibits fat breakdown at even low levels.

The catecholamines are trickier and can exert a lipolytic effect or anti-lipolytic effect depending. The ratio of beta- and alpha-adrenoceptors as well as the levels of each hormone, impacts on whether a net lipolytic or anti-lipolytic effect is seen at the fat cell.

Assuming that fat has been mobilized, it moves out of the fat cell into the microcirculation. Ideally, all released fatty acids would be carried away, bound to a protein called albumin. However, the fat cell will happily store the fatty acid back in the fat cell if blood flow is poor. A number of factors control blood flow including hormones and, once again, adrenoceptor levels.

Once bound to albumin and carried away from the fat cell, fat can be used for energy in tissues such as the liver or muscle. Under certain conditions, the transported fatty acids can be stored (e.g. as intramuscular triglyceride) although this is unlikely to occur in a caloric deficit.

Now that you understand the basics of both fat storage and fat mobilization, I want to delve further into what controls fat patterning, focusing primarily on hormone levels.

Hormones and Body Fat

Having addressed the basics of how fat is both stored and burned, I next want to look at how various hormones affect fat cell metabolism and storage.

For each hormone, I'm going to talk about not only global effects on fat storage or mobilization, but also how those hormones might impact on such things as adrenoceptor number, adipose tissue blood flow or fat oxidation (i.e. the three key processes I discussed in Chapter 5).

Insulin

I've already discussed insulin in some detail in the previous chapters in terms of how it affects fat cell metabolism. Of all the hormones involved, insulin is generally put rather simplistically into the 'bad' category when it comes to fat loss and there is some truth to that. As a general storage hormone, insulin is involved in fat storage and clearly inhibits fat mobilization (by inhibiting HSL) as well.

However, insulin is one of the hormones that signals the brain about the body's energy status; quite in fact, injecting insulin into rat brains makes them lose their appetite and lose weight. Unfortunately, the human body doesn't seem to respond so nicely. One study that gave inhalable insulin did show a decrease in food intake in humans. However, chronically elevated insulin (as occurs with obesity) doesn't seem to do much to turn off appetite or cause weight loss. This is probably because the brain has become resistant to the effects of insulin.

Insulin is primarily under control of the diet and can be affected by either changing the quality (type) or quantity (amount) of carbohydrates that you're eating. Insulin levels will be reduced if you pick lower Glycemic Index (GI) carbs or simply reduce the quantity of carbohydrates that you're eating.

Interestingly this actually appears to have different effects on such things as insulin resistance and fatty acid release; reducing the quantity of carbs eaten causes insulin resistance (actually a good thing on a diet for reasons I discussed in the UD2) and increases fatty acid release from fat cells. Changing only the quality of carbs eaten does not have

the same effect, maintaining good insulin sensitivity and blunting fatty acid release. In this context, lowering carbohydrate quantity is probably the more beneficial approach.

Glucagon

Since I've just talked about insulin, I should talk about its 'sister' hormone which is glucagon. Released when blood sugar falls, glucagon's primary responsibility is to stimulate the liver to break down stored glycogen and release glucose. A lot of people talk about the insulin:glucagon ratio and this is related to the carbohydrate content of the diet (protein also plays a role in glucagon's release).

Based on animal research, glucagon is often claimed to be lipolytic; but this only holds for animals (mainly of the rat and mouse variety). In humans, glucagon has no effect on lipolysis and I have nothing more to say about glucagon than this: I wish the people who still go on about how glucagon is a fat mobilizer would read research past 1972 and stop spouting out of date nonsense.

The catecholamines: Adrenaline and noradrenaline

Like insulin, I already discussed the catecholamines to some degree but I'll recap here. Adrenaline is released from the adrenal gland, noradrenaline from the nerve terminals. Collectively they are often termed the fight or flight hormones since they tend to be released under those conditions. As mentioned, adrenaline and noradrenaline are called epinephrine and norepinephrine everywhere in the world except the US. We just rock that way.

Adrenaline and noradrenaline are involved in mobilizing fuel for the body. In the liver, they promote the breakdown of liver glycogen to glucose; in fat cells (as discussed last chapter) they generally increase fatty acid mobilization to provide fuel to the body. Of course, they have numerous other effects such as increasing heart rate and blood pressure, making you empty your bladder if it's full (but preventing urine production), blunting appetite, and inhibiting digestion (this is why you cramp if you exercise too close to eating).

Just about any stressor from physical to psychological will increase the release of adrenaline and noradrenaline but exercise is, generally speaking, what we'll use to control these hormones. As I'll detail in a subsequent chapter, the type and intensity of exercise that you do affects how adrenaline/noradrenaline are released and this is a big key to targeting stubborn fat.

Diet can also impact levels of these hormones, lower carbohydrate diets often increase catecholamine levels (to help with fuel mobilization) although eating carbohydrates can raise levels too (by first raising insulin which stimulates nervous system output). Quite in fact, the thermic effect of eating carbohydrates is directly related to the increase in insulin which drives adrenaline/noradrenaline release.

Of course, supplements can raise levels of these hormones. Caffeine has a small effect although this is mostly lost with chronic use. Ephedrine not only mimics adrenaline/noradrenaline in the body, it increases the output of those hormones. The amino acid L-tyrosine, which I'll mention again in Chapter 9, can also help to increase catecholamine release in response to other stimuli (such as exercise, ephedrine, or caffeine).

Growth hormone (GH)

GH is a hormone that has gotten altogether too much attention in the sports nutrition world. Claimed as a muscular anabolic, GH does do amazing things in people who are deficient and have their levels replaced. However, increasing GH in otherwise normal individuals has far less of an impact. Injectable GH, at least by itself, does nothing to increase muscle growth although it appears to synergize with other compounds/drugs. However, raising GH with various supplements and training strategies does nothing for muscle growth. In fact, many of the strategies aimed at increasing GH are probably detrimental to muscle growth in that they require avoiding food or fluids for long periods.

When it comes to fat loss, GH does affect lipolysis; in fact, preventing the body from releasing GH under conditions of starvation increases muscle loss because the body can't optimally mobilize fatty acids. Injectable GH is known for having profound effects in dieting bodybuilders; but the levels seen with injection simply aren't attainable in any non-drug fashion.

In the big scheme of all things lipolytic, GH is of secondary importance and quite delayed in its effects. While normal physiological pulses of GH increase lipolysis, the effect is small and occurs several hours after the GH pulse occurs. Of some interest, the normal nighttime GH pulse appears to be required for optimal lipolysis the next day.

GH is sensitive to diet of course, typically going up when carbohydrates are restricted to help mobilize fuel. Exercise of just about any sort raises GH as well. While various combinations of amino acids (usually arginine and ornithine) have been touted as GH releasers, they generally taste like bleach and require massive, vomit inducing doses. Low dose glutamine (2 grams) has been shown to increase GH levels and I recommended taking some at bedtime and prior to training in the UD2. Whether it actually impacts on fat loss is unknown. It may do nothing, although it certainly won't be hurt anything.

Cortisol

Traditionally, athletes have put cortisol in the 'bad' category of hormones; in more recent years, cortisol may have become the most maligned hormone of them all. Cortisol makes you fat, cortisol makes you weak, cortisol is probably responsible for the situation in Iraq. At the same time, individuals who don't have enough cortisol (this occurs in certain disease states) know that producing no cortisol is not a good thing. You have no energy, joints hurt, injuries won't heal; cortisol is required for life.

The reason for this discrepancy is that cortisol is a very schizophrenic hormone and can have either profoundly beneficial or profoundly negative effects. The basic situation is that normal physiological cortisol pulses are beneficial, adaptive and a good thing to have. Such pulses stimulate lipolysis (in fact, the morning cortisol pulse is required for optimal lipolysis), improve memory, and improve adaptation. To my knowledge, cortisol does not significantly impact on either adipose tissue blood flow or fat oxidation in other tissues. But it is part of mobilizing fatty acids for fuel. Like GH, cortisol plays a distinctly secondary role in stimulating lipolysis, having a minor effect that doesn't appear for several hours. Physiological pulses of cortisol are a very good thing.

However, chronically elevated cortisol, which can occur during conditions of sustained stress (such as excessive training, dieting or a combination of the two), is distinctly a bad thing. It is heavily involved in depression, causes memory and verbal problems, impairs immune function and has a host of other negative health effects. Chronically elevated cortisol is a very bad thing.

In conjunction with elevated insulin levels (and I'd note that high cortisol causes insulin resistance, which can lead to high insulin levels), cortisol is extremely lipogenic, especially in visceral fat. Individuals with chronically elevated cortisol often gain fat around the midsection. Cortisol (along with the hormone aldosterone) also causes water retention; this can mask true fat loss. Additionally, cortisol also induces leptin resistance (leptin is discussed below), preventing leptin from exerting its metabolic effects.

Cortisol shows a daily rhythm, ideally peaking in the morning and going down at night. This can be thrown off by a number of things. Hardcore food restriction can cause the normal pattern to shift forwards, you see lower levels in the morning and elevated levels in the evening. Low-carb diets tend to initially increase cortisol (to help mobilize fuel) although levels may come down after the body adapts to running on ketones.

One of cortisol's primary effects is to mobilize glucose to sustain blood glucose levels, it does this by increasing the production of glucose in the liver. This is on top of mobilizing protein (often from muscle) to use to make that glucose. Logically, higher carbohydrate diets tend to keep cortisol levels down although excessive training and/or general caloric restriction offsets this.

As I mentioned in a previous chapter, one area of current interest has to do with the metabolism of cortisol within fat cells themselves, via the enzyme 11-beta-hydroxysteroid-deydrogenase (11-beta-HSD). There is evidence that this local cortisol metabolism may be impacting (usually negatively) on fat cell metabolism and ways of modulating it may have some use. I'll mention an intriguing one in Chapter 9.

I bring up the excessive training aspect because it is profoundly easy to overtrain when dieting to extremely low body fat levels and most people try to train too hard too often while they are dieting. People who ignore my warnings when I present the stubborn fat protocols later in this book will end up doing themselves great harm when they drastically overtrain on lowered calories.

When dieting to extremely low levels, it's crucial to keep training volume under control to avoid overtraining. Keeping carbohydrates higher (or refeeding/taking breaks) is also of

value. Generally trying to keep away from stressful aspects of life is great advice but not always practical.

In terms of supplements, Vitamin C (2 grams/day) may help with elevated cortisol levels, phosphatidylserine (400-800 mg/day) has also shown some efficacy. There are a number of herbs (theanine, bacopa and many others) that are often suggested to help with elevated cortisol levels. They might be worth considering if someone is having problems and nothing else was working; I can't say I've seen much convincing data that they do much.

Thyroid

In the same way that cortisol has generally been categorized as 'bad' in the bodybuilding subculture, thyroid hormones are generally thought of as 'good'. As usual, this is a terrible oversimplification.

There are two primary thyroid hormones which are T4 and T3 (I'll spare you the full names); T2 also has some acute metabolic effects. The body releases both hormones in roughly an 80:20 ratio of T4:T3 from the thyroid gland, most T3 production occurs in other tissues via an enzyme called Type II Deiodinase (which removes one of the iodine molecules from T4 to produce T3), much of this occurs in the liver.

T3 has profound impacts on all aspects of metabolism and is one of several hormones (along with the catecholamines, insulin and leptin) which regulate metabolic rate. I'd point out that thyroid is not the only important hormone in terms of regulating metabolic rate; if it were then keeping diets moving would be no more difficult than just taking some form of thyroid (whether synthetic or natural). But this doesn't work because other hormones are relevant as well.

Which isn't to say that thyroid doesn't play a major role in regulating metabolic rate, just that it's not the only hormone involved. Metabolically, thyroid has both short-term and long-term effects on metabolism. Acutely, the breakdown of T3 to T2 has uncoupling effects in mitochondria. Long-term, T3 has effects on gene expression that not only take time to fully exert themselves but may last for 3-4 weeks before disappearing after thyroid levels drop.

Of extreme relevance to fat loss, thyroid hormones interact synergistically with the catecholamines: catecholamines increase thyroid conversion and thyroid increases catecholamine action. Bodybuilders have often stacked clenbuterol and thyroid medication and this is a very potent combination.

Thyroid hormones affect a number of processes important to the issue of fat loss and stubborn fat. Studies have shown that individuals with low thyroid have poorer adipose tissue blood flow and that correcting this fixes the problems; hyperthyroid individuals have high levels of adipose tissue blood flow.

As mentioned above, thyroid hormones also directly impact on how well the catecholamines stimulate lipolysis; when thyroid is low, lipolysis is inhibited. Of course,

by affecting overall metabolic rate, thyroid helps the body to burn off fatty acids by increasing how rapidly all tissues use fuel.

Which isn't to say that more thyroid hormone is better. Excessive thyroid levels have a negative effect on muscle mass. Studies using high dose thyroid while dieting find greater protein loss; studies using much more realistic physiological doses generally don't.

Of some interest to the issue of stubborn body fat, it's interesting to note that women are far more likely to have sub-optimal thyroid levels than men and something like 9 out of 10 thyroid prescriptions are made for women. Given the impact of thyroid hormones in modulating overall lipolysis and adipose tissue blood flow, along with its overall effects on metabolism, the fact that women are starting with lower than optimal thyroid levels is yet another reason that lower body fat is such a problem.

And that's before even considering the drop in thyroid that occurs with long-term dieting which I want to talk about next. In contrast to true hypothyroidism (which occurs due to a defect in the thyroid gland), the drop in active thyroid hormone with dieting (referred to as Euthyroid Sick Syndrome or ESS) is due to reduced conversion of T4 to T3 in the liver.

A number of mechanisms mediate this including decreased T4 uptake into the liver along with decreased activity of the deiodinase enzyme. High blood fatty acids are involved in the inhibition of T4 uptake into the liver, while high levels of cortisol along with decreased liver ATP levels are primarily responsible for the drop in deiodinase activity.

Both men and women have to contend with ESS on a diet but, clearly, women who are starting out with sub-optimal levels of thyroid hormone in the first place are in an even worse boat. A female who is starting out with low-normal thyroid levels to begin with will have levels crash on an extended diet; not only does this slow overall fat loss but it makes getting rid of stubborn lower body fat all that much more difficult.

Clearly if someone is starting out with low thyroid levels, there's not much that can be done short of obtaining either a synthetic thyroid compound (in the US, Cytomel is the most common T3 drug) or natural compound (for example, some holistic physicians will use a compound called Armour which is made from desiccated pig's thyroid gland). This may be worth pursuing as low thyroid levels cause other problems such as depression and water retention; low thyroid levels also hurt protein synthesis. I know of at least one diet guru who has had his female clients respond amazingly to small, replacement doses (12.5 mcg) of T3. This is enough to raise levels slightly without causing muscle loss or any of the problems inherent to higher dose intakes.

If someone is not willing to go that route, there's not much that can be done to correct a true low-normal thyroid level. Getting sufficient iodine helps, excessive soy protein intake can hurt (especially in conjunction with too little sodium) and that's about it.

In regards to ESS per se, a number of strategies have been suggested. Guggulsterones may increase either uptake or conversion of T4 in the liver and various phosphate salts were suggested to do the same, but they don't seem to have had much real world impact on fat loss or anything else. Ephedrine increases T4 to T3 conversion as well.

Frankly, taking proper diet breaks (periods of 10-14 days when calories are raised to maintenance with at least 100-150 grams per day of carbohydrate) during a diet is probably the best way to go about getting thyroid levels back up while dieting. While this won't fix a true hypothyroid state, it will help against the ESS related drop in T3 levels that occur with prolonged dieting.

Leptin

If you've read any of my books, you've seen me write about leptin. If not, I'm going to give you the short course here. Leptin is possibly one of the single most important hormones in terms of body weight regulation, appetite, etc. Released primarily from fat cells (muscle, stomach and even the brain also produce leptin), leptin signals the brain about how much fat you're carrying and how much you're eating.

Originally it was thought that leptin acted as an 'anti-obesity' hormone but this turns out to be incorrect. Leptin doesn't do much to prevent folks from gaining weight. Where it shines is as an 'anti-starvation' hormone; leptin controls most of the body's response to fasting/caloric restriction/weight loss, acting to slow further weight loss to keep people alive until food becomes available again.

When someone diets, leptin goes down much faster than fat mass and this has a primary controlling effect on metabolic rate, hormones, appetite, immune function and a host of other processes. And while studies that gave injectable leptin to fat people have consistently crapped out, other studies which replaced leptin to pre-diet levels after weight loss found improved thyroid levels, increased metabolic rate and continued fat loss. That is to say, increasing leptin above normal has little effect; keeping leptin from dropping on a diet has absolutely massive effects.

Leptin stimulates fat oxidation in skeletal muscle and liver, and plays a role in fat mobilization from fat cells. As well, it impacts on other hormones (e.g. thyroid, cortisol, estrogen, testosterone) which affect fat cells and fat loss; thus, leptin has both direct and indirect effects in this regards. That's in addition to the zillion other things it appears to do in the body. Injectable leptin would fix most of the problems that occur with dieting; currently costing $500 PER DAY for an effective dose, this is just another pipe dream.

At maintenance calories, leptin scales frighteningly well with fat mass. Cut calories or get lean and leptin drops precipitously. In men below 10% body fat, leptin is often undetectable in the bloodstream and this is part of what contributes to the general systems crash that can occur in male bodybuilders dieting to low body fat levels.

At any given level of body fat, women typically produce 2-3 times as much leptin; there is also some indication that leptin falls faster in women than men and that women's bodies may respond differently to this drop. Tangentially, this gender difference in how the body responds to leptin may be part of why women generally have more trouble losing fat than men. This is currently a topic I'm investigating in great detail and will be the topic of a future book project.

Leptin can be modulated somewhat by diet. Dieting of any sort will lower leptin. Overfeeding with high calories and high carbohydrates (i.e. the refeeds and diet breaks I talk about in my books) raise it. Other nutrients such as zinc and Vitamin E have been shown to increase leptin production. Contradictorily, fish oils reduce leptin but they appear to do this primarily by decreasing body fat levels.

Other hormones such as cortisol and estrogen also impact on leptin levels as well. Cortisol increases leptin levels, but decreases the sensitivity of tissues to leptin. Estrogen appears to increase leptin levels and is probably a lot of why women have higher levels than men; estrogen may also determine leptin sensitivity in women's brains and recent work suggests that estrogen may actually mimick many of leptin's beneficial effects in the brain.

Atrial natriuretic peptide (ANP)

So far in this chapter, I've focused on hormones that not only have well established effects on some aspect of fat loss (or stubborn body fat) but that can also be manipulated or controlled in some way. Before moving onto the topic of the sex hormones, I want to change gears and talk about a hormone of extreme interest that, as of yet, I'm not sure can be controlled. However, the possibilities inherent in this hormone are so fascinating with respect to stubborn fat loss that I think it's worth mentioning.

That hormone has the involved name of atrial naturiuretic peptide (ANP). Natriuretic peptides are actually a class of different hormones of which ANP is simply one. Released from the heart, ANP is involved heavily in water balance (it often goes up in hypertension, presumably to get rid of some body water and bring things back to normal).

So why is this important for stubborn body fat loss? As it turns out, ANP has been found to stimulate lipolysis. Nothing exciting there. Except that ANP works through an entirely new pathway that doesn't involve the adrenoreceptors. That statement will make a lot more sense after you've read the next chapter but, as I've mentioned previously, different depots of fat have different ratios of the adrenoceptors and that influences how easy or difficult it is to mobilize fatty acids. A huge part of the problem with stubborn fat is that the adrenoreceptor ratios present make it difficult to mobilize stubborn body fat.

A hormone that stimulates lipolysis that works around the adrenoreceptors is something exciting indeed. Unfortunately, I've found no practical way of modulating ANP. Hyperhydration (achieved with saline infusion) seems to raise ANP but I don't know if this is achievable with something like massive water intakes (perhaps with sodium). Interestingly some bodybuilding contest prep gurus are now recommending massive sodium intakes during contest prep, along with tons of water; they claim that after the competitor gets sloppy soft, they start leaning out and dry out. Perhaps this is helping via an ANP mediated pathway. Perhaps it's just affecting water balance. In any case, ANP is a hormone to keep an eye on. We may not be able to do much with it now, but that could change with new research.

The sex hormones: Testosterone, estrogen and progesterone

As I mentioned in an earlier chapter, little boys and girls typically have similar body fat patterns and levels until they hit puberty at which point the typical sex differences in fat patterns develop.

Of course, since the primary changes that occur at puberty are increases in testosterone (males) and estrogen/progesterone (females), this suggest a primacy of these hormones in determining fat patterning and physiology.

Studies of transsexuals clearly bear this out. Male to female transsexuals put on androgen blockers and female hormones lose their belly fat and develop hip and thigh fat. Female to male transsexuals who are put on testosterone therapy show a shift to a male fat patterning, gaining visceral fat and losing hip/thigh fat.

Interestingly, over the long-term, there is often a subsequent return to the previous fat patterns along with the new ones; that is, male to female transsexuals can end up with both visceral fat and hip/thigh fat. This is an important point that I'll come back to below as it is one indication that levels of the hormones per se are not all that's important.

In a related vein, as females age and enter menopause (at which point their ovaries shut off), there is often a shift away from female fat patterning (loss of hip/thigh fat) towards male fat patterning (increase in visceral fat). Females who go on hormone replacement therapy early enough show no such shift, maintaining the normal female fat pattern.

With regards to stubborn male body fat, testosterone is actually another schizophrenic hormone; this is because it has effects on both LPL and HSL (discussed in a previous chapter) depending on the levels of testosterone present. Men with low testosterone levels will often show fat loss when given testosterone replacement therapy; men who have above normal levels of testosterone (either genetically or from drug use) often accumulate visceral fat. Subcutaneous abdominal fat is also responsive to testosterone.

In addition to potentially affecting both fat storage and mobilization via LPL and HSL, testosterone also affects adrenoreceptor number. While it increases beta-receptor number (increasing lipolysis), it can also increase alpha-2 receptor number (inhibiting lipolysis). Depending on which is affected more, this could either help or hurt lipolysis. To my knowledge, testosterone doesn't affect adipose tissue blood flow.

Although testosterone clearly plays a role in visceral and abdominal fat accumulation, it would be fairly silly to suggest lowering testosterone to try and get rid of that fat. Visceral fat is relatively easy to get rid of (except perhaps in heavy androgen users) and abdominal fat will eventually come off if men are patient enough. Testosterone generally crashes when men get super lean anyhow so it's not something folks need to worry about or work to achieve.

Which brings us to the primary female hormones, estrogen and progesterone. Given the impact of female puberty on female fat patterns, it's fairly easy to see how the idea that 'Estrogen causes lower body fat' came into popularity; that of course leads down the road to 'Blocking estrogen will eliminate lower body fat'. If it were only that simple. As Dan

Duchaine pointed out years ago, anti-estrogens only helped a little bit with fat loss, they didn't solve the problem. Ergo, estrogen alone is not the problem.

If that doesn't convince you, consider this. As women get super lean, estrogen levels drop; this is a big part of why the menstrual cycle goes bye bye. Yet losing lower body fat doesn't get any easier when this happens. If estrogen were THE cause of stubborn lower body fat, it should get easier to lose as estrogen drops and that simply isn't the case. You'll learn why next chapter.

To say that estrogen is a schizophrenic hormone in terms of fat loss and gain is a vast understatement. I'm tempted to say something poetic like 'Estrogen is as mysterious as women themselves' although my frustration with what these hormones do in the body makes me more likely to say 'Estrogen is just as crazy as women themselves'.

Part of the problem is that women show huge shifts up and down in both estrogen and progesterone during the month and they tend to have over lapping and synchronizing effects. Figuring out what is being caused by estrogen, what by progesterone and what by estrogen priming the body for progesterone's effects is extremely difficult.

Let's look at some of the conflicting data so you can see why this is more complicated than saying one or the other is good or bad.

On the one hand, women are known to burn more fat than men during aerobic exercise especially during the part of their menstrual cycle when estrogen is dominant; quite in fact if you inject males with estrogen they will burn more fat for fuel during aerobic activity. This *should* help fat loss.

However, estrogen can negatively impact on fat mass as well. One study showed that even acute exposure to estrogen increases alpha-2 receptor number. Estrogen may also directly lower levels of cAMP by decreasing the activity of one of the key enzymes involved in producing it in the fat cell. This *should* hurt fat loss.

Furthermore, postmenopausal women often gain body fat and estrogen/progesterone replacement can fix this. Studies suggest that estrogen may play a key role in maintaining proper leptin sensitivity in the brain as well. As mentioned above, estrogen may mimic many of leptin's signaling in the brain. This *should* all help fat loss.

Do you see the problem? Can you see why it's a huge problem to simply blame estrogen in this case? Estrogen has effects that are both beneficial and distinctly negative depending on what tissue and what physiological process you're looking at. Which brings us to the next logical question which is 'Why does estrogen act so strangely?' Recent research may have brought us closer to understanding what in the hell is going on.

As it turns out, there are two different estrogen receptors (estrogen receptor alpha and beta) which are found in different amounts in different tissues; when activated they can have vastly different end results. This is, in fact, very similar to how the adrenoceptors work, the ratio of different receptors on a tissue can drastically impact on what actually happens when the catecholamines are present.

The existence of two different estrogen receptors actually explains an earlier observation whereby estrogen antagonists could have anti-estrogenic effects in one tissue but pro-estrogenic effects in others; different amounts of receptors and binding affinities cause differential effects to be seen in various tissues. Tangentially current research is trying to find selective estrogen receptor modulators (SERMS) that will allow the effects of the drug to be much more targeted. In the future it might become possible to block the 'bad' effects of estrogen in terms of fat loss without affecting some of the 'good' effects.

If that weren't complicated enough, estrogen can also exert effects by binding to the surface of a cell. It may turn out that whether estrogen has an overall positive or negative impact on fat loss depends on what tissue you're talking about and whether estrogen is working through the alpha receptor, beta receptor or cell surface receptors.

And I haven't even brought up progesterone yet.

Several studies suggest that progesterone is actually the big player in fat storage; however estrogen primes the progesterone receptor so it can't be ignored either (before you go looking for anti-progesterone compounds, note that they all have pretty horrible side-effect profiles).

In any case, effects on fat storage or gain may be the synchronized effect of estrogen and progesterone at play, or there may be something else going on entirely. At this point, it's just really hard to get to the bottom of this issue and tease out the individual effects of each hormone.

And the above is only dealing with the effects of estrogen/progesterone on fat storage or oxidation. Both also clearly also affect appetite (the drop in estrogen appears to be the cause of increased appetite towards the end of the cycle moreso than the increase in progesterone) and can cause water retention; this can mask true fat loss. I suspect that some of the perceived effect of various anti-estrogen compounds (such as di-indole methane or indole-3 carbinole, compounds found in broccoli that impact on estrogen metabolism) has to do with dropping water more than actually affecting body fat.

Looking at metabolic rate, while estrogen has no effects I'm aware of, progesterone is thermogenic and can raise metabolic rate by 3-5%. Of course, during that week of the cycle is when hunger is generally off the map and it's easy for most women to out-eat the slight metabolic boost that progesterone gives.

Sufficed to say, and this is really my main point, estrogen isn't the sole cause of women's lower body fat problems for reasons I hope I've made clear. At least not in the sense of 'Simply taking an anti-estrogen will make all of the problems magically disappear.'

But it may still be playing a huge role in another sense, which I'll discuss next chapter.

7

Why is Stubborn Fat Stubborn

So you're now nearly 50 pages into this book and still wondering why stubborn fat is stubborn. Perhaps you've picked up some of the reasons by inference but finally, in this chapter I can put everything together.

Fat cell overview/review

In the past 6 chapters, you've learned a ton about fat cells and fat cell metabolism. One of the points I've tried to get across is that fat cells are not the same, different depots have different functional characteristics in terms of how easily they store fat, how easily they give up that fat, etc.

In general there are clear gender differences that show up at puberty, suggesting that sex hormones play a role in how fat cells develop. And there is much truth to this. However, that's not all that's going on.

It turns out that if you take a fat cell from a man's thigh and a woman's thigh, they are functionally identical and essentially indistinguishable physiologically. This is true even though the man generally has extremely low levels of estrogen. His lower body fat cells are still identical to a woman's. The difference, practically, is that men don't generally store fat in their legs and women do (i.e. the fat cells in a man's legs are emptier than in the woman's). As I mentioned before, men who *do* store fat in their lower body have the same problems as women. But most men don't store fat there.

The same holds true for visceral or abdominal fat from a woman versus a man. The female's visceral/ab fat is physiologically identical to the man's, although she has very low levels of testosterone. It's simply that, on average, she won't tend to store fat in that area. Again, the fat cells are identical, the difference is in the propensity to store calories there or not.

What this suggests is that fat cells in different areas of the body have certain physiological characteristics that occur irrespective of hormone levels. So while the hormonal setting may affect where ingested calories get sent, they aren't really controlling the underlying physiology of the fat cells.

Which is fundamentally why blocking estrogen doesn't fix the lower body fat problem. Lower body fat cells act a certain way whether estrogen is present or not, that's how they are genetically wired to act. The same goes for abdominal fat. Regardless of the person's testosterone levels, abdominal fat cells are simply wired to be a certain way. Now it's time to learn what that wiring is and what makes stubborn fat cells stubborn.

Adrenoceptor redux

Recall from Chapter 5 that there are two types of adrenoceptors that control not only fat cell metabolism but also blood flow into and out of the fat cell. Beta-2-receptors can be thought of as the 'good' receptors, increasing lipolysis and adipose tissue blood flow. In contrast, alpha-2-receptors are distinctly bad, inhibiting lipolysis and adipose tissue blood flow.

So why does this matter? Different areas of body fat have different distributions of alpha-2 and beta-2 adrenoreceptors and this profoundly affects how well or poorly fat can be mobilized and transported out of the fat cell.

The most extreme example of this is lower body fat (hips and thighs), which have been found to have roughly 9 times as many alpha-2 receptors as beta-2 receptors. Some research suggests that men's abdominal fat has higher alpha-2 receptor density (relative to say, visceral fat) although it's not as bad as lower body fat. While not studied, lower back fat is likely to also be relatively resistant to lipolytic stimuli due to a greater alpha-2 receptor number.

This is clearly part of why stubborn fat is so stubborn, the normal lipolytic stimuli that should mobilize fatty acids don't work effectively. Quite in fact, due to the high alpha-2 receptor density, certain types of exercise can actually be anti-lipolytic. You'll learn more about that in the next chapter.

Now couple that with the information I presented earlier about how men and women store calories after eating. Women's bodies may preferentially shuttle calories into lower body fat after a meal, on top of possibly redistributing fat from upper to lower body fat. Yet, they can't be mobilized *out* as rapidly due to the adrenoceptor ratios. Basically it's a double whammy: women tend to store more fat in their lower bodies after meals, their bodies may transfer fat from the upper to lower body, yet they can't as easily get the fat back out.

Years ago I remember some women claiming that while their upper bodies leaned out, they swore their legs were getting fatter. I dismissed it as nonsense at the time but the above physiological facts lend support to that idea. A woman might be mobilizing fat from her upper body fine, yet storing some of that fat (or incoming calories from meals) in lower body fat later in the day. Upper body gets leaner, lower body gets fatter. My friend Elzi Volk said it best years ago 'When it comes to fat loss, women are screwed.' She had no idea.

Blood flow redux

In addition to differences in responsiveness to lipolytic stimuli, certain fat depots have significantly poorer blood flow than others. You can test this yourself, touch an area of your body where you lose fat easily, it should feel fairly warm. Now touch your butt, hips or thighs. Probably stone cold.

Studies have shown that blood flow in the lower body fat may be as much as 67% lower than in other depots and this holds true for both men and women. Visceral fat has extremely good blood flow, it also goes away very quickly. If you could drive your hand into someone's stomach and feel their visceral fat, it would probably feel fairly warm.

Poor blood flow has two consequences of importance here. First and foremost, it means that blood borne hormones (such as the catecholamines which, recall, don't work well to mobilize stubborn body fat in the first place) can't get to the fat cells. Second, poor blood flow makes it harder to get mobilized fat away from the fat cell so that it can be burned elsewhere.

Why the blood flow is so poor isn't well established. Part of it may simply be fewer blood vessels, imaging studies show very few in that area. As well, it appears that the blood vessels in the lower body have more alpha- than beta-receptors; this has the same consequence as for lipolysis. More alpha-receptors means more vasoconstriction and less vasodilation which adds up to less blood flow.

Insulin redux

As I noted, after a meal, blood flow to the lower body increases preferentially in women, due to the effects of insulin. Lower body fat is also more sensitive overall to the anti-lipolytic stimuli of insulin. Contrast that to visceral fat which is not only super-sensitive to lipolytic stimuli but also relatively insensitive to insulin. Ab fat is somewhere in the middle for both, somewhat sensitive to insulin, somewhat sensitive to the lipolytic effects of the catecholamines.

Differential fatty acid storage

Another factor that I haven't discussed yet has to do with a relatively unknown fact about fat cell metabolism. Studies have shown that the type of fat stored (e.g. saturated versus unsaturated) affects how well it is mobilized from the fat cell. Unsaturated fats are mobilized more easily than saturated fats and the polyunsaturated fats (i.e. fish oils) are mobilized the most easily of all.

What this means is that the more unsaturated/polyunsaturated fat that is stored in the fat cell, the easier it will be to get out. This is also a big part of why fish oil supplementation is so crucial while dieting, the body preferentially depletes its own body stores for energy.

Guess what the punch line of this section is? Stubborn fat depots are more likely to store saturated fat than unsaturated or polyunsaturated fat. You can test this out too, pinch

some of the fat in a relatively easy to lose area. It should be soft and somewhat squishy. Now pinch your thigh or butt fat. Hard as a rock, right? That's because saturated fat is structurally different than unsaturated fats (saturated fats are solid at room temperature, unsaturated fats are liquid).

Now, before continuing on in this chapter and summing up, I want to make a practical comment about the difference in fatty acid type between depots. And that comment is basically that, while I can tell you how to modulate everything else I'm discussing in this book, what type of fat you currently have stored in your fat cells right now is out of your control. What you've eaten over the last 20 or 30 years while storing your current body fat determines the types of fat stored there. If you're dieting down for the first time, there's simply nothing you can do about it.

However, after your diet is over and you regain some fat (trust me, you will), what you can do is try to keep saturated fat intake down and unsaturated/polyunsaturated fat intake higher while you're doing it. That alone should make your next extreme diet easier since the fat stored in your fat cells will now be the easier to mobilize unsaturated/polyunsaturated fats. I don't usually quibble that much over food quality but this is one place where, practically, it might make a difference in the long run.

Fat cell size

Another issue that potentially contributes is fat cell size which turns out to impact on a number of aspects of fat cell physiology, especially lipolysis (which is greater in larger fat cells). As fat cells shrink, lipolysis decreases.

Of more relevance, some people seem to have relatively more but smaller fat cells while others have fewer but larger cells. The latter group will, generally, have an easier time dropping fat. However, that I can tell, research hasn't consistently identified differences between fat cell depots in terms of size versus number. Rather, it may just be the genetic lottery that determines this. In any case, it's out of your control and not worth worrying about. I mention it only for completeness.

A more global issue

Of course, moving a little beyond the issue of fat cells themselves, there's another big reason that stubborn fat is stubborn and that has to do with when it has a chance to come off in the first place. That time, of course, is at the end of a long diet. As I've noted, the body will always take the easier to mobilize fat first and only when that's gone will it even touch the stubborn fat.

Again, that generally comes after a long dieting period and there are lots of other factors occurring then that conspire to make stubborn fat harder to get off. The first are hormones: leptin levels will be down, thyroid will be down, cortisol will be up, nervous system output is typically down. This causes an overall reduction in metabolic rate and lipolysis above and beyond the issues discussed above. Of course, fat cells are smaller at this point, which further inhibits lipolysis. Typically dieters are starting to get hungry,

making it more likely that they'll break their diet and stop fat loss in its tracks. Motivation is often down and maintaining training intensity in the face of a seemingly endless diet becomes difficult.

Short of using various strategies (nutrition or drug) to try and fix the hormonal issues, about the only thing that can be done is the proper implementation of refeeds and diet breaks to try and restore hormones to something approximating normal to help the fat come off a bit easier. I'll talk about this in a bit more detail in later chapters but interested readers should either pick up my **UD2** or **Guide to Flexible Dieting** which deals with the issues I've mentioned here in some detail.

Summing up

So this chapter basically is the first punch line of the book, finally defining, physiologically, what makes stubborn fat so stubborn. And, rather than being any single hormone or factor, it's essentially a combination of issues including adrenoceptor number (which determines the sensitivity to lipolytic stimuli), blood flow, sensitivity to insulin, what type of fat is stored, and whether the fat cells are small and numerous or large and less numerous. Of those five factors, three are under our immediate control, the last two are not. Table 1 summarizes the first three factors for the different types of body fat.

Table 1: Comparison of Characteristics of Different Fat Depots

Fat depot	Lipolytic Sensitivity	Insulin Sensitivity	Blood Flow	Ease of loss
Visceral fat	Very high	Very low	Very high	Very easy
Abdominal*	Moderate	Moderate	Moderate	Moderate
Hip/thigh	Very low	Very high	Very low	Very hard

*Recall that abdominal fat can be further subdivided into deep, superficial upper and superficial lower depots with ease of loss going easiest, medium hardest, hardest respectively.

Additionally, there is the global fact of how the body adapts to long-term dieting. Reductions in metabolic rate, thyroid, leptin, nervous system output, etc. all make it harder to reduce fat mass in general, this just compounds the problems already associated with stubborn fat.

In any case, having identified what makes stubborn fat stubborn, we can (finally) set about figuring out how to overcome the problem. Clearly finding a way to overcome the issue of a high alpha- to beta-receptor ratio is a first key. Improving blood flow into and out of the

cell is the second key. Avoiding issues with high insulin sensitivity (or simply a propensity to store fat in the first place) is the final key.

Again, nothing can be done in the short-term about the type of fat stored in stubborn fat depots; that can only be dealt with after the fat has been emptied in the first place. Fat cell number and size is outside of our control as well, I mention it only for completeness.

Dealing with the global issue of dieting adaptations is a matter of properly used refeeds, diet breaks and either supplements or drugs to fix the metabolic problems.

And now, with that out of the way, we can start moving on to more practical aspects of stubborn fat, looking at how diet, training and supplements affect the issues discussed above.

Diet, Exercise and Supplements

In the last chapter, I finally explained what, physiologically speaking, makes stubborn fat so stubborn. I finished that chapter by listing three factors that we might affect (along with two that we can't) to directly control fat metabolism: these were alpha- to beta-adrenoceptor ratios (which directly affects how well fat is mobilized from fat cells), blood flow (which is also under some control by adrenoreceptors), and the impact of insulin on inhibiting lipolysis.

As a more global factor, I mentioned the impact of long-term dieting on overall fat loss in terms of reducing metabolic rate, lowering thyroid, leptin and nervous system output, etc. This is something that we can modulate to a limited degree to help with getting rid of stubborn body fat as well.

Obviously, the factors that are within our control in regards to the above issues are diet, training, and supplements. Some might add drugs but I'm not going to address those in any detail in this book.

At least two things will come out of this chapter. First it will lay the groundwork for me to explain the specific protocols for getting rid of stubborn fat in the next two chapters. And, as promised, you'll learn why one of the most commonly used approaches to dieting (generally advocated by male coaches) is about the single worst thing a female could do to address stubborn lower body fat.

I'm making a number of assumptions about the knowledge base of my readers at this point. If you're lean enough and have dieted long enough to need the information in this book, I feel it's safe to assume that you don't a need basic course in either nutrition or training. If you don't know a carbohydrate from a protein or interval training from steady state aerobics, you have bigger problems than this book can fix.

A bit about hormones and agonists/antagonists

To understand the next section, I need to give a bit of background on hormones and define a couple of terms. Some have likened the way that hormones work as a key fitting into a lock; each lock requires a specific key and this is how hormone work. The old idea

was that each lock had one specific key but this turns out to be simplistic. Some receptors can respond to multiple hormones. For example, progesterone can bind to the androgen receptor (which normally binds testosterone).

Now, with that understanding I need to explain the concept of a receptor agonist and antagonist. A receptor agonist is any compound that activates a specific receptor, in the same way that the bodies own hormones would. Essentially, an agonist looks enough like the normal key to fit into the lock, causing stuff to happen.

In contrast, a receptor antagonist does the opposite. When it binds to the receptor in question, it either sends a negative signal or, by preventing the body's normal hormone from binding, prevents the normal signal from being sent. In either case the end result is the same: an antagonist blocks the effects of a given receptor. Stretching the key/lock analogy to its limits, an antagonist pretends to be the right key and then either sends a negative signal or simply prevents the right key from going into the lock.

So let's consider a beta-agonist compound such as ephedrine. By binding to the beta-receptor, a beta-agonist will raise blood pressure and heart rate (in addition to hopefully affecting lipolysis). Similarly, if we used a beta-receptor antagonist, we would block the effect of beta-receptors, preventing heart rate and blood pressure from rising. Drugs called beta-blockers decrease lipolysis and heart rate/blood pressure by decreasing (antagonizing) beta-receptor activity.

In a related vein, an alpha-agonist compound would activate alpha-receptors. This would slow heart rate, lower blood pressure and decrease lipolysis in fat cells. An alpha-antagonist compound (I'll describe one below) would have the opposite effect: it would tend to raise blood pressure and heart rate and increase lipolysis. If this is unclear, you can think of an alpha-antagonist as acting like a double negative. Normally alpha-2 receptors inhibit lipolysis. Well if you inhibit an inhibitor, the net result is stimulation. Got it?

Adrenoceptors one more time

Since I can't assume that you didn't just turn to this chapter without reading the previous chapters, I want to go over the whole adrenoreceptor issue one more time. Recall that fat cells and blood vessels (and most tissues in the body) have some combination of alpha- and beta-receptors.

You can essentially think of beta-receptors as accelerators of whatever system you're looking at. Agonism of beta-receptors will increase heart rate, increase blood pressure and increase lipolysis. Similarly, you can think of alpha-receptors as acting like a brake. When activated, they lower heart rate, lower blood pressure and inhibit lipolysis. From the standpoint of fat loss, beta-receptors are 'good' and alpha-receptors are 'bad'.

What this means is that there are basically two approaches to increasing lipolysis. The first is via beta-receptor agonism. Exercise does this, so do compounds like ephedrine and clenbuterol; caffeine even has mild effects. And this works just fine for fat cells that have lots of beta-receptors and not too many alpha-receptors.

But as you learned, stubborn fat often has more alpha-receptors than beta-receptors. And since the same hormones can bind to both receptor types, the end result of just trying to raise certain hormones (such as the catecholamines) may not have the intended effect. It all depends on the *ratios* of adrenoceptors as well as the levels of each hormone present.

The upshot is that, to best target stubborn fat, we need to block the effects of alpha-receptors. Alternately, we can exert such a profound hormonal effect that beta-stimulation still wins out. Of course, we can use a combination of the two, blocking alpha-receptor activity and increasing beta-receptor activity for maximum results.

A quick note of warning: recall from above that tissues like the heart have both alpha- and beta-receptors and that heart rate and blood pressure are sensitive to both. Inhibiting alpha-receptors while stimulating beta-receptors can often do absurd things to heart rate and blood pressure. You can think of it as revving the accelerator (via beta-receptor stimulation) while taking the brake off (by inhibiting alpha-receptor activity). Of course, by the time most dieters are at stubborn body fat level, blood pressure and heart rate are so low from dieting that this tends to be a non-issue. However, that doesn't mean that it can't be a problem. Combining beta-agonists with alpha-antagonists can cause problems with heart rate and blood pressure in susceptible individuals; simply be aware of this potential interaction.

Ok, so we can attack this problem from two ends: alpha-receptor antagonism or massive beta-receptor agonism. Or both. First let's look at diet and how it affects things.

Chronic (four days or more) low-carbohydrate (20% or less of total calories from carbs) diets inhibit alpha-receptors naturally. Of course, lowering carbs also reduces insulin which is beneficial in limiting the anti-lipolytic effect of insulin on stubborn fat cells. Of potentially greater benefit, low-carb diets tend to naturally increase catecholamine levels (the hormonal response to exercise is also increased), which will tend to have benefits on fat loss as well. Low-carbohydrate diets also increase the catecholamine response to other stimuli such as ephedrine, caffeine or exercise.

Empirically, people have noticed that low-carb diets seem to increase stubborn/lower body fat mobilization and the above mechanism, along with the reduction in insulin levels, is clearly part of the reason. I used this approach in my **Ultimate Diet 2.0** and it's a possibility here as well. However, as not everyone is able to do a low-carb diet for extended periods, it's not the only approach I'm going to present.

Even if dieters don't go to a strictly low-carbohydrate diet, reducing insulin by lowering total carbohydrate quantity (even if the diet is non-ketogenic) or quality (by choosing lower Glycemic Index carbs) or both will be helpful. Nobody knows for sure where the carb cutoff is to get the natural inhibition of alpha-2 receptors; that is, do carbs have to be reduced to 20% or can they be kept at a higher level while still obtaining the benefits? For that reason, I'll use 20% as the cutoff point: to naturally inhibit alpha-2 receptors, you will need to reduce carbohydrates to 20% of total calorie or less for at least four days.

However, for individuals who don't want to use a strict low-carbohydrate diet (many can't train effectively or find that it negatively affects their mood and energy levels), there is a supplement option. That option is yohimbe (or yohimbine HCL). Coming from the bark

of an African tree, yohimbe originally got an (incorrect) reputation as a testosterone enhancer but it doesn't affect hormones at all. Rather, it is a naturally occurring alpha-2 receptor antagonist.

If you're confused about the names, yohimbe is the herbal/bark-derived version, yohimbine HCL is the synthetic version. For reasons I'm going to explain below, I strongly recommend that dieters who want to use this compound obtain the synthetic yohimbine HCL and I'll refer to yohimbine from here on out. Keep in mind that all of my comments apply equally to both unless otherwise specified.

As mentioned in a previous chapter, alpha-antagonism tends to increase blood flow and yohimbine is known for having benefits for treating impotence. Back in the day, anything that gave men wood was assumed to be doing so via testosterone production and that's where the idea came from. But again, despite what it says on many supplement bottles, yohimbine does NOT affect testosterone levels.

However, yohimbine does inhibit alpha-2 receptors and studies have shown that this increases fatty acid mobilization from stubborn fat cells. Yohmbe affects not only alpha-2 receptors on fat cells but also will impact on the alpha-2 receptors in the circulation, improving blood flow. Finally, yohimbine will help keep the body's production of noradrenaline higher by inhibiting the normal feedback loop that decreases levels while dieting. All of the effects of yohimbine do nothing but help with the mobilization of stubborn body fat. Various studies have shown these effects and one recent study found that oral yohimbine decreased body fat significantly in soccer players over only 3 weeks of use.

As above, yohimbe can be found in both herbal and pharmaceutical forms (as yohimbine HCL). The herbal is not without problems, mainly side effects related to other compounds found in the bark. Chills accompanied by sweats and severe stimulation can occur with the herbal; at one time it's all that could be obtained (Twinlab herbal Yohimbe Fuel was the only product I ever trusted to contain what it claimed). Unless it's all you can get, I don't recommend the herbal product.

Yohimbine HCL is a much cleaner compound with all of the benefits and few of the side effects of the herbal. The biggest problem is that Yohimbine HCL can only be sold in 2.5 mg pills; with an effective dose of 0.2 mg/kg (14 mg for a 70 kg athlete), this means having to swallow a bunch of pills. But it's worth it to avoid the side effects.

I'd note that the effects of yohimbine are eliminated by even small amounts of insulin which means that yohimbine needs to be taken several hours from a meal or first thing in the morning. I'll come back to this in the application chapters. As well, yohimbine increases the insulin response to carbohydrate; for people who fear insulin (you know who you are), the meal consumed after yohimbine consumption should be lower in carbohydrates as well.

Note that yohimbine has a sort of passive stimulant effect. By itself it won't do a whole lot but it tends to amplify the effects of other stimulating things such as exercise, ephedrine or caffeine; I don't recommend it be taken with ephedrine (or within 4 hours of an ephedrine dose) for this reason. Some people can tolerate it but this is not universal. Taking

yohimbe before high intensity activities such as weight training or intervals can make people feel like their heart is going to come out of their chest.

I should also note that yohimbine can cause anxiety attacks in susceptible individuals, by raising brain levels of noradrenaline. Anyone with such a tendency should not use yohimbine at any dose.

Interestingly, with chronic long-term use, yohimbine builds up in tissues and the effect on fat loss seems to accelerate. It's important to note that yohimbine can also cause water retention and many dieters find that their fat loss is briefly masked due to this. Going off yohimbine for about 3 days will have you pissing like a racehorse and should leave you dry and lean. If bodybuilding is your goal, make sure you do it in time to do water manipulation stuff for your show. Anyone seeking extreme leanness on a specific date, will need to do the same for maximal visual impact.

I'd note that oral yohimbine is not perfect in that it will affect all tissues in the body. It would be better if we could isolate it to fat cells only and various approaches have been tried. Some maniacs have injected it but the more sane cutting edge approach is to try to make a topical cream with various other compounds and carriers. I remain incredulous, the skin has an unbelievably good circulatory system and most of the anecdotal reports I've seen suggest that topical yohimbine is just ending up in the systemic circulation anyhow (people still report heart rate/blood pressure increases which shouldn't occur if the effect is isolated to fat cells). In my opinion, skip the topical and stick with oral yohimbine HCL which is, at least, a known quantity.

While I'm talking about topicals, I would note that some intriguing research has occasionally suggested that these types of products can actually work for 'spot reduction' (although some of the early work may have actually simply been measuring water loss). Early studies used topical theophylline or aminophyilline although I don't think anybody ever brought it to market, another product used some type of slimming liposome that had alpha-2 receptor antagonist activity, I never saw that one commercially either.

One of the more intriguing current bits of research is a topical licorice cream that was shown to reduce thigh fat. Yes, licorice, the same stuff in the candy. As it turns out, the active ingredient in licorice, Glycyrrhetinic acid, impacts on cortisol metabolism. Remember from an earlier chapter that local cortisol metabolism (via 11-beta-HSD) appears to be very relevant for local fat cell metabolism and the active ingredient in licorice blocks 11-beta-HSD activity. Unfortunately, it seems to have become impossible to actually source the stuff and I've been unable to test it out. And, no, you can't just consume tons of licorice (in candy form or otherwise orally), as this has very negative impacts on hormone metabolism (lowering testosterone levels for one). But if you can find a topical licorice product, it may be worth investigating.

And what about exercise? For this section, I have to give a bit more background about the hormonal response to exercise. To keep things simple I'm going to divide exercise into low and high intensity and pretend that there's nothing in the middle. Low intensity activity is any steady state aerobic exercise that can be done for extended periods below the lactate threshold (LT, this is the point at which the body starts producing tremendous amounts of acid, causing fatigue; and yes I know that it's not an accurate description of what's going

on; when I write my book on endurance training, I'll deal with it in detail. Until that time, let me keep ONE concept in this book simple). Weight training or interval training (of any high intensity nature) that is done above lactate threshold will be considered high intensity activity.

Now remember from an earlier chapter that there are two catecholamine hormones, adrenaline and noradrenaline. Adrenaline is released from the adrenal gland and has to travel through the bloodstream until it gets to a specific tissue where it exerts its effects. In contrast, noradrenaline is released from nerve terminals and generally has only local effects (noradrenaline can often 'spill out' into the general circulation if there's enough of it). Both can bind to adrenoreceptors and exercise is the primary way of achieving the beta-receptor activation I talked about above. Compounds such as ephedrine, clenbuterol or other stimulants do the same thing.

As it turns out, during low intensity activity the primary hormone released is noradrenaline, with levels increasing as intensity increases. Now noradrenaline does a plenty fine job of stimulating lipolysis for non-stubborn fat cells where there aren't too many more alpha-receptors than beta-receptors. But in stubborn fat, with their preponderance of alpha-receptors, the low levels of noradrenaline can end up exerting an anti-lipolytic effect, inhibiting fat release from the fat cell. Don't misread me, this doesn't mean that low intensity activity is useless (the interval freaks reading this were about to draw that very conclusion), just that it has to be used correctly. I'll tell you how in the next chapter.

Now, as exercise intensity goes up, noradrenaline levels continue to increase. Adrenaline output gradually increases with increasing intensity as well. Until you cross the lactate threshold, at which point both adrenaline and noradrenaline output increase exponentially.

So you're walking, you're walking, you're walking and your body is just punting out noradrenaline from the nerve terminals. Small amounts of adrenaline are being released as well. Then you break into an all-out run and both adrenaline and noradrenaline get pumped out like crazy.

This has a number of effects in the body. One is to increase the liver's production of glucose (often blood glucose goes up during high intensity activity because the liver makes it faster than tissues can take it up). Another is that heart rate and blood pressure go through the roof. Skeletal muscle also starts using tons of glucose for fuel, producing the acid that causes burning and fatigue.

Of more importance, high intensity activity seems to be able to overcome the lipolytic insensitivity associated with stubborn fat. This has to do with differences in how the different hormones bind to alpha and beta-receptors. Essentially high intensity work can overcome some of the problems associated with alpha-receptor dominance in stubborn body fat. Aha, scream the interval freaks, we knew we were right all along: intervals rule and steady state drools.

Hold on, I'm not finished.

While the hormonal response to interval training does some nice things as far as breaking down fatty acids in the fat cell is concerned, there is also a downside. At high intensities of exercise, the body doesn't burn fatty acids for fuel, preferring to use glucose instead. Additionally, high levels of lactate (released into the bloodstream from exercising muscle) traps fatty acids in the fat cells. As it turns out that there is a rather large release of fatty acids into the bloodstream about 5 minutes after high intensity activity ends and the lactate induced block is released. You'll see how this factors into the protocols in Chapter 10.

So all is not perfect in interval lover's land in this regards. But, you say, the afterburn/post-exercise calorie burn effect will take care of it. Nope, remember that stubborn body fat has poor blood flow and will happily re-esterify those now mobilized fatty acids. The end result is no result because all of the fatty acids that were mobilized (but not burned off) during the exercise bout simply get stored again.

So even if high intensity activity better mobilizes the fatty acids (and it does), it doesn't magically solve all of the problems. You may have an inkling of how to solve them based on what I've written above, or you may not. Better odds are you saw someone present my original stubborn fat protocol and know what my original solution was. But I'm getting ahead of myself. Let me finish up this section before I move onto the next topic of adipose tissue blood flow.

On a related note, research has shown that doing exercise in a mode that you don't usually use increases the hormonal response. So if you always use the EFX, you'll get a greater adrenaline/noradrenaline response if you use the Stairmaster or Treadmill or whatever. I'll come back to this when I talk about application and how to optimize the stubborn fat protocol.

Now you know basically how adrenoceptor function is mediated by diet, training and supplements. We can inhibit alpha-receptors naturally with either a very low carbohydrate diet or by using oral yohimbine/yohimbe. We can increase beta-receptor activation several different ways. Low-carbohydrate diets will do this naturally to some degree, compounds such as caffeine and ephedrine can do it. And exercise is arguably the most potent weapon in our arsenal to accomplish the goal of beta-receptor stimulation.

Adipose tissue blood flow (ATBF)

Having figured out how to mobilize fatty acids from stubborn fat cells, the next step to address is ATBF and how to increase it.

As it turns out, much of what I discussed in the previous section holds for blood flow as well since ATBF is controlled by the same adrenoceptors that control lipolysis.

Inhibiting alpha-receptors can only increase ATBF and it's been shown that blood flow increases with long-term fasting. While fasting is inappropriate for a lean dieter, low-carbohydrate diets mimic fasting and may have the same impact on ATBF.

If a low-carbohydrate diet isn't workable, oral yohimbine will have similar effects on blood flow as it does on lipolysis, inhibiting alpha-receptors. Again, chronic use will cause a tissue buildup and have increasingly greater effects.

Even aerobic activity has been shown to overcome the sluggish blood flow of stubborn fat although, based on hormonal considerations, I'd expect high intensity work to have a greater impact. Just keep in mind the negatives I mentioned above in how lactate inhibits fatty acid release and how fat oxidation is blunted at high intensities.

As I mentioned previously, temperature seems to impact on ABTF as well, it's conceivable that wearing those silly rubber wraps could have an impact. You certainly wouldn't want to try to attack stubborn fat in the cold. I'd note that duration of exercise also impacts ATBF although it takes something like 4 hours before any major effect is seen. Leave that to the endurance guys.

As I mentioned previously, most other approaches to improving ATBF are hamstrung by the fact that the same compounds that increase ABTF often inhibit lipolysis. Adenosine (released from fat cells), nitric oxide and insulin all increase ATBF but inhibit lipolysis, making them essentially unworkable.

Insulin levels

There's not much to say about insulin that I haven't touched on already in this chapter. Reducing insulin will mainly be a function of reducing carbohydrate quantity (amount), quality (type), or both. As I mentioned, there are different effects of reducing quantity versus quality in terms of insulin resistance and fatty acid release. Reducing quantity tends to cause insulin resistance (again, a good thing on a diet) and increases fatty acid release, keeping total carbohydrate intake high but choosing lower GI carbs has neither effect. Choosing different quality carbs (in this case, less insulinogenic carbs) while reducing carbohydrates also impacts on this.

The main thing to keep in mind is that insulin should be lowered prior to attacking stubborn body fat. Which doesn't meant that a low-carbohydrate diet is the default. Again, there are several possible options.

One is to do the protocols I'm going to describe in the next chapter first thing in the morning before eating. Now, fasted cardio is an area of huge debate; under most conditions I don't think it makes an iota of difference when cardio is done. Stubborn fat is one of the places where it might.

I'd note that some of the people who originally tested my first stubborn fat protocol did it without changing their diet, with no supplements, and not fasted and it still worked. I'm just awesome that way.

However, going into the protocol with insulin lowered to some degree is not a bad idea. Again, this could mean doing it fasted first thing in the morning. If that's not possible or preferred, it could be done within the context of a very low-carbohydrate diet (which keeps insulin low all day). If that's not workable and the dieter is on a carb-based diet, the

protocol can be done at least 3 hours after the previous meal. This will allow insulin to have gone down somewhat from whatever carbohydrates were consumed.

Finally I'd note that even low intensity aerobic activity lowers insulin rapidly. Even performing an easy 5-10 minute warm-up prior to any other activity will lower insulin, removing the normal anti-lipolytic block that would otherwise be in place.

A few other dietary comments

Although this doesn't relate specifically to the mobilization or transport of stubborn body fat, I'd note that diet can impact on how well or poorly fat is oxidized by skeletal muscle. Lowering carbohydrate intake, especially in conjunction with exercise that depletes muscle glycogen, is known to enhance whole body fat oxidation. This is yet another potential benefit of reducing carbohydrate when attacking stubborn body fat.

In practice, most dieters at that level of leanness will already be reducing carbohydrates to some degree or another. Since training will tend to deplete muscle glycogen, it may not be necessary to address this issue explicitly. However, adding some type of higher rep glycogen depleting exercise (e.g. high rep weight training) to the normal diet of heavy work (to maintain muscle mass) may be beneficial for ensuring that mobilized fat from stubborn body fat is burned more effectively. My **Ultimate Diet 2.0** goes into more detail on this.

Again, while this doesn't necessarily impact directly on stubborn fat mobilization, transport, or burning, there may be a potential benefit to attacking the fat storage issue. This is especially true for women. Given the issues I've addressed in previous chapters, and the possibility that women's bodies may find a way to mobilize fat from the upper body while storing it in the lower body, ensuring that fat isn't being stored following meals is a worthwhile strategy.

This brings us back to the MCTs and DGs I discussed early in this book. Replacing a majority of the dietary fat you're eating (while ensuring daily fish oil intake) could be beneficial in ensuring that ingested dietary fat is being burned in the liver with no chance of storage in lower body fat cells. It also may make no impact at all except to make your diet that much more bland and unpleasant.

In the last chapter, I mentioned briefly the global issues involved with dieting that can negatively impact on stubborn fat loss indirectly. This has to do with what happens to leptin, thyroid, etc. during a long-term diet. If you've read any of my last books, you know about free meals, refeeds and diet breaks. In this context, refeeds (lasting from 5-24 hours) and full diet breaks (lasting 10-14 days) are the most relevant.

Empirically, I've often noticed that stubborn fat mobilization seems to be better (I'll explain how I 'know' this in the next chapter) the day after a reefed (a period of high-carbohydrate over-feeding). So after a 5-hour refeed on a Wednesday night, stubborn fat mobilization seemed better during stubborn fat protocols done the next morning. Perhaps raising leptin helps with fat mobilization. Perhaps pushing some water into the fat cells increases their volume (remember that fat cell size impacts on metabolism). Perhaps I'm

totally full of shit on this point and hallucinated it. I accept all of these as possibilities. But it is worth trying when fat loss stalls, inserting a reefed and following it the next day with one of the 4 stubborn fat protocols may be helpful in shifting the last bit of stubborn fat.

And then there are diet breaks. As I've said before, I think most people diet too long without a break. Fat loss slows, they overtrain, metabolism crashes completely and they end up banging their head against the wall for no gains (more accurately, no fat *losses*). Better at that point to come back to maintenance calorie levels, bring carbs to 100-150 g/day minimum, cut training back to allow recovery to occur, and do that for 10-14 days before continuing the assault on stubborn body fat. You would have to plan it into your dieting period of course but it works better, trust me.

Boys have a penis, girls have a vagina

Other than the obvious issue relating to hormones and fat patterning, for the most part I've treated men and women similarly throughout this book. But I told you up front that one of the things you'd find out by this point was why one of the strategies that is often advocated by male coaches (and which works fine for them) ends up being absolutely the worst thing a female can do to get rid of stubborn body fat.

Of course, these same male coaches are usually still stuck in 1982 blaming estrogen and thinking that Nolvadex (an anti-estrogen drug) will fix all the problems. And when their girl's legs don't come in, they just throw more drugs at the problem. And it still doesn't work.

Remember from previous chapters that, no matter how you cut it, men's abdominal fat is never ever ever as stubborn as women's lower body fat. It's more sensitive to lipolytic stimuli and less sensitive to insulin, it responds well enough to normal cardio approaches such that nothing special is usually needed to get rid of it except patience.

Sure, low back fat can be a bear and with striated glutes becoming required to win shows in natural bodybuilding, men seeking extreme leanness now have to deal with that issue too. Just throw more clen, thyroid and GH at it. Men usually tolerate higher carbohydrates better than woman (for reasons I don't want to get in to). That's on top of all of the other natural hormonal differences.

At the professional level (and many bodybuilding coaches are pro-bodybuilders themselves), the inherent advantages that male dieters have are further accentuated by the drugs that are being used. Simply throwing the same drugs at women and expecting them to have the same effect is silly and doesn't always work.

What this means is that a male can often get contest ripped eating lots of carbohydrates and doing nothing more esoteric than fasted morning cardio. Women, generally speaking, can't.

Hopefully by this point in the book you have an idea why. Women's lower body fat is profoundly sensitive to insulin meaning that too many carbohydrates will make it damn

near impossible to mobilize stubborn body fat in the first place. Add to that what I discussed about the hormonal response to very low intensity steady state cardio, the small noradenaline response can end up having an anti-lipolytic effect.

Even done fasted, if the diet is chronically carb-based, with nothing but low intensity cardio, lower body fat will never come off. And no amount of anti-estrogens or other compounds will fix the problem. Even adding oral yohimbine would do most of the work but most coaches seem to be unaware of that compound. I guess none of them read **BodyOpus**.

So you end up with male bodybuilders, who have no problems getting contest lean on carb based diets and low intensity morning cardio giving women the same advice. And it doesn't work. In fact, it's about the single worst thing that could be done.

Between the extreme sensitivity of lower body fat to insulin, coupled with the fact that low-intensity exercise can end up having an anti-lipolytic stimuli, coupled with an odd phenomenon whereby women may be able to redistribute fat from their upper to lower bodies (or simply store incoming dietary fat in lower body fat without remobilizing them), not only don't the legs lean out with the typical male approach, sometimes women's legs actually can get fatter.

Summary

This chapter is too involved to summarize. Read it again and pretend you're doing bullet points and call that the summary. It's finally time to get to the protocols themselves.

The Stubborn Fat Protocols: Introduction

We're almost there. You've made it through over 50 pages of detailed information about fat cell metabolism and physiology, you know more about fat cells than you thought there was to know (or ever wanted to know). And I'm still not going to give you the protocols. Rather, in this chapter I want to cover some introductory information that is crucial to what I'm going to talk about in Chapter 10. Please don't skip it, it's important.

Four protocols for four situations

In the next chapter, I'm (finally) going to present four distinct protocols that integrate all of the information from the previous chapters (especially Chapter 8). Although I'm going to present them in order from least to most complex, I want to make it very clear that they aren't being presented in any order of importance or as a progression that you should follow. We are all unique snowflakes (just like mommy said) and each reader's individual needs and situation will determine which protocol is most appropriate.

Although all of the protocols I'm going to present work well, depending on the context, I do have a special affinity for the Stubborn Fat Protocol 2.0 (SFP2.0) for one big reason. The stubborn fat issue has been a topic I've pursued for over 10 years now and this book and the SFP2.0 represent the culmination of that work.

I've accumulated stacks of research for over a decade that have gone into writing this project; I've been carrying them with me in a big box while adding to them as new research came out. I've tweaked, played and tested various ideas and kept my eye out for any bit of data that could fix the problem once and for all. Once I present the newly developed SFP2.0, I feel that I'll have said everything I have to say about the topic and I can move onto the next project that will consume my every waking thought.

In the same way that I'm not presenting the individual protocols as a sequence or in order of importance, nor should they seen as mutually exclusive of one another. Some of them can be used together. As you'll see, some of them *should* be used together for optimal results.

The more advanced protocols (including the original SFP) are not appropriate to use daily which leads me into one bit of ranting that I want to get out of the way before continuing this chapter.

The obligatory warning

It's not an over-exaggeration to say that individuals trying to strip off that last bit of fat came become a bit obsessed. A little less kindly: most of them lose their freaking mind towards the end of the diet.

It's easy enough to overtrain on a diet, most people in my experience train too hard too often without taking a break. They destroy themselves and, as often as not, they still don't get to where they want to be.

Now, a problem with modern dieting is that, for reasons ranging from reasonable to completely wrong, old school steady state cardio has gotten a bad rap. Never mind that four decades of bodybuilders got contest lean with nothing but low-intensity cardio; many have been convinced that steady state is either worthless (or in some cases actively detrimental) to fat loss. This is sheer nonsense, of course; I'm simply reporting what I'm seeing others advocate or attempt to do in practice. Clearly I disagree completely.

The fact is, right or wrong, most people who are trying to eliminate the last bit of stubborn fat (whether for a contest or another goal) do some type of training nearly daily when they are contest dieting; many train more than once daily.

This would be fine if they'd stay sane and keep the intensity of their training under control. Unfortunately, many don't. If someone has been convinced that low-intensity work is useless, and they want to do metabolic work every day, where exactly does that leave them? It leaves them trying to handle a training load that no human being eating a lot of calories could recover from, and they try to do it while dieting. I'd note, and I'll try to do so without being ugly about it, that some contest prep coaches are advocating this stupidity. Dieters are getting destroyed.

In addition, there's this weird mentality by which people think that their training volume and frequency should go UP when dieting; this is backwards as recovery is always hampered when calories are restricted. Realistically, this idea came about in the 80's when bodybuilders started really using anabolics to help with bodybuilding contest prep. This had the effect of artificially elevating recovery ability and allowed for insane levels training; it also gave naturals a false idea of what they could or should do. Without drugs, this can't work and natural dieters simply get nuked. They overtrain, lose muscle mass and end up giving up before reaching their goals.

I'm bringing this up here because both the SFP1.0 and 2.0 are very intense; dieters who lose sight of that can get into real problems. I've heard some crazy stories, at least one of which I'll describe later. You can't do the protocols every day. You can't do them even every other day under most circumstances. At best, the SFP1.0/2.0 can be done twice weekly and the occasional freak of nature might get away with it three times weekly.

I want to make it clear that, despite my little joke above about you being a unique snowflake, you are not the exception to this. You are not the person who can handle high intensity interval training daily without ruining yourself. Especially if you're doing it while resistance training your legs heavy twice/week (as many do). Something will give: it might be your knees and it might be your mind. But you can't do it and I encourage you not to try. I won't repeat myself, this should suffice. Simply realize that if you ignore my warnings, you're setting yourself up for some real problems.

Ok, back to the book

Hopefully you realize that trying to do the higher intensity protocols described in the next chapter every day won't work. If not, please read the previous bit again. Yet, as I noted, most extreme dieters will be doing some type of metabolic work daily (or more often) trying to strip off that last bit of fat. Where does that leave you?

The seemingly obvious answer is to combine the protocols. If the SFP1.0/2.0 are appropriate for you, they can be done perhaps twice per week (again, the occasional freak might do them three times per week but this is the exception, not the rule). On the other days, do the less intensive protocols. Clear?

In addition to my comments above on general approach, as I detail each protocol, not only will I tie it in with previous information presented in this book, I'll also try to address how a given protocol might integrate relatively better or worse with other aspects of your training or diet.

That is to say, I don't know what kind of training you are doing or intend to do during your diet. It might be a body part split where you hit everything heavy once/week. Or it might be a three-day per week full body ordeal that's all heavy. Or all metabolic type work, or some combination of heavy and metabolic work.

Perhaps you're an athlete trying to get rid of the last bit of fat for performance reasons and your training mandates that you must be doing a certain amount or type of training on a certain day which limits what you can and can't do. Since I'm not giving specific recommendations for the rest of your diet or training, I can only tell you how a given protocol will or will not work for a given set of situations.

You'll have to make some judgment calls yourself here. When in doubt, err on the side of conservatism, doing a little bit less for longer usually works better than trying to do too much and blowing up. If you really can't figure it out, email me or go to my support forum at:

http://forums.lylemcdonald.com

The phantom tingle

Before getting to the protocols themselves, there's one thing I want to mention first. In the last chapter, I made this vaguely nonsensical comment about how I 'felt' that stubborn

fat was better targeted the morning following a 5 hour high-carbohydrate reefed and I want to expand on what I observed.

This may also give you a little bit more of an objective way of determining if the following protocols are doing anything productive or not. Of course, drops in your skinfolds or visible fat loss is still the ultimate metric. Just realize that, between issues inherent to dieting such as water retention, a weird lag in fat loss that often occurs (see the end of this chapter for more information) and some other issues, skinfolds don't always drop as expected; this tends to be especially pronounced in women.

This doesn't necessarily mean that fat loss isn't happening, although it *can* mean that. At the same time, don't wait for weeks with no change before changing some aspect of your training or diet, otherwise folks with a time limit can run out of time if they aren't careful.

In any case, what I felt on those mornings following a refeed was something I call the phantom tingle. During various approaches to targeting stubborn body fat, many people (not all, but many) will note a slight tingling or itching on or under the skin where their stubborn fat is. So men will get it on their low back or lower abs, women will get it in their hips or thighs.

Invariably this comes along with the body mobilizing stubborn body fat and appearance improves and skinfolds drop soon thereafter. This is what I always noticed in a pronounced fashion the morning after a reefed. I'd take my caffeine and yohimbe and head to the gym for the treadmill and, without fail, a few minutes in, my low abs would start itching like crazy. Skinfold drops came quickly thereafter.

I'm not 100% sure what causes this, my guess is that improved blood flow during the various protocols is causing the itching/tingling effect. Maybe a histamine response of some sort. I simply don't know.

All I can tell you is to watch for the effect. Again, not everybody gets it and the absence of the phantom tingle is not an automatic indication that the protocols aren't working. However, if you *are* getting the phantom tingling during the following protocols, that almost ensures that what you want to happen is actually happening.

Of whooshes and squishy fat

Before you freak out and think you've entered some weird Internet forum where people talk about stalls and whooshes, please bear with me; there's actually some physiological rationale to what I'm going to discuss.

Many people have noted that fat loss is often discontinuous, that is it often happens in stops and starts. So you'll be dieting and dieting and doing everything correctly with nothing to show for it. Then, boom, almost overnight, you drop 4 pounds and look leaner.

What's going on? Back during my college days, one of my professors threw out the idea that after fat cells had been emptied of stored triglyceride, they would temporarily refill

with water (glycerol attracts water, which might be part of the mechanism). So there would be no immediate change in size, body weight or appearance. Then, after some time frame, the water would get dropped, the fat cells would shrink. A weird way of looking at it might be that the fat loss suddenly becomes 'apparent'. That is, the fat was emptied and burned off days or weeks ago but until the water is dropped, nothing appears to have happened.

For nearly 20 years I looked for research to support this, I was never sure if it was based on something from the 50's or he just pulled it out of thin air as an explanation. Recently, one paper did suggest that visceral fat can fill up with water after massive weight loss but that's about it.

Somewhat circumstantially, people using bioimpedance body fat scales (which use hydration to estimate body fat levels) have noted that body fat appears to go up right before a big drop. This implicates water balance as the issue here.

As well, women, who have more problems with water retention, seem to have bigger issues with stalls and whooshes than men. Further, some individuals who have done dry carb-loads (high carbohydrate refeeds without drinking a lot of water) have seen them occur; presumably the body pulls water into the muscles and out of other tissues (fat cells). In lean individuals, appearance is often drastically improved with this approach, it doesn't do much for those carrying a lot of fat.

I'd note that dry carb-loads suck because you're so damn thirsty. Interestingly, even normal refeeds often work in this regards, perhaps the hormonal effect 'tells' the body to chill out and release some water. So not only do refeeds seem to improve stubborn fat mobilization the next day (as discussed above), they may help the body drop some water so that you can see what is happening.

Finally, many have reported whooshes following an evening which included alcohol. A mild diuretic, this would also tend to implicate water balance issues in the whoosh phenomenon.

I'd also note that this isn't universal, lean dieters often see visual improvements on a day to day basis; a lot seems to depend on whether or not they tend to retain water in general. Folks who do have problems with water retention tend to have stalls and whooshes, those who don't show nice consistent visual changes.

On a related topic, I wanted to discuss something else that often happens when people are getting very lean and dealing with stubborn body fat: the fat gets squishy, feeling almost like there are small marbles under the skin. Yes, very scientific, I know. That's the best I can do.

As folks get very lean, down to the last pounds of fat, the skin and fat cells that are left will often change appearance and texture. It will look dimply (as the fat cells which are supporting the skin shrink and the skin isn't supported) and feel squishy to the touch. This is bad in that it looks really weird, but it's good because it means that the fat is going away. I have nothing truly profound to say about this topic, just realizes that it happens and usually indicates good things are happening.

A final introductory topic: Rating of Perceived Exertion (RPE)

I'm assuming in this book that you know the difference between weight training, steady state cardio and interval training. If not, this probably isn't the right book for you. However, there's a concept I'm going to use in Chapter 10 that I can't be sure that everyone will be familiar with and it's faster for me to explain it up front than to keep repeating myself. Don't worry, this is the last introductory bit before I finally get to the protocols themselves.

Most exercise prescription is done using heart rate and there are reasons (good and bad) to do it that way. But long, long ago, a man named Borg decided to come up with a scale of rating exercise that didn't use HR. The Borg scale (or more commonly 'rating of perceived exertion' or RPE) originally ran from 6-20 for reasons I won't bother explaining. A Borg rating of 6 was basically rest, 20 was maximal exercise. Studies repeatedly showed that, once people got used to it, using RPE was just as accurate as using heart rate.

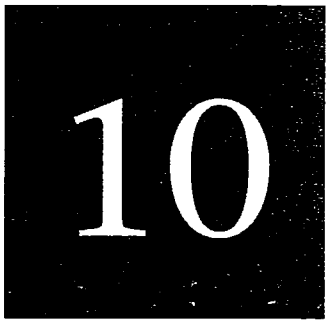
Since people have trouble understanding a scale that doesn't start at 1, this was later amended to a 10-point scale. As with the original, 1 is essentially sitting on your couch. I'd liken a 10 to the time you had to sprint down the airport terminal to make your flight and thought you were going to die. Most exercise falls between those two extremes.

Under certain circumstances, and this includes three of the four stubborn fat protocols, heart rate becomes less accurate for rating intensity. Instead, I'm going to give RPE recommendations for you to use. Towards that end I've reproduced the 10-point RPE scale with descriptions in Table 1 below.

Table 1: RPE vs. Effort

RPE	Effort level
0	Nothing at all
0.5	Extremely weak
1	Very weak
2	Weak (light)
3	Moderate
4	
5	Strong (heavy)
6	
7	Very strong
8	
9	
10	Extremely strong (maximum)

Typical low intensity steady state cardio would be around a 3-4 RPE. Working at lactate threshold (the highest intensity you can perform continuously for 20 minutes or so) is a 5-6 RPE. Longer intervals of 30-60 seconds are typically done at a 7-8 and shorter intervals of 15-30 seconds) can be up at a 9-10 RPE.



The Protocols

After the last chapter, I'm sure you're really ready for me to present the protocols and we're almost there. I want to reiterate that the four protocols are not being presented in any order of importance; nor do I want you to think that you need to work through them sequentially. I am going to start with the 'simplest' approach and move towards the most labor (and effort) intensive as I go.

As you'll see, the protocols are also not meant to be mutually exclusive; that is, you don't have to only pick one to perform. Rather, and I'll detail this below, the protocols can (and often **should**) be mixed throughout the week. This is especially true for the SFP1.0 and SFP2.0. This is for the reasons I discussed last chapter (having to do with the potential for overtraining on a diet) along with more issues I'll discuss here.

For each entry, I'll discuss the protocol itself, explain how it ties in with the information in the previous chapters, and talk about any other issues such as diet or how to implement it with your other training.

Protocol 1: Low-carbohydrate diet plus low intensity aerobic activity

So let's start at the simplest end of things. As discussed last chapter, low-carbohydrate diets accomplish some nifty things in terms of inhibiting alpha-2 adrenoceptors and increasing adipose tissue blood flow. Reducing carbs to 20% or less of total calories (so on 1800 calories, 20% is 360 calories or 90 grams per day) will allow the good stuff to happen.

That alone can be coupled with basic steady state cardio which can be done fasted or not. Ideally, the cardio would be done after consumption of some caffeine (100-200 mg) consumed 30-60 minutes before to help jack up catecholamine response a bit. Throwing in a bit of L-tyrosine (1-3 grams) will kick things up a bit too.

As far as duration, 45-60 minutes per session is plenty; if you need to do more than that, you should split the sessions up. Intensity should be low to moderate, maybe a heart rate of 130-140 (this is the one protocol where heart rate should be fairly accurate) or an RPE of 3-4. The old talk test holds here, if you can hold a broken conversation during your cardio, it should be about the right intensity. It'll be brisk without being so intense that

folks start getting anaerobic and either losing muscle or causing other problems with recovery. It should allow for a decent calorie burn although it won't be massive. But most of what's burned should come from stubborn fat so that's ok too.

There's not a whole lot more to say about this approach to stubborn fat loss. Protocol 1 can easily be used daily and will fit safely into just about any training program anybody could come up with. The exception would be folks who go absolutely nuts with volume; you shouldn't need three hours per day of low intensity cardio to get super lean in my opinion. Protocol 1 fits well with the more advanced protocols as well and can be used on the days when you can't do the more advanced stuff.

The biggest drawback of Protocol 1 is that it requires that a very low-carbohydrate diet be used. Many individuals can't train effectively on very low carbs. They either feel terrible mentally, or get depressed (probably from low serotonin), or have a constant headache or what have you. I'd note that ensuring sufficient mineral intake (sodium, potassium, magnesium, calcium) can help with fatigue and cramping but sometimes it just isn't enough. In which case another protocol should be used.

Of course, performance athletes doing high intensities or volumes of training generally can't follow a low-carbohydrate diet (there are exceptions depending on the sport) because it kills their ability to train effectively. Protocol 1 would be unworkable in that situation.

But for individuals who handle very low-carbs well and/or can't fit in the more intense protocols and/or can't or won't use yohimbine, this approach is plenty workable. The very low-carb intake will inhibit alpha-2 receptors, the cardio will then mobilize transport and oxidize the fatty acids. Of course there has to be a reasonable caloric deficit for any fat loss to occur; don't think the protocol will magically get it done if you're eating too much.

Another dietary option worth considering (along with the low-carbohydrate intake), especially for those women who seem to store thigh fat as their upper bodies lean out, is to switch out most or all of your standard fat intake for either MCTs or DGs. That's along with your daily fish oil intake of course. MCT/DG will be preferentially burned after meals preventing any odd storage of dietary fat in lower body fat cells.

Protocol 2: Oral yohimbine plus low intensity aerobic activity

As noted above, not everyone can or will follow a low-carbohydrate diet. At the same time, the higher intensities of the upcoming stubborn fat protocols 1.0 and 2.0 may not be workable either. They may simply need something to do on the days they aren't doing SFP 1.0 or 2.0.

This brings us to Protocol 2 which is to use oral yohimbine at a dose of 0.2 mg/kg (take your bodyweight in pounds and divide by 2.2 to get kg, then multiply by 0.2 to get your yohimbine dose in milligrams). So a 155 lb. athlete (70kg) would consume 14 mg of yohimbine per dose. Although many recommend splitting the yohimbine dose throughout the day, this is not the best way to take it for stubborn fat mobilization; the entire dose should be taken all at once, ideally with caffeine (100-200 mg depending on body weight) 30-60 minutes before low intensity aerobic activity. Again, L-tyrosine (1-3

grams) could be added as well. With this approach, the yohimbine inhibits the alpha-2 receptors, caffeine and/or tyrosine helps jack up catecholamine output and the low intensity aerobic activity does the rest.

I'd note that while I'm talking about this approach as if it's on a carb-based diet, it will also work fine (if not a bit better) coupled with low-carbs. It's just that a strict low-carbohydrate diet isn't *mandatory* when oral yohimbine is brought into the equation.

I'd remind readers that even small amounts of insulin shut down yohimbine's effects pretty much completely. The yohimbine/caffeine/cardio combination should either be done fasted first thing in the morning or 3-4 hours after a meal (that should ideally contain either smaller amounts of carbs, very low glycemic carbs or both).

Similar to the first protocol, Protocol 2 is more or less appropriate for everyone and can be used daily if needed or desired. Keep in mind that yohimbine does have some stimulant effects which users should be aware of. Folks who do their cardio at night (either because they are doing double sessions or that's the only time they can do it) need to be aware that caffeine/yohimbine can cause problems with falling asleep. Try to take the combination at least 4 hours before habitual bedtime and you should be ok.

It would also be best to start with a half-dose of yohimbine for the first few times through the protocol to asses your tolerance. Recall from a previous chapter that yohimbine can cause anxiety attacks in predisposed individuals.

Athletes who can't indiscriminately add high intensity work to an already high training load can use Protocol 2 on off days or as an additional low-intensity workout (the aerobic work should have an active recovery effect to some degree). Of course, readers who choose to use the SFP 1.0 or 2.0 can use Protocol 2 on the days that they don't do the higher intensity protocols to avoid destroying their legs.

The same comments about duration and intensity of exercise from Protocol 1 apply here. A duration of 45 to 60 minutes at a low to moderate intensity (3-4 RPE) is appropriate. Do note that yohimbine (especially if combined with caffeine and/or tyrosine) can falsely elevate heart rate during activity which makes heart rate misleading. Hence my preference for RPE here.

Alternately, you can simply work at the aerobic intensity that would have generated a 130-140 HR prior to the yohimbe combination. That is, say that walking at 4mph and 1% grade normally puts you at a 135 HR. But on yohimbine it takes you to 145-150. You wouldn't need to slow down or eliminate the incline to bring HR back down, the yohimbine is simply giving you a falsely elevated value.

Protocol 3: The original Stubborn Fat Protocol (SFP 1.0)

Some of you may be familiar with the original SFP (now known as SFP1.0 to distinguish it from the final approach). I want to give credit where credit is due here, much of the idea for this one came out of involved conversations with a close friend, Elzi Volk. A true nerd

when it comes to adrenoceptors and yohimbine, she was actually the first female bodybuilder I prepped, and we used oral yohimbine with steady state cardio and the Bodyopus diet to get her ready for her show.

This got her fascinated in the topic of stubborn fat, alpha-receptors and much of what I've discussed in this book. You can still find her articles (a bit technical) on the web about yohimbine and adrenoceptors. In any case, I want to give her credit for helping me to develop the original SFP1.0.

The basis of the SFP1.0 comes out of the discussion of exercise from the previous chapter. Recall from there that high intensity activity (above lactate threshold) can overcome the lipolytic resistance inherent to stubborn fat cells due to the pronounced hormonal response.

While wonderful, this is counterbalanced by the fact that high levels of lactic acid in the blood trap fat in the fat cells and fat is not oxidized well during high intensity activity. But remember how I mentioned that a few minutes after high intensity activity ceases, there is a big release of fatty acids into the bloodstream? Maybe you can see where I'm going with this. By taking a short-break after the intervals and then doing steady state cardio, we can now burn off the mobilized (and more importantly, released) fatty acids.

The original SFP as outlined was the following. I'll comment on it further below

The Original Stubborn Fat Protocol (SFP1.0)

1. Done first thing in the morning fasted.
2. 30 minutes beforehand: consume 200 mg caffeine, 0.2 mg/kg yohimbine, and 1-3 grams L-tyrosine. NO ephedrine.
3. 5-10 minute easy warmup.
4. 10 minutes of interval training. This could be anything from 10X30 seconds on/30 seconds off up to 5X1 minute on/1 minute off. Ideally this would be done on a machine that the dieter doesn't usually use to increase the catecholamine output. These aren't all out but are done at an RPE of perhaps 7-8 so that the workout can actually be completed and fatigue isn't too monstrous.
5. Rest completely for 5 minutes to allow fatty acids to be released into the bloodstream.
6. Perform 20-40 minutes of aerobic activity, ideally near the lactate threshold as this is where fat oxidation tends to be maximized; in practice most did this at a lower intensity to spare their legs.
7. Wait an hour to eat following the protocol.

Now, some of you may have seen this protocol (in one fashion or another) before. I originally presented it publicly (I honestly don't recall where I first posted about it) a number of years ago after I had first developed it. Since that time, it's taken on sort of a life of its own, I know of several online trainers who are using it to prepare their clients.

Unfortunately, people don't realize that I have made major modifications to the original protocol and it's still being presented as described above many places. Therefore, I want to look at it point by point so that you can see how (and more importantly *why*) I modified the original protocol in the first place.

Originally the protocol was meant to be done fasted to ensure that insulin was low. Given that the low intensity warm-up will lower insulin, this turns out not to be necessary. Many test subjects did the SFP1.0 after having eaten and it worked fine.

The caffeine and yohimbine should be obvious. As mentioned, L-tyrosine is an amino acid involved in the synthesis of the catecholamines, using it helps to ensure maximal hormonal response. I disallowed ephedrine as it's not always a good idea to combine it with yohimbine; additionally one study showed that ephedrine blunted the adrenaline response to aerobic activity.

The timing of the yohimbine/caffeine was actually pretty specific. Since yohimbine before high intensity activity often makes people feel like they are going to die, I was trying to get the yohimbine into the system shortly after the intervals were done. I'd note that many test subjects often did the full protocol without any of the supplements, no caffeine, no yohimbine, no tyrosine. It still worked fine.

The length of the intervals was a subject of some debate. On the one hand, longer intervals will deplete more glycogen and tend to cause a greater metabolic perturbation. However, they tend to be hard as hell with a short rest and generate a ton of lactate, possibly interfering with fat release when the intervals are over.

Some folks experimented with shorter intervals, stuff like 10X15 seconds at near maximum with a 45 second rest. This seemed to work just as effectively as the same hormonal response was accomplished without generating too much fatigue or lactate.

Some also used interval style weight training prior to the steady state with seemingly good results. Studies have shown that weight training can generate a lipolytic response if the loading parameters are right and some of the high rep/short rest training that is popular now can probably substitute for formal interval training.

The intensity of the steady state bit was also up to debate. As noted above, fat oxidation is probably maximized near lactate threshold but this tends to be too high of an intensity for most dieters. There's simply too much fatigue, too much high threshold fiber recruitment, too much potential for muscle loss. So the intensity was scaled back to the 130-140 heart rate range or a 3-4 RPE.

The final bit about waiting an hour to eat was based on the (very stupid) idea that maximal fat loss would occur by allowing the body to burn off excess fatty acids with the post-exercise calorie burn. Essentially, I didn't want the dieter raising insulin and causing fatty acid re-esterification. As it turns out, the body keeps burning fat for fuel after high intensity training even in the presence of insulin so there was no reason to wait to eat. At the very least dieters should have some protein, but a normal meal was acceptable too.

I'd note that the SFP1.0, while it might theoretically work better on a low-carb diet seems to work equally effectively on a carb-based diet. That's with or without the yohimbine, caffeine and/or tyrosine. Simply, the high intensity part at the beginning overcomes the alpha-2 adrenoceptor inhibition of lipolysis, the break gives the fatty acids time to appear in the bloodstream, and the steady state cardio burns it off.

I'd note that a study came out after I'd developed the original SFP1.0 showing that doing intervals prior to steady state burned more fatty acids than doing steady state before intervals. Always nice to be validated, eh?

In any case, after a couple of years of playing with it and getting feedback, the new and improved SFP1.0 is the following.

The New and Improved Stubborn Fat Protocol 1.0

1. Can be done any time of the day. Ideally, the protocol should come 3 or more hours after a meal.
2. Caffeine (100-200 mg), yohimbine (0.2 mg/kg), tyrosine (1-3 grams) 30 minutes before. All supplements are optional.
3. 5-10 easy warmup.
4. 10 minutes of intervals OR up to 20 minutes of high rep/short rest weight training. Anywhere from 10X15 seconds hard/45 seconds easy at a perceived exertion approaching 9-10 during the hard bits (make the easy bits very easy) up to 5X1 minute hard/1 easy at a perceived exertion of 7-8 during the hard bits. Intervals would ideally be done on a machine you don't usually use to maximize the hormonal response.
5. Rest completely for 5 minutes.
6. 20-40 minutes steady state cardio at an RPE of 3-4. This should be done on the type of cardio machine you habitually use, as you'll generally be able to burn the most calories this way since you can usually perform more work at the same low heart rate.
7. Can eat immediately afterwards.

Notes:

Point 2: I don't want it to sound like you have to take either all three supplements or none at all. The protocol will work without them although it will tend to work better with them. You can use any or all of the suggested supplements depending on preference and what you have available (e.g. yohimbine is not available everywhere).

Point 4: Intervals are ideally done on an alternative machine to what you normally use but this is not required. If it's not possible for some reason or another you can simply use the same machine for both the interval and steady state bits. As a general comment about doing intervals, unless someone has been trained to do it properly, I'm generally no fan of running for the interval part. Most people have horrible running form and heavier athletes often get joint problems. I'd recommend sticking with a non-impact form of cardio here.

Additionally, I offered the possibility of using metabolic style weight training (generally made up of whole body exercises performed for relatively higher repetitions with short rest periods) in place of the interval portion of the workout; this is because the overall hormonal and metabolic effect of that kind of training is quite similar to interval training itself. Studies dating back to the 80's showed that that kind of training (which was once popular with elite bodybuilders) mobilizes fatty acids.

If dieters choose to use resistance training rather than intervals, they will probably generally be doing a slightly greater volume of training, which is why I allowed for twenty minute of metabolic weight training versus the 10 minutes of intervals. The same basic work to rest ratios should be kept for the weight training. That is, you could do something like 15 seconds of a weight training exercise (it would need to be something very full body of course) with a short rest before doing it again for the total duration. Many individuals are using a protocol called the Tabata protocol which consists of 8 'rounds' of 20 seconds hard/10 seconds easy.

While I don't personally advocate Tabata with weights (I am concerned about form degradation and injuries), it can be done. Alternately, using longer sets, in the 45-60 second range with roughly equal rest would be effective. This might mean 12-15 repetitions (on a fairly controlled cadence) with a 30-60 second rest until the total volume of training was done. This would still be followed by the five-minute break before continuing with the steady state cardio to burn off the now mobilized fatty acids.

And that's the current SFP1.0. As mentioned in my warnings in the previous chapter, the original protocol sort of took on a life of its own and some people got a little bit crazy with it, overtraining themselves into the ground. One desperate bodybuilder did the above twice per day every day for 2 weeks going into his show. He ended up horribly burned out and overtrained and it was his own damn fault.

Realistically, the SFP 1.0 should only be used twice per week under most situations. And here I'm assuming that heavy weight training is being done for the legs twice/week. Someone doing more leg training than this (which would probably be a mistake while dieting anyhow) would need to cut back the SFP1.0 frequency to once/week. If someone were willing to cut back their heavy leg training to once/week, they might get away with a three times per week frequency of performing the SFP1.0. Yes, that means a more traditional bodypart split but that's perfectly acceptable when contest dieting.

But a three times per week frequency would be the absolute maximum and, again, the exception rather than the rule. I'd refer you to last chapter's warning rather than repeat myself here.

As one more programming note, I'd comment that most tend to perform heavy leg training and interval training on different days but this isn't necessarily ideal. I've long advocated putting interval training on the same day that people train legs. Yes, this makes for one hell of a hard day. But it gives the legs more total days of recovery per week. So instead of lifting legs twice/week and performing intervals on two other days/week (which only gives the legs three rest days); by doubling up the sessions, the legs are only trained two days per week heavily (although the individual days are much harder) allowing for five days of rest per week.

Ideally, when doubling up, you'd do the two workouts with at least a 4-6 hour break (realistically most will do it around their work schedule); this isn't always possible. So within the context of a diet, you might do the SFP1.0 in the morning (or at lunch) and then perform weights later in the evening (after work). Yes, weights will probably suffer but that's how it goes; life is full of compromises and this is one of them.

If someone can't train twice daily, there are a couple of options. One is to perform the SFP1.0 immediately after weights (preferably leg training). If they are performing some type of high rep/short-rest weight training, that can act as the interval part of the SFP as mentioned above. If that's still not doable, it would be best to cut leg-training frequency and perform the SFP1.0 on the days you'd have normally trained legs. That is, let the SFP replace a heavy leg day instead of adding to it.

Let me reiterate, the SFP1.0 is intense, it will over train you if you don't respect it and try to put it in with too much other leg training, or do it too frequently or what have you. If it fits into your training (and this is likely not the case for performance athletes who must do a ton of other specific training), use it sparingly, a couple of times per week and cut back your other leg training to help with recovery. On the other days, use Protocol 1 or 2.

Have I mentioned that the SFP2.0 is even tougher?

Another warning from your friendly author

In the last chapter, I had a whole section of warnings about how trying to do the higher intensity stubborn fat protocols too often is a recipe for disaster. Without repeating myself endlessly, I want to really reiterate that point with some warnings specific to the SFP2.0. Everything I said in the previous chapter applies here, so do my comments on the SFP1.0 above.

Except that they apply that much more to the SFP2.0. I've had a number of people test out the protocol. Some, including the guy on the cover, absolutely thrived on it. Another coach I know used it with many of his clients, finding that it performed best used sparingly. They did the protocol roughly 4 times in 2 weeks, any more frequently and they couldn't recover.

However, others, including a group coached by a friend who helped me optimize the original SFP1.0, simply got wrecked by the modified protocol. They could not recover from it on reduced calories no matter how he adjusted their other training and he went back to the SFP1.0 coupled with some of the low intensity options on the other days.

What's the difference? Were the folks who thrived just studs, was my other friend's group of trainees just weak? I don't know. The guy on the cover is a bit of a freak, he can handle insane training loads and has amazing recovery (he's also natural, by the way). He was also very smart in his implementation of the SFP2.0.

First and foremost, he cut his leg training back to once/week as he got further into his diet. Additionally, if on any given day he was feeling tired, he'd cut back the intensity of the

intervals a bit, or reduce the steady state part to only 20 minutes. The other tester who made the protocol work uses a low-volume, bodypart split approach to training his clients. With only one heavy leg day per week, they were able to do the protocol (still infrequently) effectively. Honestly, I don't know why the third coach couldn't make it work; I'll only note that his athletes couldn't handle the full SFP2.0.

The commonality seems to be keeping the overall workload down (and adjusting it based on the individual's recovery) to allow for recovery for the absolute assault of the SFP2.0. While ideal, this tends not to describe the average extreme dieter.

Many, regardless of how they feel, will try to perform every rep of every set that their training program calls for, even when they feel miserable and exhausted; the spiral into overtraining is usually not far behind. These folks will take the SFP2.0 protocol as written and attempt to complete it as often as possible doing every interval, every minute of steady state, and they won't cut back their other training. And they'll pay a heavy price as I talked about before.

Interestingly, with the above said, certain types of performance athletes probably have a better chance of integrating/surviving the SFP2.0 than the typical hardcore dieter or physique athlete. That's simply because certain types of sports could use the basic structure of the SFP2.0 to get a workout that would be pretty optimized for improving performance in the first place. That is, even though I've aimed it at absolutely maximizing stubborn fat loss, it has a performance element to it as you'll soon see.

So those are the warnings that are specific to the SFP2.0, please pay attention to them or you will pay for it. This protocol is not for everyone and that's just reality. If you can handle it, it will outperform anything you've ever tried. If not, stick with the other protocols; you'll still reach your goals.

And now, finally.....

Drum roll please.

I've explained the physiology, I've given the warnings, I've done all I can do except for one thing: It's taken me 10 years of obsessiveness to get to this point but finally I give you the SFP 2.0.

Protocol 4: The Stubborn Fat Protocol 2.0

To fully understand how I arrived at the SFP2.0, I need to walk you through the history of its development. As you'll see, the idea came out of the original SFP1.0.

Back in 2007, I was sitting around thinking about some of the details of the SFP1.0, clearly it worked and worked well but my OCD brain wasn't happy.

One thing that bothered me was this, although I have zero data to back it up, I had this weird hunch that doing the steady state cardio after the intervals would have the effect of essentially blunting any extra calorie burn from the intervals. Sure, it's turning out that

this effect isn't nearly as large as people think or claim. Realistically, you might burn an extra 30-50 calories following a typical interval session but when you're down to the last pound of fat or two and it's all stubborn, every gram counts.

Well, the solution to that was pretty damn simple: rather than doing all of the intervals up front, just split them before and after the steady state cardio. What I didn't want to do was to double up the interval volume by adding more intervals after the middle bit, so splitting the volume up made more sense. Ok, so now we have a protocol consisting of a warm-up, 5 minutes of short intervals, 5 minute break, 20-40 minutes of steady state cardio, 5 minutes of additional intervals, short cool down and done. It was ok, but it wasn't perfect.

I still had concerns about the optimal interval length to make it do exactly what I wanted it to do. I wanted to get maximal fatty acid mobilization up front, maximal fat oxidation during the steady state bout and then any additional benefit from intervals at the end in terms of afterburn. Ideally without killing the person.

Now there are pros and cons to both longer and shorter intervals. Looking at the hormonal response I wanted, shorter intervals seemed to fit better up front. You can jack up the intensity to extremely high levels which really gets adrenaline/noradrenaline pumping. Even better, shorter intervals accomplish that without exhausting the athlete (either systemically or just for the low intensity cardio bit that came afterwards). By avoiding huge lactate spikes, there should be less of an impact on trapping fatty acids in the fat cell. I had found a study or two showing that shorter intervals had a greater impact on lipolysis during the exercise bout and did so with less fatigue so that was pretty positive. So far so good.

I didn't see any need to change the break or the steady state bit in the middle since, outside of playing around with intensity, there wasn't much to worry about there. As mentioned in the section above on SFP1.0, it might be ideal to work very close to the lactate threshold during this part of the protocol for maximal fat oxidation. In practice, this ends up being too high of an intensity for most people (performance athletes are the most likely to be able to get away with this). But this section stayed basically the same as the SFP1.0.

But what about the intervals at the end? Short intervals are nice and all but they don't cause the metabolic perturbation or glycogen depletion of longer intervals. I've mentioned that glycogen depletion enhances full body fat burning, it also gives a bigger 'sink' for incoming calories to go to muscle. And even though the afterburn effect is turning out to be a lot smaller than once thought, when you're down to the last pound of fat, every bit counts as I mentioned above. And longer intervals, due to the greater metabolic effects will have a bigger impact here. Putting it all together, we end up with the protocol detailed on the next page.

The Stubborn Fat Protocol 2.0

1.Can be done fasted or not. If not, try to do it at least 3 hours after eating.
2. Can take caffeine (100-200 mg), oral yohimbine (0.2 mg/kg), L-tyrosine (1-3 grams) 30 minutes before hand.
3. 5-10 minute easy warm-up. This will drop insulin if you've eaten beforehand.
4. 5 minutes of short intervals. Five repeats of 10-15 seconds at near maximum intensity (RPE of 9-10), 45-50 seconds recovery done at a very low intensity. Ideally do this on a machine you don't usually use. If you have *amazing* work capacity and recovery, you can increase this to 10 minutes of total intervals.
5. Rest for 5 minutes.
6. 20-40 minutes of steady state cardio at a low to moderate intensity, 130-140 HR or an RPE of about 3-4. This should be done on whatever cardio machine you typically use since you'll generally be able to burn the most calories for a given heart rate this way.
7. 5-10 minutes of long intervals. Along the lines of 5X30 seconds on/30 seconds off up to a maximum of 5X1 minute on/1 minute off. RPE 7-8 (higher for the shorter intervals and lower for the longer intervals) and don't hesitate to cut volume or duration if your legs are fried. These intervals can be done on the machine of your choice, it can be the same one you did the steady state cardio on or a different piece if you prefer.
8. 3-5' easy cooldown.
9. Feel free to eat a normal diet meal afterwards, at least have a protein shake if you're not hungry.

Notes:
Point 4: I'm hesitant to even offer the possibility of doing 10 full minutes of intervals at the outset because I'm afraid that most people will try to use the maximum as the default. Do NOT do this. When you first try this protocol, if you try this protocol, start with only 5 minutes of short intervals up front. If you recover ok from it, you can gradually increase this to a total of 10 minutes of 15 seconds at near maximum with 45 seconds very easy for rest.

Point 7: The same comment as for point 4. Folks with amazing work capacity may be able to work up to a total of 10 minutes of intervals after the steady state portion of the protocol. But everyone should start with 5 minutes here for the first several times they do the protocol to see how they can handle it. On any given day, if your legs are trashed, consider cutting back the volume of either the pre- or post-steady state interval portions.

As a general note on the SFP2.0, the same comment from Point 4 of the SFP1.0 applies to both interval sections of the SFP2.0: unless you're trained to sprint, I don't recommend running for the intervals (especially the short ones). Pick something non-impact that you can go hard on without getting hurt.

As mentioned in the SFP1.0, metabolic type weight training could be substituted for either (or both) of the interval parts of the workout above. For the intervals at the front of the protocol, you'd want to keep the sets short (and intense) with relatively short rests. Again, something like the Tabata protocol (8 sets of 20 seconds hard/10 seconds easy) could be used. For the intervals at the end of the protocol, keep the work sets longer (30-60 seconds) with relatively longer rest periods (30-60 seconds between sets). Of course,

there's nothing that says you have to use weight training both before and after. You might prefer to use traditional intervals first and metabolic weight training second or the converse (metabolic weight training first, regular intervals after the steady state portion).

When my first test subject (on the cover) first performed the protocol, he noted not only the phantom tingle but also a pronounced thermogenic effect for the rest of the day. That told me I had nailed it with the combination of short intervals up front and long intervals at the end. He used it during the entirety of his contest prep and his condition at the end of it speaks volumes. As a natural, he came in leaner and tighter than ever with striated glutes, winning his pro card.

I've already mentioned that he cut his leg training volume way back, this was the only way that his legs could handle the metabolic assault of the SFP2.0 without overtraining. I mentioned issues that other testers ran into above and I'll spare you the rehash here except to say that you must respect the SFP2.0 and be willing to adjust your training (or eliminate the protocol completely) as you go.

Assuming that they can handle it at all, the SFP2.0 might be doable by the average dieter twice per week; it'd be one hell of an individual who could do it three times per week on non-consecutive days (don't even think about doing it two days in a row) and I'm tempted to say, in absolute terms, don't do it.

The same scheduling issues I described for the SFP1.0 apply here. If you absolutely must train legs heavily twice per week, try do to the SFP2.0 on the same days, your legs will get more recovery. The other days should be used for either Protocol 1 or 2 above.

I'd note that there's absolutely no reason you couldn't do the SFP1.0 once/week, the SFP2.0 once/week and then uses Protocol 1 or 2 on the other days. Someone who can't recover from the SFP2.0 twice/week might very well handle it once/week in conjunction with the other protocols.

Ideally, if you double up your training, you'd do the SFP2.0 with at least 4-6 hours between the SFP2.0 and weight training. Since the SFP2.0 is specifically for dieting, I'd generally say to do the SFP2.0 in the morning and weights in the evening. Your weights will probably suffer but that's the price you have to pay. It's better than systemically overtraining by hitting legs too hard too frequently on a diet.

Of course, not everybody can or wants to do two workouts per day, or their schedule won't allow the ideal break. Some people will be forced to do the SFP2.0 after weights. If you do the SFP2.0 after leg weights, be very aware of the potential for injury doing intervals on tired legs. I'd strongly suggest using a mode of exercise for the intervals where you aren't at risk for turning an ankle or knee from fatigue.

Many also perform some type of high rep or metabolic weight training; as noted above this can substitute for either the first, second or both interval portions of the SFP2.0. Many will also perform a bit of heavy work first before doing the metabolic work. So on a leg day, you might do a low volume of lower-body work (for muscle mass and strength maintenance) and then move into your metabolic work to start the SFP2.0 (so do metabolic weight training, rest 5 minutes, steady state, finish off with long intervals).

As I mentioned above, performance athletes, especially endurance athletes, can probably use the SFP2.0 effectively as part of their training. Doing short intervals prior to a harder aerobic section and then finishing off with longer intervals would make a pretty good performance workout for a variety of reasons outside of the scope of this book.

I'm sure other athletes can come up with some creative way to apply the SFP2.0 to their sport. Warm-up followed by short sprints, followed by brisk walking, followed by some high repetition kettlebell work? Nobody has tried it yet but when you do, please send me an email to tell me how (well) it worked.

And that's basically that, the SFP2.0 for the world to see. I'd only reiterate that anybody who decides to use the SFP2.0 must be prepared to cut back their other training and adjust the protocol as necessary. If you're exhausted from dieting, save it for another day. If you're just not recovering, drop it completely.

As a semi-tangential note, trainees should always be watching out for impending signs of overtraining such as lethargy, fatigue, lack of motivation, sleep problems, etc. This risk is even higher on a diet. Just realize that by the time you've noticed the signs, it's usually too late.

The End

So that's it, that's the solution to stubborn fat. At this point in time, what I put in this book is the absolute cutting edge in terms of removing stubborn fat on a diet, and I feel that I can now move onto another project (next up: women and fat loss, why do they have the problems that they do).

I've covered a tremendous amount of information, perhaps too much. I suppose I could have just put out the protocols without the background but, as people who know me know, that's not how I write books. Just saying 'Do this' without making you understand why wouldn't have made me happy.

At the end of the day, even if you never apply word one from this book to your own training, you now know more about the topic of body fat (and especially stubborn body fat) than most anybody out there (except me, of course).

You've learned what body fat is and what purposes it serves in the body. In contrast to the long held belief that fat is just a passive (and ugly) store of energy, fat is turning out to be an endocrine organ in its own right. It's metabolically active, it releases a ton of hormones, and it communicates with every other tissue in your body including your brain in order to control various aspects of your physiology.

You learned about the different 'types' of body fat as well. Essential fat, brown adipose tissue, visceral fat, subcutaneous fat and stubborn fat were all detailed. To start understanding why stubborn fat is stubborn, you had to learn about fat cell metabolism, how fat cells actually store and mobilize fat for fuel. You also learned the three primary processes controlling how fat is 'burned': the mobilization, transport, and oxidation of stored body fat.

How hormones impact body fat is a key to understanding why stubborn fat is stubborn. Far beyond the simple 'estrogen is bad' and 'testosterone is good' you saw how myriad hormones impact on fat cell metabolism. Frankly, this is still an area ripe for new research. Seemingly every week a new hormone or factor is found, some turn out to affect fat cells, others do nothing (or only work in mice and rats).

Finally I addressed the issue of what makes stubborn fat stubborn. You learned about adrenoceptors, blood flow and how insulin affects different depots of fat. A couple of other

factors including what types of fat are stored in the cell, cell size, and how global dieting concerns such as metabolic rate slowdown all affect stubborn fat.

Then I was able to address how the primary factors that are in our control (namely diet, exercise, and supplements) can impact on fat loss in general and stubborn fat in specific.

All of that was just a lead up to the protocols themselves. Some of you may already have been familiar with the first three protocols. My new stubborn fat protocol (2.0) is brand new and has been kept secret outside of the people who tested it for me. It represents the absolute most cutting edge approach to stripping stubborn fat that I can come up with.

Finally, 10 years after it started, my mind can rest.

Using the approaches outlined in this book, I don't feel that any dieter should run into problems with stubborn fat, at least not physiologically. There are other issues that can stop a diet in its tracks unrelated to stubborn body fat. Hunger, overtraining, just being tired of feeling like crap; all of these can stop dieters before they reach their goals. But assuming these can be overcome, the methods in this book should prevent the kinds of dieting disasters of the 'My upper body got ripped but I still had fat legs' kind.

Is this book the final word? For the time being I'd say yes although there are still new pathways of interest. Atrial natriuretic peptide (ANP) which I talked about in Chapter 6 is incredibly promising. Figure out how to manipulate that and you can side-step most of the problems with stubborn fat in the first place. Topical fat reduction creams, such as the licorice cream I mentioned in Chapter 8 also seem to hold some promise. So far, nothing seems to have really panned out.

But at this point, other than keeping track of the topic tangentially, I probably won't be the one to really delve into those issues. I've put in my time, I've put it all down in this book, I'm done.

Thank you...and good night.

References

As I mentioned, this project represents the culmination of nearly 10 years of research and thinking about the topic, a comprehensive reference list would be impossible. However, since I know some people will want to follow up on some of the statements I've made, I've decided to post an abridged list of what are key references on the topic. By going to one of the free Pubmed or Medline sources, readers can find the key articles and hit the 'Related articles' tab to turn up as much information as they want on the topic.

I've grouped the references roughly by category and made some comments on each as necessary.

Adipose tissue blood flow

Astrup A et. al. Skin temperature and subcutaneous adipose tissue blood flow in man. Scand J Clin Lab Invest (1980) 40: 135-138.

Demonstrated how skin temperature can impact on adipose tissue blood flow.

Bulow J and J Madsen. Influence of blood flow on fatty acid metabolism mobilization from lipolytically active adipose tissue. Pflug Arch (1981) 390: 169-174.

A study (in dogs) showing the crucial importance of adipose tissue blood flow for optimal lipolysis.

Bulow J and J Madsen. Adipose tissue blood flow during prolonged heavy exercise. Pflugers Arch (1976) 363: 231-234.

Showed that 3 hours of continuous activity will drastically increase adipose tissue blood flow.

Crandall DL et. al. A review of the microcirculation of adipose tissue: anatomic, metabolic, and angiogenic perspectives. Microcirculation (1997) 4: 211-232.

One of the only two review papers (I have both of course) looking at how adipose tissue blood flow is regulated.

Engefeldt P. et. al. Subcutaneous adipose tissue blood flow in the abdominal and femoral regions: effect of fasting. In J Obes (1992) 16: 875- 879.

A paper showing the impact of fasting (which can be mimicked by a low-carbohydyrate diet) on adipose tissue blood flow.

Galitzky J et. al. Role of vascular alpha-2 adrenoceptors in regulating lipid mobilization from human adipose tissue. J Clin Invest (1993) 91: 1997-2003.

A paper showing that alpa-2 adrenceptors are just as involved in blood flow as lipid *metabolism.*

Hjemdahl P and BB Fredholm. Influence of adipose tissue blood flow on the lipolytic response to circulating noradrenaline at normal and reduced pH. Acta PHysiol Scand (1976) 98: 74-79.

Showed that lowering pH (as might occur with high intensity intervals which generate acidosis) can impair adipose tissue blood flow.

Wennlund A and B Linde. Influence of hyper- and hypothyroidism on subcutaneous adipose tissue blood flow in man. J Clin Endocrinol Metab (1984) 59 258-262.

Examines the impact of thyroid hormone status on adipose tissue blood flow.

Adrenoceptors

Lanfontan M et. al. Alpha-2 adrenoceptors in lipolysis: alpha2 antagonists and lipid-mobilizing strategies. Am J Clin Nutr (1992) 55: 219s-227s.

An early paper looking at the role of alpha-2 adrenoceptor antagonist (such as yohimbe) and how they might help with fat loss.

Lafontan M Adrenergic regulation of adipocyte metabolism. Hum Reprod. (1997) 12 Suppl 1:6-20.

One of many reviews on adrenoceptors and how they modulate fat cell metabolism.

Mauriege P. et. al. Heterogeneous distribution of beta and alph-2 adrenoceptor binding sites in numan fat cells from various fat deposits: functional consequences. Eur J Clin Invest (1987) 17: 156-165.

One of the first papers showing differences in adrenoceptor ratios between different fat depots.

Basic fat cell metabolism

Arner P. Human fat cell lipolysis: Biochemistry, regulation and clinical role. Best Pract Res Clin Endocrinol Metab. (2005) 19:471-82.

A paper that everyone who still wants to use rat/mouse models to develop fat loss approaches should read since it points out that murine fat cell metabolism is basically completely different than what goes on in humans.

Bjorntop P. The regulation of adipose tissue distribution in humans. Int J Obes (1996) 20: 291-302.

Another good review looking at how the distribution of body fat is regulated in humans.

Carmen GY, Víctor SM. Signalling mechanisms regulating lipolysis. Cell Signal. 2006 Apr;18(4):401-8.

A detailed look at the mechanisms involved in lipolytic signaling.

Fielding BA and KN Frayn. Lipoprotein lipase and the disposition of dietary fatty acids. Br J Nutr. (1998) 80:495-502.

An excellent review of the role of LPL in fat cell metabolism.

Frayn KN. Adipose tissue as a buffer for daily lipid flux. Diabetologia. (2002) 45(9):1201-10.

A good paper explaining the crucial role of body fat in storing fatty acids during the day (after meals).

Frühbeck G. A heliocentric view of leptin. Proc Nutr Soc. (2001) 60(3):301-18.

One of about a zillion reviews on leptin physiology, I remember this one being particularly good.

Hauner H. Secretory factors from human adipose tissue and their functional role. Proc Nutr Soc (2005) 64: 163-169.

One of a zillion review papers looking at all of the hormones that fat cells release.

Laaksonen DE et. al. Changes in abdominal subcutaneous fat water content with rapid weight loss and long-term weight maintenance in abdominally obese men and women. Int J Obes (2003) 27: 677-683.

One of the few papers examining the issue of how fat cells may refill with water after the fat has been removed.

Malcolm GT. et. al. Fatty acid composition of adipose tissue in humans: differences between subcutaneous sites. Am J Clin Nutr (1989) 50: 288-291.

A paper showing that different depots of fat store different fatty acids (i.e. saturated vs. unsaturated) preferentially.

Raclot T. et. al. Selective release of huma adipocyte fatty acids according to molecular structure. Biochem J (2997) 324: 911-915.

A paper showing that different types of fatty acids are easier or harder to mobilize fat from fat cells.

Diet

Bach AC et. al. The usefulness of dietary medium-chain triglycerides in body weight control: fact or fancy? J Lipid Res. 1996 Apr;37(4):708-26.

A good review of the impact of MCTs on fat loss and obesity.

Bisschop PH. et. al. The effect of carbohydrate and fat variation in euenergetic diets on postabsorptive free fatty acid release. Br J Nutr (2002) 87: 555-559.

A look at how diet affects fatty acid release between meals.

Coppack SW et. al. Nutritional regulation of lipid metabolism in human adipose tissue. Exp Clin Endocrinol Diabetes (2001) 109: S202-S214.

A good review of how nutrient intake affects lipid metabolism in fat cells

Gesta S et. al. In vitro and in vivo impairment of alpha2-adrenergic receptor-dependent antilipolysis by fatty acids in human adipose tissue. Horm Metab Res. (2001) 33(12):701-7.

The paper showing that 4 days on a low-carb/high-fat diet naturally inhibits alpha-2 adrenoceptors.

Patel JN et. al. Norepinephrine spillover from human adipose tissue before and after a 72-hour fast. J Clin Endocrinol Metabolism (2002) 87: 3373-3377.

Found that the body increases nervous system output after 3 days of fasting (mimicked by a low-carbohydrate diet).

Rudkowska I et. al. Diacylglycerol: efficacy and mechanism of action of an anti-obesity agent. Obes Res. 2005 Nov;13(11):1864-76.

A comprehenive look at the impact of diglycerides (DGs) on metabolism.

Different types of body fat

Ardioluze J-L. et. al. Subcutaneous tissue blood flow varies between superior and inferior levels of the anterior abdominal wall. Int J Obes (2004) 28: 223-233.

Showing that superficial abdominal fat can be subdivided into upper and lower pieces in terms of blood flow.

Monzon JR et. al. Lipolysis in adipocytes isolated from deep and superficial subcutaneous adipose tissue. Obes Research (2002) 10: 266-269.

A paper looking at the differences in superficial and deep abdominal fat.

Enevoldsen LH et. al. In vivo human lipolytic activity in perperitoneal and subdivisions of subcutaneous abdominal adipose tissue. Am J Physiol (2001) 281: E110-E114.

A paper showing that deep and superficial abdominal fat are metabolically different with deep ab fat acting more like visceral fat.

Rebuffe-Scrive M. et. al. Fat cell metabolism in different regions in women. Effect of menstrual cycle, pregnancy and lactation. J Clin Invest (1985) 75: 1973-1976.

A good review of how fat cell metabolism in different part's of women's bodies changes during different hormonal events.

Exercise and lipolysis

Arner P et. al. Adrenegic reguation of liipolysis in site at rest and during exercise. J Clin Invest (1990) 85: 893-898.

Showed the adrenoceptor regulation of lipolysis is different at rest and during exercise.

Astorino TA. Is the ventilatory threshold coincident with maximal fat oxidation during submaximal exercise in women. J Sports Med Phys Fitness (2000) 40: 209-216.

A paper looking at where fat oxidation (in terms of absolute grams) is maximized.

Bell DG et. al. Effects of ingesting caffeine and ephedrine on 10-km run performance. Med Sci Sports Exerc (2002) 34 344-349.

The paper showing that ephedrine could inhibit the normal adrenaline/epinephrine response to exercise.

Christmass MA et. al. Effect of work and recovery duration on skeletal muscle oxygenation and fuel use during sustained intermittent exercise. Eur J Appl Physiol (1999) 80: 436-447.

Showed that shorter intervals had less of a negative impact on lipolysis during exercise than longer intervals, and that muscle oxygenation stayed higher.

Egan D and T. Head. Energy substrate metabolism during dual work rate exercise: Effects of order. J Sports Sci (1999) 17: 889-894.

The paper showing that doing intervals before steady state exercise works differently than the reverse order.

Goto K, et. al. Effects of resistance exercise on lipolysis during subsequent submaximal exercise. Med Sci Sports Exerc. (2007) 39(2):308-15.

Paper showing that resistance exercise can improve lipolysis during aerobic activity done some time afterwards.

Horowitz JF. Fatty acid mobilization from adipose tissue during exercise. Trends Endocrinol Metab. 2003 Oct;14(8):386-92.

A good review of the impact of exercise on adipose tissue metabolism, including the impact of intensity.

LaForgia J et. al. Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. J Sports Sci. (2006) 24(12):1247-64.

An epic review on the impact of exercise on post-exercise calorie burn, pointing out that the magnitude of the effect is much much smaller than many believe or claim.

Price M and P. Moss. The effects of work:rest duration on physiological and perceptual resonses during intermittent exercise and performance. J Sports Sci (2007) 109. Epub before print.

Showed that shorter intervals generate less metabolic and perceptual strain than longer intervals.

Stallknecht B. et al. Are blood flow and lipolysis in subcutaneous adipose tissue influenced by contractions in adjacent muscles in humans? Am J Physiol Endocrinol Metab. (2007) 292(2):E394-9.

This is the paper I mentioned in Chapter 1 that everybody took out of context to try to support spot reduction.

Gender differences

Doucet E. et. al. Reduction of visceral adipose tissue during weight loss. Eur J Clin Nutr (2002) 56: 297-304.

A paper looking at how visceral fat loss is affected by gender.

Horton TJ et. al. Postprandial leg uptake of triglyceride is greater in woman than men. Am J Physiol (2002) E1192-E1202.

Another paper showing preferential deposition of calories in women's legs after a meal.

Jensen MD et. al. Effects of gender on resting leg blood flow: implications for measurement of regional substrate oxidation. J Appl Physiol (1998) 84: 141-145.

Jensen's lab has done a ton of work on the topic of adipose metabolism between genders.

Hellstrom L et. al. Gender differences in adrenergic regulation of lipid mobilization during exercise. Int J Sports Med (1996) 17 439-447.

Another good review on gender differences in fat cell metabolism during exercise.

Power ML and J Shulkin. Sex differences in fat storage, fat metabolism and the health risks from obesity: possible evolutionary origins. Br J Nutr (2007) 1-10.

A good look at gender differences in fat storage with evolutionary reasons things might have developed that way.

Romaniski SA et. al. Meal fatty acid uptake in adipose tissue: gender effects in nonobese humans. Am J Physiol (2000) 279: E455-E462.

A paper showing clear gender differences in how fat is taken up into fat cells after a meal.

Shadid S et. al. Direct free fatty acid uptake into human adipocytes in vivo: relation to body fat distribution. Diabetes (2007) 56: 1369-1375.

This is the paper showing that fat cells can directly absorb FFA from other cells along with suggestion that women's bodies may redistribute fat from one depot to another.

Vortruba SB and MD Jensen. Regional fat deposition as a factor in FFA metabolism. Annu Rev Nutr (2007) 27 149-164.

A very recent and thorough review looking at how men's and women's bodies differentially regulate fat store and mobilization and how this impacts on regional body fat distribution.

Hormones and fat cell metabolism:

Anderson LH et. al. the effects of androgens and estrogens on preadipocyte proliferation in human adipose tissue: influence of gender and site. J Clin Endocrinol Metab (2001) 86: 5045-5051.

Goes part way to explaining how changes in hormones at puberty impacts on the development of fat cells, their metabolism, and fat deposition patterns.

Cianflone K et. al. Acylation stimulating protein (ASP) an adipocyte autocrine: new directions. Seminars in Cell Developmental Biology (1999) 10: 31-41.

The paper that everyone still locked into the 20 year old insulin/LPL model of fat storage should read, detailing the role of ASP in fat cell metabolism and fat storage.

Cianflone K. et. al. Critical review of acylation-stimulating protein physiology in humans and rodents. Biochimica et Biophysica Acta (2003) 127-143.

A good recent review on ASP physiology.

Gravholt CH et. al. Effeects of a physiological GH pulse on interstitial glycerol in abdominal and femoral adipose tissue. Am J Physiol (277) E848-E854.

Showing that normal GH pulses impact on lipolysis.

Gravholt CH Physiological levels of glucagon do not influence lipolysis in abdominal adipose tissue as assessed by microdialysis. J Clin Endocrinol Metab. (2001) 86:2085-9.

One of many papers showing that glucagons is not lipolytic in humans. Give it up, guys.

Jensen MD. Cytokine regulation of lipolysis in humans? J Clin Endocrinol Metab. (2003) 88:3003-4

A short review looking at the major hormones impacting on lipolysis.

Lafontan M et. al. Recent development on lipolysis regulation in humans and discovery of a new lipolytic pathway. Int J Obes (2000) 24: S47-S52.

The first paper I recall that mentioned atrial-natriuretic peptide as a possible non-adrenoceptor mediator of lipolysis, also a good review of lipolytic regulation in general.

Mayes JS and GH Watson. Direct effects of sex steroid hormones on adipose tissue and obesity. Obes Rev (2004) 5: 197-216.

An excellent overview of how testosterone, estrogen and progesterone affect body fat.

Meek SE. et. al. Insulin regulaion of free fatty acid metabolism. Diabetes (1999) 48: 10-14.

A paper showing distinct differences between the types of fat and how insulin affects fatty acid metabolism.

Ottonson M et. al. Effect of cortisol and growth hormone on lipolysis in human adipose tissue. J Clin Endocrinol Metab (2000) 85: 799-803.

Title should be self-explanatory.

Pedersen SB et. al. Estrogen controls lipolysis by up-regulating alpha-2A-adrenergic receptors directly in human adipose tissue through the esrogen receptor alpha. Implications for the female fat distribution. J Clin Endocrinol Metabolism (2004) 89: 1869-1878.

Showed that estrogen could upregulate alpha-2 receptor number rapidly in human fat cells.

Samra JS et. al. Effects of physiological hypercortisolemia on the regulation of lipolysis in subtaneous adipose tissue. J Clin Endocrinol Metab (1998) 83: 626-631.

A paper examining the schizophrenic effects of cortisol and how it can either mobilize or store fat depending on the specifics of what's going on.

Seckl JR e. al. Glucocorticoids and 11beta-hydroxysteroid dehydrogenase in adipose tissue Recent Prog Horm Res. (2004) 59:359-93.

A recent review looking at the impact of 11-beta-HSD and how it impacts on fat cell metabolism and cortisol 'reactivation'.

Topical fat reduction creams

Armanini D et. al.. Glycyrrhetinic acid, the active principle of licorice, can reduce the thickness of subcutaneous thigh fat through topical application. Steroids. (2005) 70(8):538-42.

The study showing that a topical licorice cream could reduce thigh fat.

Caruso MK et. al. Topical fat reduction from the waist. Diabetes Obes Metab. (2007) 9(3):300-3.

This paper used a topical aminophylline cream for topical fat reduction.

Greenway FL et. al. Topical fat reduction. Obes Res. (1995) 3 Suppl 4:561S-568S.

The study showing that a theophylline cream could reduce fat, it appears that the effect may have mainly been water loss (theophyilline has mild diuretic effects).

Greenway FL and GA Bray. Regional fat loss from the thigh in obese women after adrenergic modulation. Clin Ther. (1987) 9:663-9.

Tested a variety of topical creams including yohimbe, forskolin, and others to try to induce local fat loss.

Tholon L et. al. An in vitro, ex vivo, and in vivo demonstration of the lipolytic effect of slimming liposomes: An unexpected alpha(2)-adrenergic antagonism. J Cosmet Sci. (2002) 53(4):209-18.

The slimming liposomes paper I mentioned, I'm surprised nobody ever brought this to market.

LYLE MCDONALD

Hardcore dieters have no problem getting certain areas super lean. Shoulders, upper back, etc. all come in just fine. But most people have those trouble spots. For men it's usually the abdominal and low-back area, women's lower body fat has been a problem for years. Various solutions have been proposed over the years, usually based on simplistic explanations of what causes the problem.

The Stubborn Fat Solution represents the culmination of a 10-year obsession I've had with the problem of stubborn body fat and how to eliminate it. Presenting four different training, nutrition and supplement protocols, it will give dieters all of the tools they need to get the stubborn fat off once and for all.

Topics covered include:

- What body fat actually is and what it does in the body
- An explanation of the different 'types' of body fat
- How fat cells store and mobilize body fat
- What it means to 'burn' body fat and the key processes involved
- How hormones affect stubborn body fat
- Why stubborn body fat is stubborn in the first place
- How nutrition, training and supplements impact on stubborn body fat loss
- The four different protocols that dieters can use to attack stubborn body fat to get rid of it once and for all

If your previous diets have been stalled because you couldn't get the last bit of stubborn body fat off, *The Stubborn Fat Solution* will be the answer to all of your questions. First you'll learn why stubborn fat is so stubborn, then I'll give you the tools to get rid of it in the fastest and most efficient way possible.

About the Author

Lyle McDonald is a physiologist and author who has spent over a decade obsessively finding ways to apply cutting edge scientific research to sports nutrition, fat loss, and muscle gains. His first book **The Ketogenic Diet** is generally considered to be the most comprehensive book ever written on the topic of low-carbohydrate diets. His latest book **The Protein Book** is the definitive reference on protein intake for athletes. His other books: **The Ultimate Diet 2.0**, **The Rapid Fat Loss Handbook** and **A Guide to Flexible Dieting** have helped thousands to lose fat and keep it off.

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