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 The Women's Book: Volume 1 A Guide to Nutrition, Fat Loss, and Muscle Gain by Lyle McDonald w/Eric Helms 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. ISBN: 978-0-9671456-9-3 For information contact: Lyle McDonald Publishing 1200 Hatteras Drive Austin, Tx 78753 Email: A portion of the sale of this book will benefit the Women's Sports Foundation, a 501c3 charity founded by Billie Jean King and dedicated to creating leaders by ensuring all girls access to sports. Acknowledgements A number of people contributed to the completion of this book that I want to acknowledge. Several including Keith Caskey, Adam Rafalowsky, J Fred Muggs and others helped me to get some of the research papers that I needed. Lisa Vickers suggested a title change and the book is better for it. I want to especially thank Eric Helms, natural bodybuilder, powerlifter and coach for not only giving me invaluable feedback on various chapters of this but also providing the peak week and making weight sections of this book. Finally, I want to thank the many, many women online who made me aware of female specific issues that I would likely not have thought to address if they hadn't. Preface Having been in the fitness field for nearly 20 years, it's been clear to me for quite some time that women face issues that men simply don't. They have more overall trouble with fat loss, seem to gain weight and fat more easily along with endless other differences. And while I had made observations regarding this over the years, I had never really examined it in any sort of enormous detail. But this lack of attention to the issue came to a head in 2007 which is when I can say that this book really started. I had a female trainee who I simply could not figure out. Her performance was all over the place in the gym with her coordination, strength and mood changing seemingly weekly. She'd hit personal bests one week and be unable to lift 60% of her best in others with smaller variations in other weeks. Her mood shifted constantly and she suffered from fairly debilitating PMS along with the typical female body fat issues. One day, in the midst of a tangentially (and unfinished) related project, I decided to finally get this worked out and to "solve PMS". I spent the day reading endless research papers and, without exaggerating, was done in about a day. At least in regards to her training, a basic model of what needed to be done fell right into place. While that should have been the start of the project it wasn't. I'd write my Stubborn Fat Solution (addressing women's bodyfat issues) shortly thereafter and essentially retire for about 8 years. In 2015, after a few years of craziness, I finally got back to work. About February of that year, I found out that one of my earlier books, A Guide to Flexible Dieting (written in 2004), had been plagiarized by someone claiming to have pioneered the concept. It wasn't even the first time I'd been plagiarized but this made me angry. Very angry. For little to no reason other than spite, I decided to rewrite that book. A lot of my thoughts had changed and, being 11 years old, it was a little rough around the edges. As I started that project, I realized that I needed to add a section and add a section and all of a sudden it had turned into a completely different book, one on general fat loss. I'd needed to write that kind of book for a while so that's what I began doing. I pulled information from my other books, from my website and suddenly what started as a basic rewrite had spiraled into a 400 page tome. My purpose, ignoring dealing with my anger, became to write the be-all, end-all book on the topic of fat loss. If it wasn't discussed in my book, it didn't matter or the concept didn't yet exist. As a secondary, and also anger driven goal, I wanted to give people in the field something to plagiarize for the next decade. They were going to do it anyway so I figured I might as well give them a comprehensive (and correct) resource to rip off. Women's Issues Which brings me in a very roundabout way to the book you hold in your hands. I had reached a point where I was about 90% done with the mega fat loss book, at least in its initial form when I realized there was one last topic I needed to discuss. A topic that I had promised a book on years previously but had avoided (consciously or unconsciously) as I knew the difficulty it would entail. Of course that topic was women's issues as they pertained to diet, fat loss and training. As trite as it sounds, it was clear that women are "not just little men" even if they are often treated as such by coaches, physiologists and the medical establishment alike. There are physiological, anatomical, neurological and psychological differences to begin with and that's just as a baseline. During the course of the menstrual cycle, changes in a woman's hormone levels cause large-scale shifts in a woman's physiology. Her insulin sensitivity, whether she uses fat or carbohydrates for fuel, her metabolic rate, hunger, propensity to store fat along with her strength, endurance, coordination, injury risk and almost any other topic you could think of all change. In contrast, men are basically the same every day. Originally I figured I could cover the topic in maybe a chapter or so. Hahahahaha. Not only would it have been totally unfair to relegate women's issues to one short chapter, it became rapidly clear that it was impossible. Women make up ~51% of the population and a single chapter wouldn't do even if I could do the topic justice in that few pages. I figured I'd expand it to the length of the other sections in that book, that I might get it done in 40-50 pages. How wrong I was. As I started writing that section and was putting up excerpts on my Facebook wall or in my group, women on both started going kind of nuts and clamoring for that information. They didn't want to wait for the tome to be finished and wanted it earlier rather than later. Given the general lack of information that was out there, and knowing my generally obsessive approach to projects, they knew anything that I wrote would cover the topic in a way that only I seem able to. Make no mistake, some of it already existed (I had personally read an older book by two Australian sports scientists/coaches on the topic that I am fairly sure nobody else has seen) and was on the web and I Googled what was out there myself to see what had been written. What I was found was either vague, incomplete or, in some cases, incorrect. And it then dawned on me that, while information on women would be included in the mega-project to one degree or another, the topic truly needed its own stand-alone book. I'd have to pull some information from the big book since I couldn't say "Refer to Chapter 17" from a book that hasn't been release but that was fine. And I started writing and researching and writing and researching. As I got deeper into the topic, the complexity would multiply seemingly exponentially. The menstrual cycle alone introduced complications in women's physiology that simply don't occur in men. Even here, there is an added complexity. The "normal" menstrual cycle really isn't with a great deal of variability. Any two women may have different cycle lengths and even the same woman may have her cycle length change from month to month. Women also show differences in terms of how their mood, hunger, etc. change with almost no two women having the same exact pattern. The cycle can also be disrupted. In oligomenorrhea, menstruation occurs infrequently. In amenorrhea (altogether too common in dieters and athletes), the cycle may be lost completely. Even that was only the tip of the iceberg due to the presence of what I call hormonal modifiers. Birth control (which is supremely complicated) is one of the most common but Poly-Cystic Ovary Syndrome (PCOS) along with the changes that occur around menopause are all important issues. There are endless other situations, disease states (some of which women are more susceptible to than men), that change the system but they are far beyond the scope of this book. But each of the above situations are subtly or not so subtly different from the others. The hormonal profile may change (or not) with a given reproductive hormone being relatively more dominant in terms of impacting or changing a woman's physiology. In contrast, a man's primary hormone is effectively a flat line that changes only minimally day to day and goes down gradually with age. Certainly levels of that hormone vary between men which may have implications for fat loss and training but these are really slight variations on a theme rather than being distinctly different physiologies entirely. Comparatively speaking, men are profoundly simple from a physiological (and some might say other) standpoint. The Writing While I was writing this book, I joked repeatedly that I had been putting it off for a decade and that's not really untrue. As I mentioned above, while I had recognized some of the issues purely from experience, I knew that delving into the topic thoroughly would be exhausting and I wasn't wrong. Covering even the general physiological differences would have been difficult enough but by the time the hormonal modifiers were added, addressing the topic in any degree of detail was overwhelming. A project that I thought I'd get done quickly and easily would rapidly spiral out of control. While I originally intended to talk about fat loss and training in the one book, I would end up having to split the information into two volumes (as I write this preface in September of 2017, the training book is only partially written). None of this was made any easier by the fact that I was basically starting completely from scratch in terms of even the terminology that is used. I was a man writing about a topic that I have no fundamental (and certainly not personal) familiarity with studying information that doesn't resonate with me on any sort of intuitive level. I don't have a menstrual cycle and the entire concept is fundamentally foreign to me beyond my observations of female trainees. I would find that many women don't really understand the menstrual cycle so what chance, as a man did I have? Over the next 2.5 years, I would manage to wrap my head around the topic although I would be lying if I didn't say it was exhausting. In podcasts I would describe it as soul crushing with no negative intention meant. The topic is simply overwhelming. At the same time writing this book has been immensely gratifying (finishing it perhaps moreso) Not only did it allow me to expand my knowledge base, both on women and physiology in general by an enormous amount, but I also knew that I was ultimately contributing positively to the field, in a way that had never really been done before with this book. This Book This book is a book about women's physiology, diet, nutrition and fat loss. As I mentioned above, originally it was meant to cover training issues but doing those justice will require a second volume. In it I will look at what may seem like an endless number of topics. This will include a woman's general physiology, focusing on the menstrual cycle itself and what changes are occurring throughout it. Since they are so common, the hormonal modifiers will be discussed in some detail in terms of how they impact on or alter a woman's physiology. For background I'll look briefly at exercise types and some of the common goals a woman might seek To ensure that readers are clear on certain concepts, I will look at some fairly general topics such as body composition (what it is, tracking it and altering it) along with the issue of energy balance and metabolic adaptation. The next sections of the book will look in detail at how women utilize different nutrients, store and mobilize fat along with the potential differences in fat gain and fat loss. Since it is such a critical issue, I will address the topic of menstrual cycle dysfunction in some detail along with a large chapter on stress (a place where women and men differ drastically). This will lead into a chapter where I look at how to fix the various issues women face. The remainder of the book will be aimed at providing practical recommendations on a number of topics with a primary focus being on dieting and fat loss as that is such a prevalent goal for women (it is also a place where there is simply a staggering amount of either incorrect or outright damaging information present). This includes setting and adjusting calorie levels, determining nutrient requirements along with nutrient sources, fluid intake and others. I will discuss the concept of flexible eating strategies (originally discussed in the 2004 book that indirectly led to this one), around workout nutrition, meal frequency and patterning and finally supplements (including phase or hormonal specific supplement recommendations). I will also talk about estimating dieting times, identifying and breaking the inevitable dieting plateaus and adjusting the diet over time. Since training is a critical part of fat loss, I will address it in brief, once again a full discussion of women's training issues will have to wait for Volume 2 of this series. Finally I will provide hormonal templates for each of the potential situations a woman might find herself in along with diet templates for women of different starting body fat. Since lean females have the most issues, they will get their own chapter along with an examination of what should happen when and if they develop amenorrhea. Since recovery from amenorrhea is absolutely critical, I will spend a chapter discussing how that is optimally done. In many places in this book, I will be addressing some of the mistaken ideas that exist for women in terms of dieting and fat loss. There is simply a tremendously large amount of mistaken beliefs and information on the topic. And the recommendations, which as often as not come from men (especially in the athletic realm) are either ineffective or damaging to a woman's health. And while I will, as often as not, compare a woman's physiology to a man's (primarily to differentiate women), the fact is that this is a women's book. Others have been written (and I've read them all) but they tend to be either clinical and aimed at researchers or, frankly, simply aren't that good. Many are incomplete and it's not uncommon to find flatly incorrect information in them (based on the research available). While this book may have started out as part of another book before becoming what was meant to be a relatively small side-project, it morphed over 2.5 years into something completely unique. It's not just a general guide to women's physiology as books of that nature exist. Rather it exists as a comprehensive guide to not only women's unique physiology but what implications that uniqueness has in terms of optimizing her nutrition, diet, health, etc. Finally, the book I promised years ago is here. Well, Volume 1 anyway. An Important Note About This Book While I have tried to make this comprehensive regarding women in different types of hormonal situations, there are several topics that I have chosen not to cover. The first are any medically based pathophysiologies. There are far too many of them to address in any type of detail, it is an area far outside of my expertise and which should be treated by a health practitioner or OB/GYN in any case. I will mention the occasional issue in brief but, beyond that, the topic will not be addressed. While I will address some of the changes that occur throughout a woman's lifespan (especially the menopausal transition), I will not explicitly address girls between puberty and the age of 18. Certainly much of the information in this book applies in terms of good nutrition, improving bone mineral density and the consequences of things such as amenorrhea (Common in young female athletes) but there are too many issues involved with the growing female that I have neither the expertise nor ability to cover. Similarly, I will not be covering the issue of pregnancy or breast feeding. Women's hormones go through enormous changes at this time with some of their effects effectively reversing in some ways and addressing that in detail would be impossible. Of more importance, I am both unqualified and unwilling to provide dietary advice on either situation. The developing fetus and newborn are too sensitive to changes in a mother's food intake and the idea of giving suggestions that might cause harm fills me with dread (I will mention pregnancy in one or two places, however). There are already numerous books on the topic, written by people far more qualified than I available and I would recommend that women interested in the topic use them as a resource. Finally is the topic of eating disorders (ED's). While absolutely relevant to the topic of women, diet and fat loss, it is another topic that requires professional help and intervention rather than advice from a book (no matter how well researched). As with pregnancy, I will mention it once or twice in this book, primarily as it pertains to other topics, but I will not address treatment or recovery. A Few Qualifications In the modern world, discussions of sex or gender can be problematic for any number of reasons and I want to make some qualifications about the language and concepts that I'm going to discuss first. This section may seem excessive or pedantic but I want to make absolutely sure that none of what I will write throughout this book will be misconstrued. First and foremost, in terms of their specific meaning sex and gender are not identical concepts. Sex refers to an individual's biology in terms of their genetics and which reproductive organs are present. In contrast, gender refers more to the roles an individual plays in society or how they self identify. Someone with female reproductive organs (female by sex) might identify as a male (male by gender) or perform what are traditionally referred to as a male gender role. The opposite can hold true and there are many more possibilities than just those two (my choice of that example is not meant to be exclusionary). That said, in the scientific literature, and certainly among a majority of the lay-public, sex and gender are used interchangeably, with scientific researchers showing no real tendency towards one or the other. This is especially true in physiological research which is what most of this book focuses on. While acknowledging that it is technically incorrect, I will do the same throughout this book using the terms sex and gender or sex differences and gender differences synonymously. In most cases, I am likely talking about sex differences since my focus is on physiology and biological differences but I will still use both at varying times. I just want to make it clear that I am in no way dismissing or denying the differences. In a similar fashion, I may occasionally refer to female-like or male-like characteristics or personality traits. With no intent to imply or maintain traditional gender roles, this is simply a descriptive shorthand that I will be using since I expect readers to be familiar with what the terms have traditionally represented. Clearly, women can show what are traditionally thought of as male-like characteristics (in terms of behavior or personality) and vice versa and there is a tremendous range of behaviors that might be seen between any given extreme. Again, it's nothing more than a descriptive shorthand since most know what the terms refer to and I use it only for convenience. About the Title This book has gone through a number of title changes. My first working title was "50 Shades of Hormones" which became "More than Just Little Men" but I was thankfully convinced that neither were appropriate. While I frequently compare women and men's physiology throughout this book, my primary goal was simply to point out those differences and nothing more. To compare women to men in the title misses the point of what this book is. That is, this is a book about women and the differences and situations that they face. Hence the change to simply "The Women's Book" which describes exactly what it is. Chapter 1: Introduction to Women's Physiology While this is meant to be a book about women's specific physiology and how that impacts on diet, nutrition and fat loss, to at least some degree it will be a book about the differences between women and men. This is a topic that is sometimes dangerous to discuss as, to many in the modern world, it smacks of inherent sexism to even consider that there are any differences between women and men. Certainly in the past, the idea that there were gender differences got co-opted into the idea that one gender was superior or inferior to the other. Since it was usually men who were writing about this, and since they tended to assume that they were the default setting, the idea that women were different from men came to mean that women were inferior to men. Many seem to feel that to discuss or even suggest gender differences is to tie into what some see as inherent male-dominant (or androcentric) sexism. I think this confuses issues. In my mind, the concept of a difference is no way implies an inherent inferiority or superiority even if many interpret it that way. Even in the case where women and men are different, there are clear places where women show a greater response than men and others where they show a lesser response. In most sports, at the elite level, women's performance is about 8-10% below men. However, in ultra-endurance running and cold water swimming, women's performance is generally superior. Women also show better endurance an tolerate heat better than men. While they often lose fat more slowly, they also lose less muscle than men. Regardless of whether the response is better or worse, I will simply consider them to be differences going forwards and nothing more. Honestly, the only reason to address them as differences (rather than simply focusing on women's physiology) is that so much of the information is based on men with the flawed idea that it automatically applies to women. But even this raises the question of why it took so long to even recognize that there were differences between the two. Scientific Research: Part 1 For quite some time, it was basically assumed that research on men, and this cut across most disciplines including general physiology, exercise, fat loss, etc. would apply directly to women. It just wasn't really questioned on any level. In the realm of sports performance, it wasn't until about the mid 80's that any amount of comparative studies on women and men started to be done (1). But even from the earliest research, it became clear that there were significant differences. A singular example will hopefully make the point. Endurance athletes such as runners or cyclists will often use a dietary strategy referred to as carbohydrate loading where they combine intense exercise with a drastically increased amount of carbohydrate in their diet. The goal of this is to increase the store of carbohydrate in the body (muscle and liver) to improve performance. Early studies showed that this worked well for men. They increased the storage of carbohydrate in their bodies and their performance improved. But in women it didn't seem to have the same effect. In one comparative study, while men increased their muscle carbohydrate stores when fed a 70% carbohydrate diet, women did not. For fairly logical reasons, biological differences were assumed to be the case since, as often as not, it does explain the differences that are seen. But this was at least partially wrong. It turned out that part of the reason the female subjects didn't carb-load as well as the men was due to the fact that the same 70% carbohydrate diet provided much smaller amounts of total carbohydrate due to the women being smaller and having a lower energy expenditure. That is, a woman burning 2000 calories per day and eating 70% carbohydrate is getting 350 grams of carbohydrate while a man burning 3000 calories per day and eating 70% carbohydrate is getting 525 grams. The percentage is identical but the total amounts aren't. When women were fed equivalent amounts as the men, most of the differences went away When both women and men were given even larger amounts, there was no gender difference in carbohydrate loading. It had purely to do with the total amount of carbohydrate. But this raised another problem, it created an impossible diet for the women. To get enough total carbohydrate meant eating too many total calories. Their smaller size and energy expenditure basically made it impossible to achieve what men could without eating too much food. There was another issue. Many females are restricting their food intake to one degree or another for various reasons. If they need or want to lose fat, they have to eat less than they burn and that may not leave enough total food to support their training. There are solutions to this, what some call nutritional periodization (alternating time periods of restricting food intake with increasing it) and I'll talk about them in a variety of contexts later in this book. But even this singular example brings up one of the key problems that often shows up which is that dietary approaches that work for men (or are used by male coaches) don't work or prove impossible for women to implement. There will be more examples of this throughout this book. 1 Scientific Research: Part 2 So why did it take so long to include women in research, especially in sports science and exercise research? Some it probably represented pure chauvinism: the majority of scientists were male and they tend to bring a male-oriented mentality to things. But perhaps a bigger part of it was that for the first half of the 20th century, and even into the 70's, women simply weren't as involved in sport as men. In certain sports, it was long thought that women were incapable of performing them or that they would do physical damage to themselves. Up through the 70's, for example, women were barred from even competing in marathons (there is a famous picture of referees trying to pull a woman off of the course). Some of this mentality persists today as many sports still maintain shorter distances or slightly different events for women versus men. A lot of it was sociocultural as well; there was a lot of social pressure against women entering sports. It was seen as being unfeminine (or outright masculine) and simply remained mostly a male domain. This started to change in America in the 70's with the passing of Title IX which required similar sporting availability for women. And as they started to enter sport, gradual changes started to occur. I'd note that this wasn't true in many other countries. Due to the status for winning at the world and Olympic level, females were both allowed and encouraged to compete. In the 80's, for example, the German Democratic Republic (GDR) women were absolutely dominant in swimming and track. But the point is that most research in sports science was done on men because men represented the majority of people involved in sport at the time. There wasn't much reason to study women since they didn't make up a large proportion of athletes. Even when research was done either on women or to compare women to men, early research was poorly done and the results were questionable at best. The reason for that has to do with the incredible complexity of women compared to men which due to the menstrual cycle. This is the roughly 28 day cycle a woman goes through monthly during which her hormonal status and physiology can change in subtle or not so subtle ways. The cycle is typically divided evenly into two phases called the follicular (from menstruation to ovulation) and the luteal phase (from ovulation through PMS) and each is distinct from another. There are even shifts that occur in the earlyand late- phases of each and some even divide each phase into an early-, mid- and late- phase. During this cycle, a woman's primary reproductive hormones (estrogen and progesterone) show complex overlap. This makes studying women incredibly difficult. Researchers have to control for the phase of the cycle itself and even this can be difficult as determining where in her a cycle a woman is isn't always easy (recent studies will use ultrasound and blood work to determine this but this is not easy or cheap). Add to that that no two women have an identical menstrual cycle and even an individual female's cycle may vary from month-to-month. A woman who starts menstruating a day early might have to wait a month to be retested. To begin to compare women to men, you must control for the phase of the cycle. But when you study her may change what conclusion you reach. In one phase a woman's response may be identical to a man's; in the other it may be different or even the compete opposite. As a singular example, women's metabolism of caffeine is similar to men's during the first half of her cycle but different in the second half. If you don't study all of those conditions, you can draw incorrect conclusions and early studies didn't even pay attention to where in her cycle a woman was. She'd come to the lab to be measured and that was it. The results were meaningless. In contrast men have one primary reproductive hormone which changes very little day-to-day. So long as you control a few simple variables like time of day and whether or not the eaten, you can test them any day of the month. Ultimately, it's just simpler to study men. An Amusing Exception There is an amusing exception to the above that I only mention for completeness, a place where by and large there is relatively more research on women compared to men and that is in the realm of diet studies. Here, women tend to be drastically over-represented, making up the majority of subjects. This is primarily reflective of the fact that women are far more likely to be dieting than men. Hence they are more likely to enter the studies. For example, there is a database of successful weight losers called the National Weight Control Registry (NWCR) and it is composed of roughly 80% women. In the same way males represented the majority of athletes in the 70's, women continue to represent the majority of the dieting population now. While I mention it only as a point of trivia, the reality is that a great many weight loss approaches, studies, etc. are either done exclusively or predominantly on women and there has been some recent question whether or not this information applies equally to men. Dieting, diet groups, etc. are often seen as a "woman's domain" and men may be less likely to pursue either for a variety of reasons. To address this, researchers have been working on more "male oriented" weight loss programs including male humor (which I take as fart and poop jokes) and sports team affiliation (2). 2 Gender Differences: Introduction Regardless of all of the above, it's abundantly clear that there are distinct differences between men and women and they cut across nearly all domains including those covered in this book such as diet, nutrition and fat loss (exercise and training being covered in Volume 2). Let me state up front that the differences I will be discussing, especially many of the physical differences represent no more than averages. Usually researchers study a huge number of subjects and look at the average response. But an average response says nothing about any given individual and it's trivial to find an exception to just about any topic I will discuss. For example, despite the fact that women are, on average, shorter than men, clearly you can find a woman who is taller than a man. Despite the fact that the average woman typically has narrower shoulders and wider hips than a man, you can find a woman with narrower hips and wider shoulders. As frequently as not the variation within a gender is actually greater than the difference between genders (3). And at least in some area, women may show even more variability than men. Throughout most of this book when I address gender differences I will put it in terms of "Women show such and such of a difference compared to men." and I want to address my choice of that phrasing. A recent book about the Chinese female Olympic Lifting team makes a fairly impassioned point that using men as the baseline for sports performance represents not only the history of the idea of men being inherent superior but an androcentric (male) viewpoint overall (4). That is, typically speaking, women are compared to men in terms of their physiology, biomechanics, response to training, etc. instead of the other way around. And strictly speaking it would make just as much sense to reverse it. For example, women tend to handle heat better than men (at least during some phases of the menstrual cycle) and it would be just as accurate to state that "Men handle heat worse than woman" as "Women handle heat better than men." That said, when examining gender differences I will still discuss them in terms of a woman's response relative to a man's. There are three reasons for my choice in this. The first is that is a book about women's issues related to nutrition and fat loss. To describe differences in physiology in terms of men's responses relative to women would make little sense since it is the woman's response that is important The second is that, with one odd exception discussed above regarding dieting studies, staggeringly more research has been done on men relative women (even now research on men is done at about a 4:1 ratio to that on women). It's only been fairly recently that women's response, or explicit gender difference studies have become more common place. And since there is far more research on men, comparing women's responses to men's makes logical sense to me here. Finally, and perhaps most importantly from a practical sense, the fact is that a majority of ideas about nutrition and fat loss that are applied to women typically come out of either research or practice on men. Since most athletes have traditionally been male, most coaches have as well. This is changing in modern times and there are more and more female coaches (usually coaching women) but many of the ideas and approaches to training, exercise, dieting and fat loss come out of male-oriented approaches which may not only be ineffective but damaging. Addressing that means comparing women to men to some degree. A Snapshot of Gender Differences In no particular order of importance, here is a brief look at a few of the (again, average) differences between women and men. On average, women are lighter, with less lean body mass and more body-fat than men. They also carry their fat differently with a more lower-body fat patterning. Their bodies utilize protein, carbohydrates and fats differently than men both at rest, after a meal and during exercise. They regulate what is called energy homeostasis differently than men (ultimately sparing the loss of body fat). Physically, women's wider hips alter their knee biomechanics (predisposing them to certain kinds of injuries). Women tend to be more flexible with relatively more mobile tendons and joints. And while women's muscles are physiologically identical to male's for the most part, there are differences in how they generate force or fatigue in response to exercise. As I mentioned above, on average female athletes perform at a level roughly 10% below that of men in most sports (with two exceptions). While women typically start out with lower levels of fitness than men, they respond similarly if not identically in terms of the relative improvements that they make in response to training. Perhaps the most major difference between women and men has to do with hormones, especially the existence of the menstrual cycle. Discussed in detail later in this book, this represents the roughly monthly cycle of hormonal variations that a woman undergoes (in contrast, a man's hormones are relatively stable across the month). This changes her physiology at a fundamental level and introduces a complexity that simply isn't seen in men. This can also be modified in a stunning number of ways with what I will call hormonal modifiers changing a woman's physiology subtly or not so subtly. 3 The Cause of The Differences This raises the next important question which is what the genesis of these differences is. Of some interest is the fact that most parts of a woman and man's bodies are actually identical in a physical sense. Under a microscope, a woman's bone is the same as a man's bone in terms of it's cellular structure, it's just not as dense. A woman's muscle is almost identical to a man's in terms of it's cellular structure, it's just typically smaller (there are other small differences I won't get into here). A woman's heart, lungs, etc. are all identical in cellular structure (albeit smaller) with the largest physical difference arguably being in the genitalia. Of course there are clearly differences that appear in terms of the relative amounts of fat, muscle, etc. So if the underlying structures are more or less identical, why are these differences seen? Whenever a fairly large scale difference between women and men shows up, it's been traditional to assume that it is due to underlying genetic/chromosomal or hormonal differences. In recent years, the idea that all physical differences between women and men, especially the differences in strength or sports performance, are culturally generated is being more commonly heard. As usual, the truth lies between the two extremes. Certainly it would be absurd to dismiss the role of environment or society and culture on women's, or for that matter anyone's, overall nature. But it's equally absurd to dismiss the differences between the sexes in terms of biology. And since this isn't a book about society or culture, it is the biology that I will primarily focus on. Because not only are those changes significant, it's actually the case that many of them are set in place before birth, before any social or cultural influences are present. A woman and a man's genetic code differs, readers may remember that woman have XX and men XY chromosomes which are a huge part of what "tells" the body how to develop (especially in terms of the reproductive organs). Related to this, in recent years there have been issues concerning women in sport and biological sex testing. As this topic has more to do with emotion and political agendas rather than biology or physiology at this point, I am not going to begin to touch it in this book. The Role of Hormones Other than genetics and the difference in genitalia, arguably the largest biological difference between women and men is in the relative amounts of the primary reproductive hormones. In women, these are estrogen and progesterone and in men it is testosterone. Both sexes make all three hormones but the relatively amounts in adults differ significantly. On average women having roughly 1/10th to 1/30th the testosterone levels of men; men have similarly low levels of estrogen and progesterone. It would be patently absurd to pretend that this isn't impacting on the physiology of a woman versus a man directly in addition to any interaction with aspects of her genetic programming. A singular example is that women's bodies genetically form more of what are called pre-adipocytes (think of these as baby fat cells) in their lower bodies. These pre-adipocytes are stimulated to become fully formed fat cells and this occurs under the impact of hormones estrogen and progesterone with estrogen specifically impacting on the development of lower body fat cells in women (5). Even that doesn't occur until what might be described as one of the most profound times of life for both girls and boys occurs which is puberty. Prior to that, little girls and boys are more or less physically and physiologically identical. When there are differences they tend to be small and are generally in the same direction I mentioned above (i.e. girls are still slightly shorter or carry slightly more fat). But it is at puberty, when the reproductive organs become active and hormonal levels diverge enormously that the primary physical and physiological differences between the sexes appear . A woman's specific physiology develops under the effects of estrogen and progesterone as does a male's under the influence of testosterone. Women develop their traditional body fat patterns, increasing not only total body fat but lower body fat specifically while men develop more muscle mass while losing fat. Effectively puberty is when the typical feminine and masculine physiologies develop and let me reiterate (for those who skipped the preface) that I am using these terms only as descriptive shorthand with no implication about whether or not one is a relatively more or less appropriate or superior gender role than the other. We all know what these terms refer to and it's just easier writing it this way. This is further shown by the fact that even small changes in hormones can have a profound impact. Even small changes in the levels of testosterone in women can drastically impact on her physiology, effectively masculinizing her in many ways (the effect is even more pronounced in women who use anabolic steroids, derivatives of testosterone).. I'll talk about specific examples of this in later chapters and other factors that change or modify a woman's overall hormonal profile drastically impact her physiology. Menopause is one of the most profound of those as through the process peri-menopause through the menopausal transition itself, her hormones drop from their youthful levels to nearly zero. 4 None of the above is meant to in any way dismiss the role of culture and environment in this. Clearly it plays a role and to ignore it is a mistake. When I talk about training and injury risk in Volume 2, I'll talk about a very specific place where culture/environment (specifically women's typically lower involvement in sport at a young age) interact with her biology in a profoundly negative way. For now I want to briefly discuss a topic I said I wasn't going to talk about much in the preface. Eating Disorders Perhaps no more clear an example of the above lies in an issue of enormous importance for women that I said I wouldn't discuss in this book but should at least address and that is the topic of eating disorders (EDs) such as anorexia, bulimia and others. Because, depending on what statistics you see, women may be anywhere from 3-9 times more likely to have a true ED as a man. Unbelievably, some studies indicate that 95% of true ED's are seen in women which is simply enormous (6). Quite in fact, for some time it was actually thought that ED's occurred only in women. We now know that this is false as relatively more men are developing some form of ED in the modern world. Presumably this due to increasing pressure for men to meet the same type of physical ideal that women have faced for far longer (8). Even here there can be a gender difference. Women's ED's tend to revolve around the quest for extreme thinness while men's are often focused on increased muscle mass, although extreme leanness can also be a goal. Similarly pathological behaviors are often seen here as well. The idea of bigorexia or body dysmorphia is being used to describe males who, despite objectively being muscular and large, still see themselves as small, unmuscled and weak. This is fundamentally no different than the anorexic who still sees themselves as fat despite objective evidence to the contrary. It's just working in the opposite direction. But focusing on women, there is absolutely no doubt that environmental and cultural issue that play into this with factors such as a mother with an ED, early dieting practices, stressful environments, being teased about body weight, media images or being involved in traditionally female sports such as gymnastics, ballet and ice skating that stress thinness and low body weights that are present (9). But most overt ED's occur in women under the age of 24 and tend to develop at puberty which suggests a biological factor. And that biological factor, simply, is estrogen more specifically the increase in estrogen that occurs at puberty which is when the majority of true ED's develop (10). It's clearly the interaction of a woman's biology along with a certain environment causing one of the most potentially damaging effects and one that is seen nearly exclusively in women. But the hormonal effects are simply incontrovertible and the changes that occur at puberty are what lead to the physiological, physical and other changes that are seen. Prenatal Hormone Exposure But it actually goes even deeper than the above. A great deal of a person's biology is actually set in place during fetal development. A pregnant woman's diet, environment and other factors can drastically and permanently impact the risk of disease including obesity, diabetes and even brain development in the developing fetus (10,11). There is a lot of recent concern in this regard over environmental compounds (such as environmental estrogens) and the impact they may be having on biology and I'll actually talk one specific issue relative to dietary supplements later in the book that is important here. The above is not strictly a gender difference since it can occur in both women and men. However, there is an effect of reproductive hormones, in this case the relative levels of testosterone and estrogen that the fetus is exposed to that not only plays an enormous role in the final physiology seen but can act to have a relative masculinizing or feminizing effect that is seen later in life. Prenatal hormone exposure expresses itself externally in what is called second to fourth (2:4) digit ratio: the ratio of length between the index (second) and ring (fourth) finger. When exposed to high levels of testosterone, the index finger is typically shorter than the ring finger (a male-like pattern). When exposed to lower levels of testosterone, the index finger is equal or longer than the ring finger (a female-like pattern). And while this ratio doesn't seem to show any relationship to adult hormone levels, it is clearly related to prenatal hormone exposure (11). A female exposed to relatively more testosterone may undergo different biological "programming" than a female exposed to less, developing relatively more "masculine" characteristics physiologically. The same occurs in reverse in men; men exposed to less testosterone may develop a more "feminine" physiology, body structure and set of behavior patterns. This ratio ends up being predictive of many factors such as bodyweight, waist to hip ratio (here there are common female and male patterns) and health risk (12). But there is also a strong relationship between the male-like finger pattern (again indicative of relatively more testosterone exposure in the womb) and traditionally masculine behaviors. Hopefully a few examples will suffice (and there is fairly endless research on this). 5 In general, the more male-like digit ratio correlates strongly with the typically seen differences between women and men in behavior, being related to such behaviors as aggression, thrill seeking and attention seeking (13). A male-like digit ratio is associated with better athletic performance in at least some sports, presumably those requiring higher levels of aggression and other masculine characteristics (14). Both men and women with a male-like digit ratio have more masculine faces in men this is associated with attractiveness in women (15,16). In contrast women with a more male-like digit ratio are rated as less desirable and less faithful than those with the female-like digit ratio (17). It's interesting to note that in at least one high-level sport, coaches specifically select female athletes by looking for a more male-like facial structure, smaller breasts and less triceps fat as they feel this corresponds with higher testosterone levels and better chance of sporting success (18). As well, and this is relevant to training issue that I will discuss in Volume 2, digit ratio may predict what adult preferences are shown (19). Women exposed to higher pre-natal testosterone show different preferences in terms of activities, toys, etc. that they are drawn to (20,21). Digit ratio also interacts with adult hormone levels. Women injected with testosterone show increased levels of aggression and decreased empathy (both traditionally masculine behaviors) but only if they have the more male-like digit ratio to begin with (23). In this case, the effects of pre-natal hormone exposure and programming determine how a woman's biology will be impacted by changes in her hormones later in life. Returning to the topic of eating disorders, Binge Eating Disorder (BED) which is far more prevalent in women is thought to be related to the combination of a relative lack of androgen exposure during fetal development that puts a woman at higher risk for developing BED at puberty when estrogen levels increase (24). All of this sets up an enormously interactive situation. Prenatal hormone exposure may have a relatively masculinizing or feminizing effect on women in terms of biology, body structure and personality which leads them to prefer certain types of activities, which they are then prepared to succeed in. But it's the interaction of factors with my only point being that there are clear and enormous impacts of not only the differences in reproductive hormones between women and men but that exposure to those hormones has an impact on all aspects of woman's physiology before birth or puberty. In this vein and to get back to the topic of this chapter a relatively informal piece of Russian research found a strong correlation between a relatively crude measure of personality called the Bem Sex Role inventory in terms of ratings of femininity, androgyny and masculinity in terms of what types of sports they were found in (25). Women with higher ratings of masculinity were more likely to be found in sports requiring more strength, power and aggression while higher ratings of femininity predicted the opposite effect. Women who rated for androgyny were in the middle. The more masculine women could be trained more like men due to their prenatal biological programming and adult hormone levels for this reason. As well, they were less likely to be impacted by the changes that typically occur with the menstrual cycle. Because, as I mentioned, in addition to genetics, hormonal changes at puberty, and clear cultural influences, there is still the presence of the menstrual cycle. Scientific Research: Part 3 In addition to the different levels of hormones seen between women and men, there is another factor that makes the study of women so much more complicated which is the presence of the menstrual cycle. Because, in addition to making observations, about what the differences may be from phase to phase or to men, researchers usually want to know what is causing those changes. And here the incredible swings that occur in the menstrual cycle in estrogen and progesterone can make determining what is causing what to occur very complicated. Both hormones rise, fall, overlap and interact. When looking at any given response, is it the increase in one, the decrease in the other, the combination of the two changing or the ratio of the two? It can be excruciatingly different to determine this. Frequently researchers use animal models where it's easier to control their hormone levels but this raises issues of whether or not they make a good model for humans. Usually they do not and in some cases the effect of one or the other hormones in animals is the opposite of what is seen in humans. Another interesting approach is to inject a woman's hormone (usually estrogen) into a male to see if the same effect is seen. Often it is. But sometimes the effects are different are reversed (for example, increasing testosterone levels in women often has the opposite effects as what is seen in men). Usually what is done is to first shut down a woman's hormone production completely. Then either estrogen, progesterone or some combination of the two is put back in to study the effects under isolated or at least controlled situations. And while there were endless questions as to which hormone or what hormonal interaction was causing what effect in women, in recent years, the picture has become far more clear. 6 Adding even to that, women have a number of what I will call hormonal modifiers that change the picture further. The normal cycle can be disrupted either becoming longer than normal (called oligomenorrhea) or being lost entirely (amenorrhea). Many women use birth control (BC), either a combination of synthetic estrogen and progesterone (or just progesterone) of which there are a staggering number of forms and types all of which may act slightly differently. Many women suffer from Poly-Cystic Ovary Syndrome (PCOS) which is typically marked by higher than normal testosterone levels and there is a subclinical hyperandrogenism where a woman's normally low testosterone levels are higher than normal and both change her physiology drastically. A woman's physiology also changes throughout the lifespan around menopause where her reproductive organs start to shut down. First there is a peri-menopause (with an early- and late-stage) and menopause itself. That is modified by whether or not a woman chooses to go on Hormone Replacement Therapy (HRT), synthetic estrogen and progesterone. There is also a specific estrogen only HRT used in women who have had a partial hysterectomy. All of these situations in women are subtly or no so subtly different physiologically. To study women completely on any given topic means to study each of these situations separately. In some cases it's to see what the changes are from aging; pre-menopausal and postmenopausal women may be compared. In some cases, a normally cycling female may be compared to a woman using BC or one who is amenorrheic. Different types of BC may be compared. The possibilities are nearly endless. In contrast, males may have slightly lower or higher testosterone although some men produce more estrogen than others. Men with very low testosterone (hypogonadal) do need to be studied but even with age, a man's testosterone only goes down. It doesn't drop to near zero as occurs in menopause. Getting very off topic, this is why I think the concept of andropause, means to be equivalent to menopause is incorrectly named. In menopause, a woman's reproductive hormones go effectively to zero. In the supposed andropause, a man's testosterone is simply going down with aging. They are simply not the same. Implications for this Book It's a bit of a running joke that nobody understands women and there is much truth to this. As I have worked on this project and posted a great deal about it online, it's become clear to me that many women aren't familiar with the terms I used above, the phases of their menstrual cycle or how their hormones (much less their) physiology changes. Outside of the ob/gyn, neither are their health care providers and there the focus is primarily medical rather than being applied in terms of how it impacts on diet, nutrition, fat loss or training. It should be clear that if females don't understand the topic, men, whether coach, athlete, or significant other has no chance of understanding it. There are a few implications of this. The first is that, fairly obviously, a woman has to be treated at least somewhat differentially than a man when it comes to both her diet, training and fat loss. Make no mistake, the same generalities will always hold here. While I'll talk about training in detail in Volume 2, I'll I'd mention that improving fitness, strength, power or performance requires certain general aspects of training. It's the specifics of how those aspects are specifically applied that may differ in a woman than in a man. Women face issues that men simply never will, for example some women experience changes in their performance, coordination, etc. throughout the month. This means that their training may have to be adjusted to better synchronize with those changes. This will never apply to men. There are other examples and this is a problem as, in the same way males dominated athletes for a long period of time, most coaches have traditionally been males. They know what they did as a (male) athlete and usually know how to coach male athletes. Keeping with the topic of this book, one place this perennially shows up is when it comes to dieting and fat loss. Due to the differences in all aspects of their biology, some of the approaches that are effective for men are either ineffective or outright damaging to women (this is especially true in the physique subculture where strategies to diet down to the lowest extremes of body fat were originally developed on and for men). Additionally, since they are typically larger, men have more food and calories to "work with". This allows them to use strategies that end up being inappropriate for women or at least to avoid certain problems that female athletes run into (i.e. not having enough calories in their diet to get sufficient protein and fat and still have enough carbohydrate to sustain training). This fact typically goes completely ignored by most. Some writers and coaches (generally male) have at least paid lip service to the potential differences between female and male trainees but often they reach completely opposite conclusions. Some feel that training should or can be modified across the menstrual cycle (early Russian training literature even talked about this) while others argue that women should be trained effectively the same as men with the variations 7 being more about the individual athlete than being based on gender at all. Sometimes high-level coaches change from one stance to another but this tends to go along with the use of anabolics in sport. With high enough doses of testosterone in women, the menstrual cycle usually disappears and women can be trained just like men. Even without drugs, women with relatively higher levels of testosterone (often seen in PCOS but also in other situations) can often be trained relatively more like men and, as I'll discuss later in the book, tend to show superior performance. Quite in fact, one of the dirty little secrets in women's physique sports such as bodybuilding, physique or figure is that there is often some degree of low-level drug use occurring. This might include anabolic steroids (to increase muscle size or hardness) and various fat burners that help the athletes to get into contest shape while avoiding the problems that natural women face. Since they are successful, much of the idea about how dieting and training should be done comes from them which leaves women who are not using those compounds with a very poor idea about how they should approach their diet or training. All of the above is made further complex by the variation between women. Certainly two men may show differential responses to diet, fat loss and training but, in the aggregate, it's more a matter of degrees than anything else. Two women may show differences from one another and any given woman may vary from month to month. Looking at training and considering only the menstrual cycle, one woman may see her performance vary enormously while another has no such change. Changes in mood during the final week of the cycle (i.e. when PMS typically occurs) are monstrous. One woman may have no issues while a second might have severe mood swings or fatigue and a third may be completely physically debilitated and suffer from clinical depression. The patterns of mood swings can vary as can any other aspect of a woman's physiology. While some of the hormonal modifiers actually work to stabilize this, they still change a woman's physiology to some degree and add another level of complexity. And one that no male or coach training a male will ever have to face or deal with. And that is really the crux and goal of this book. I want to not only look at the differences in physiology between women and men and to identify places where a woman's response differs in some fundamental way which may make an approach that is effective for men be less so for women. More importantly, I want to address those differences in terms of what changes a woman should (or should not) make in how she approaches reaching her goals. To help readers not only understand how the physiology of women and men differ but how a woman's approach to diet, nutrition, fat loss or training (again, in Volume 2) should differ because of that. And to begin to understand that means starting with a discussion of arguably the primary factor that differentiate women from men. That is not only the differences in the primary female reproductive hormones from that of men's but in how they change across the monthly menstrual cycle. 8 Chapter 2: The Normal Menstrual Cycle In the last chapter, I not only looked at some of the problems that have plagued both studying women and comparing them to men but looked, in brief, at some of the clear gender differences that have been found to exist in aspects of their physiology. While there are other reasons that I will discuss later in the book, a great deal of those differences are due to the relative levels of hormones, primarily the reproductive hormones, present in women and men. This is further complicated by the changes in those hormones that occur on a roughly monthly basis which act to change her physiology. And while hormonal modifiers exist that further complicate this system, understanding any of them means first understanding cycle itself. In this chapter, I want to sketch out the normal menstrual cycle from start to finish, primarily focusing on the changes that occur in estrogen, progesterone and testosterone in terms of levels and how they impact on her physiology. I will also look at several other hormones that vary to one degree or another in women and men and which are important to various aspects of this book. One hormone that I will not discuss in this chapter is cortisol, a stress hormone that is so crucial to understanding many of the issues that women's face that it will be discussed in a separate chapter. A note on terminology: Strictly speaking there is no "normal" menstrual cycle It varies in duration between women and even the same woman can differ from month to month. In using the term "normal", I in no way mean to imply that any other pattern is abnormal. It's just a descriptive shorthand for the typical cycle of hormonal changes that occurs under standard situations. It's modified enormously by other situations but it's just easier to call it "normal" for writing purposes. An Overview of the Menstrual Cycle The menstrual cycle derives from the Latin word for month and refers to the roughly one month (28 day cycle) that a woman goes through from the time she enters puberty until she loses her cycle at menopause. While I will treat the menstrual cycle as if it were exactly 28 days, a normal menstrual cycle may be anywhere from 24-32 days in duration (quite in fact, while most women report a 28 day cycle, few actually have a 28 day cycle). The primary purpose of the menstrual cycle is to prepare for the potential of pregnancy and most of the hormonal changes and their effects are aimed at this goal. When the menstrual cycle is functioning normally, this is called eumenorrhea ("eu" means well or good). By convention, Day 1 of the cycle occurs at the onset of menstruation (i.e. her period), the bleeding that occurs as the uterine lining (thickened in preparation for implantation of the egg) is expelled. This typically lasts 3-5 days. From this point, the remainder of the cycle is divided into two distinct phases, described next. The Follicular Phase The first half of the cycle is termed the follicular phase and within a 28 day cycle will typically last 14 days. Strictly speaking this can be divided up into an early, mid-, and late follicular phase of roughly 3-5 days each but I'll only use early- and late follicular in this book with the split happening halfway through (i.e. day 7). In research, women are frequently measured during the mid-follicular phase to get a more or less average indication of what is going on physiologically. Under certain conditions, the follicular phase can lengthen as well. During this phase, the follicle (hence the name) develops due to the effects of Follicle Stimulating Hormone (FSH). Technically, multiple follicles develop but only one releases an egg. Hormonally, progesterone remains very low during the follicular phase. Estrogen starts at a low level, shows a gradual increase leading up to a large surge in the final few days of the cycle. This surge causes a follicle to burst open, releasing an egg from the ovaries which then implants itself into the lining of the uterus (thickened to provide nutrients in the case of pregnancy). The surge in estrogen also causes a thickening of vaginal mucus, which makes the vagina less acidic and more hospitable to sperm. Women have often used this thickening as an indication of their fertility. The Luteal Phase The release of the egg on day 14 (the halfway point of the cycle) is termed ovulation and this marks both the middle of the menstrual cycle along with the end of the follicular phase (testosterone spikes briefly at this stage as well). At this point, a woman enters the luteal phase which can again be subdivided into early-, mid- and late phases. As with the follicular phase, I'll only use early- and late luteal phase in this book. As in the follicular phase, researchers often measure women in the mid-luteal phase for consistency and to obtain an average response in terms of physiology. 9 During this phase, the follicle which released the egg develops into a structure called the corpus luteum (hence the name luteal phase) which starts to produce the hormone progesterone. Over the first half of the luteal phase both progesterone and estrogen increase gradually reaching a peak at mid-cycle. Progesterone levels are higher than those of estrogen, which only reaches about half of the level seen during the peak of the follicular phase. Body temperature also increases slightly (about 0.2°C or 0.4°F) after ovulation and basal body temperature (BBT) can be used not only to tell when ovulation has occurred when pregnancy is the goal but is can also be used to tell when the follicular phase has ended and the luteal phase has begun. Along with this increase in body temperature is a slight increase in metabolic rate which I will discuss in more detail later in the chapter.. Late Luteal Phase In the late luteal phase , progesterone and estrogen start to drop again and this is the when PreMenstrual Syndrome (PMS), if present, typically occurs. PMS can be marked by an enormous number of symptoms including cramping (as a woman's body prepares to shed the uterine lining) mood swings, low energy, depression, breast tenderness and others (this is called dysmenorrhea). In extreme cases, women may experience debilitating pain from cramps, depression, anxiety or suicidal thoughts (often requiring medication) and this is referred to as Pre-Menstrual Dysphoric Disorder (PMDD). Depending on the source in question, PMS is reported to occur in roughly 30-40% of women with clinical relevant PMS occurring in 20% and PMDD occurring in 5-10% of women. Cramps per se are reported in 45-95% of women with 3-33% of women being physically incapacitated due to them (1a). While cramping is often thought to occur primarily during the late luteal phase, it is common for it to continue through menstruation as the uterine lining is shed. Summarizing the Cycle I've drawn an essentially idealized menstrual cycle in the graphic below, just showing the relative changes that occur in estrogen (black line), progesterone (gray line), testosterone (bottom black line) and body temperature (at the very top). I've also shown when menstruation, ovulation and PMS generally occur and how the different phases are named and divided. As I will be focusing primarily on the effects of estrogen and progesterone in this book, I have deliberately left out two fairly important hormones which are Follicle Stimulating Hormone (FSH) and Leutinizing Hormone (LH). FSH is primarily involved with the development of the follicle itself while LH controls estrogen production, ovulation, implantation of the egg and development of the corpus luteum. I mention them here as the disruption of LH release patterns can occur under a variety of stressful conditions and this is what fundamentally leads to the menstrual cycle becoming disrupted (discussed in Chapter 12). Interested readers can find more complete graphics online. In any case, you can see how the dynamics of the cycle change which I will summarize. Menstruation occurs from roughly day 1 to 5 before estrogen starts to climb slowly during the follicular phase of the cycle, surging in the final few days before dropping rapidly when ovulation occurs. Into the luteal phase, both estrogen and progesterone show a slow increase during the luteal phase hitting a peak at the middle of the cycle before gradually decreasing over the second half. If occurs at all, PMS or PMDD will occur in the last 4-7 days (roughly) before menstruation occurs and the cycle starts over. 10 As I discussed in the previous chapter, the above dynamics should help to illustrate just how much more complex women are compared to men. Women have two primary hormones that are increasing, decreasing and criss-crossing and, as you'll see, interacting in complex and fascinating way. In contrast, a man's hormonal profile would be a more or less straight line for testosterone across every day of the month with not much else changing significantly. Those gender differences are present even before considering the hormonal modifiers that I will discuss in the next chapter. Individual Variability in the Menstrual Cycle While the above graphic and description represents a sort of idealized menstrual cycle, there are enormous variations that can occur both between women or in the same woman from month to month (when women first start menstruating their cycles may be extremely variable although this decreases with age). Technically a normal menstrual cycle can last anywhere from 24-32 days with 28 simply being an average or assumed length and any two women might easily have different cycle lengths. While some women are extremely consistent in their cycle lengths, others show more variability where the length of the cycle varies from month to month. A woman might have a 30 day cycle one month and a 23 day cycle the next or what have you. While not a variation per se, a large number of factors including diet, exercise and stress can change or disrupt the cycle. One of these is a shortening of the luteal phase where the time from ovulation to menstruation is less than normal. Here cycle length may actually remain the same which means that while the luteal phase is shortened, the follicular phase is lengthened. Other variations may be present as well After ovulation, there can be variations in how much of a temperature increase is seen with women experiencing relatively smaller or larger increases. There is also significant variation in how much or how little metabolic rate increases. Even with an apparently normal cycle length, there are times when an egg is not released (termed an anovulatory cycle). This is common when women first start menstruating but can also occur due to diet, exercise and stress. While PMS lasts 5-7 days on average there is enormous variability in this with many patterns having been observed. Some women show shorter or longer phases of PMS and some women may experience PMS symptoms at other times of the luteal phase. So she might have a day or two of symptoms early in the luteal phase that then end along with several more days of PMS in the late luteal phase. As I've mentioned, there are also huge differences in how much of an effect PMS has on any given woman. This can be true in terms of the number of symptoms present, their severity and many other factors. While an average menstruation may last 3-5 days,this too can vary with women having shorter or longer durations of bleeding. The level of bleeding can vary a well. Some may only spot lightly (and it's not uncommon for women to spot briefly and then stop before experiencing true menstruation a few days later), others will bleed lightly and others have what is referred to as heavy flow (I'll mention this again when I talk about iron requirements in women). In extreme cases, women will expel what they usually call "chunky bits", clots of blood and tissue from endometrial buildup that is being expelled. I won't look into the details of these variations but they most likely relate to the relative levels of estrogen, progesterone (or other hormones) that are present along with how any given woman's body responds to them. Women's health care providers often refer to women as being relatively estrogen or progesterone dominant, meaning that they have higher than expected levels of a given hormone during a given phase of the cycle, and this can impact on every aspect of a woman's physiology including her menstrual cycle. There is also assuredly a genetic component. The existence of this degree of variation is why I said at the outset that there is really no truly normal menstrual cycle. Certainly there is no consistent one. Yes, the general pattern is the same between women but the specifics of hormone levels, durations of the phases, bleeding, presence of absence of PMS/PMDD all vary enormously. As stated, two women can show completely different responses and any individual woman may vary from month to month. I bring this up as I can't cover every possible eventuality and will be working, in a practical sense, from the idealized 28-day menstrual cycle throughout this book. Any given reader of this book will have their own pattern (or patterns) and, within the context of this book (and perhaps moreso Volume 2), women or their coaches will have to be their own best scientist. Regular tracking of factors such as energy, hunger, mood, etc. will help illustrate a given woman's individual patterns and used to adapt how diet or training is applied or utilized. But with those general patterns and changed sketched out, let me now look at how a woman's reproductive hormones impact on her physiology since that will provide a picture of how her physiology is changing across the cycle. To do so, I need to provide a bit of background information. 11 Hormones A hormone is any compound in the body that exerts a biological effect somewhere else in the body (it's technically a little more complex than this but this is good enough). That effect occurs when a hormone binds to its receptor and causes something to happen. The standard analogy here is that the hormone is a key and the receptor is a lock and only the right key can open the right lock. For the most part, every hormone has a specific receptor that it can bind to. Estrogen binds to the estrogen receptor, progesterone binds to the progesterone receptor, etc. This isn't universal as I'll discuss below and sometimes a given hormone can kind of bind to a receptor. As well, there can be different receptor subtypes, estrogen has an alpha and beta subtype for example, found in different parts of the body. This simply allows a single hormone to have different effects in the body depending on which receptor subtype is present. Estrogen might have one impact on fat cells and a different effect on muscle or breast tissue. So a hormone (key) binds to its receptor (lock) and this causes something to happen and some signal to be sent in the body. How much of an actual signal is sent by a given hormone is related to three primary factors. The first is the level of the hormone itself. Simplistically, the more of a hormone present, the more of an effect that is seen. The second is something referred to as affinity, that is how well or poorly a given hormone binds to the receptor. A hormone with high receptor affinity binds very tightly, generally sending more of a signal, while one with low receptor affinity binds poorly and sends less of a signal. The third has to do with the sensitivity of the receptor to that hormone. When a receptor is very sensitive, that means that any given level of a hormone will send a larger signal. When a receptor is insensitive or is said to be resistant, either a given level of hormone will send less of a signal or more will be required to send the same signal. Readers maybe familiar with the concept of insulin resistance (or insensitivity) which occurs when the insulin receptor no longer responds well to the hormone insulin. This means that insulin can't send its signal as well as it otherwise would which causes a number of effects in the body. One is that hormone levels may increase in an attempt to overcome the resistance. In the opposite situation, someone with good insulin sensitivity doesn't need much insulin to send a strong signal. Sometimes a given hormone can sort of bind to another hormone's receptor, called cross reactivity. When this occurs, the cross reacting hormone prevents the hormone that would normally bind from sending its signal (the lock is literally blocked). Usually this means that less of a signal is sent than otherwise would be. As one example, cortisol (a stress hormone) can partially bind to what is called the mineralocorticoid receptor (MR), which normally binds a hormone that causes water to be retained in the body. While cortisol sends less of a signal at the receptor, when enough of it is present, it can still cause water retention. Progesterone is notable in this regard as it can bind to four different receptors in the body. Finally, a hormone can also act as an antagonist at a receptor meaning that it binds and directly prevents the normal signal from being sent. So a hormone acting as an androgen receptor antagonist will prevent the androgens from sending their normal signal. Additionally, since most tissues in the body have receptors for more than one hormone, with those hormones often having different or even opposite effects, there can be complex interactions. In some cases, the effects of the hormones compliment one another, in others one hormone can effectively block the effects of another. As I'll discuss below, progesterone not only has effectively opposite effects of estrogen but blocks the effects estrogen would normally cause. And this is all mostly relevant as variations in all of the above, hormone levels, hormone ratios, receptor affinity, sensitivity, etc. are probably what are contributing primarily to some of the variability seen in women. The system can get even more complicated but the above should give readers at least a general picture. The main take away message is that specific hormones have specific effects and they do so through their specific receptor. But that there are endless ways that the system is modified, some of which are critical understanding the changes in a woman's physiology during her cycle. It's probably fair to say that there are differences in the levels (or effects) of many, if not, most hormones in a woman's body compared to a man's but I don't intend to detail all of them here. While I will look at other hormones at the end of the chapter I will focus on the reproductive hormones, testosterone, estrogen and progesterone in terms of their effects on fat gain or fat loss. Androgens/Testosterone While estrogen and progesterone are the primary women's reproductive hormones, I want to start by looking at the androgens/testosterone. The term androgen is a general one, referring to any hormone with masculinizing or androgenic effect, and includes a variety of hormones. Testosterone is probably the most well known and is produced primarily in a woman's ovaries. However other androgens such as androstenedione, DHEA, DHEA-sulfate (DHEA-S) which are produced in the adrenal gland, are extremely 12 important to women as they represent a large portion of a woman's overall androgen output. After menopause, especially, when the ovaries are no longer production testosterone, the adrenal androgens become that much more important. DHEA is especially critical in terms of a woman's exercise performance and I will discuss it more in Volume 2. That said, in this book I will primarily focus on testosterone as it tends to generally represent the effects of this type of hormone. Testosterone is a steroid hormone which simply means that it is produced from cholesterol. Estrogen, cortisol and progesterone are steroid hormones as well. While often thought of as a "male" hormone, women do produce relatively small amounts of testosterone (in the same way men produce small amounts of estrogen and progesterone). On average, women have testosterone levels about 1/15th (ranging from 1/10th to 1/30th) the levels of testosterone as men. An average testosterone level for men might be 545 ng/dL (this varies from 300-900 ng/dL) while women might have levels of 25-35 ng/dL. As mentioned, women release testosterone and other androgens from their ovaries and adrenal cortex but androgens can also be produced via chemical reaction in other parts of the body. Testosterone can also be converted to dihydrotestosterone (DHT, a hormone involved in hair loss) and estrogen (fat cells convert testosterone to estrogen, via an enzyme called aromatase, in both women and men). As I mentioned above, the receptor for testosterone, DHT and other androgens is called the androgen receptor or AR. Testosterone has a number of effects in the body which are typically subdivided into anabolic (tissue building) and androgenic (masculinizing). Anabolic effects include increasing protein synthesis and muscle mass. Testosterone also increases blood cell number and women generally have a lower hematocrit (the ratio of red blood cells to plasma) than men due to their reduced levels of testosterone. Androgenic effects can be thought of as other secondary male sexual characteristics such as increased body hair, a deepened voice, oily skin, acne, hair loss and others (the increase in testosterone at puberty is what causes these changes in boys). As mentioned, elevated testosterone also contributes to hair loss due to it's conversion to DHT in the scalp. Testosterone has a pronounced effect on tissues such as skeletal muscle and bone where it increases the size and strength of both. For the most part, the effects of testosterone in women are similar to that of men but there are exceptions. For example, in women elevated levels of testosterone impairs insulin sensitivity and causes insulin resistance while, in (hypogonadal) men, it generally improves it. Testosterone also directly impacts on both body weight and body fat levels but in a gender specific way. Elevated testosterone in women, for example, increases bodyweight and body fat (and specifically fat around the midsection) while those same elevations in testosterone (especially if low levels are being corrected) tend to reduce both in men. Overall, women's lower levels of testosterone decrease both the anabolic and androgenic effects. Women have less muscle mass and bone density, carry more total body fat with a different distribution and, unless testosterone levels are elevated, don't show the typical male secondary sexual characteristics. At the same time, even small increases in testosterone have more profound effects in women due to their higher sensitivity to it. Women with slightly or significantly elevated levels of testosterone may have increased body hair, oily skin, acne or experience hair loss. They store larger amounts of fat around the midsection and often show increased levels of muscle mass or improved athletic performance. This occurs in women who use anabolic steroids (synthetic derivatives of testosterone) and I will describe two biological causes of elevated testosterone levels in women in the next chapter. There are endless other effects of testosterone in both the female and male body but to detail them all would be impossible. They can involve behavior, with at least some linkage between testosterone and overall aggressive behavior, psychology and others and the differences between women and men are at least partially due to the differences in hormone levels (along with the potential for genetic and biological programming from prenatal hormone exposure as I discussed in Chapter 1). Testosterone levels do impact health on a number of ways and are a large part of why men are at a greater risk for certain diseases such as cardiovascular disease than women. This is primarily related to body fat patterning with a woman's lower body fat pattern being more protective than a man's more central body fat pattern (2). For women, it's actually a little bit more complicated as her relatively lower risk is due as much to having lower testosterone levels (which reduces risk) as having elevated estrogen levels (which are protective). Women who have elevated levels of testosterone and who have a more male-like body fat pattern are at an increased risk due to the elevated testosterone levels. At menopause, when women's estrogen and progesterone levels drop, there is also an increase in cardiovascular disease risk (and changes to a more male fat patterning). It is the balance of and combination of effects of the two hormones that are at work here and, overall, women who have a relatively higher levels of estrogen and testosterone are relatively healthier than the reverse (3). 13 As a piece of trivia, women live, on average, about 7 years more than men and at least part of this is due to differences in testosterone levels with men's higher testosterone levels being responsible for their earlier deaths. Studies of modern day eunuchs, men who have had their testicles removed and who produce no testosterone live 14-19 years longer than the average male (4). At the same time, men with only lower testosterone (rather than producing none) are at a higher health risk and lower testosterone levels in men tend to cause the same problems as elevated levels in women (5). But ultimately most of the effects of testosterone in women are fairly small, due to the lower levels that are typically present. As well, outside of the small burst right around ovulation, testosterone levels stay relatively stable across the menstrual cycle meaning that it's effects will be relatively stable. As the primary reproductive hormones, estrogen and progesterone play by far the greater role. Estrogen and Progesterone: Introduction The two primary female sex hormones are estrogen and progesterone and you've seen above how they change across the menstrual cycle. In the same way that women have roughly 1/15th the testosterone levels of men, men have about 1/10th (or so) the levels of estrogen and progesterone (as men's physiology is not the topic of this book I will not discuss it further). I mentioned in Chapter 1 that determining the effects of estrogen and progesterone on a woman's physiology was very difficult for researchers for quite some time. Was it the increase in one hormone, the decrease in another, the combination, the ratio? After decades of research, the effects of each hormone is fairly well established. The follicular phase is actually fairly simple in that progesterone is very low and has very little effect overall with estrogen being the primary determinant of what is occurring physiologically. The surge of estrogen in the late follicular phase has a number of effects but the overall picture there is fairly simple. Things become more difficult in the luteal phase where estrogen first drops (this drop causing one set of effects) before both estrogen and progesterone increase and then fall again, causing different effects still. While I will discuss each hormone individually below, the simplest way of looking at this issue is that estrogen and progesterone have effectively opposite effects on a woman's physiology. Importantly, when progesterone is high during the luteal phase, its effects dominate as it blocks/opposes estrogen's effects. Even here there is a further complication as estrogen sensitizes the progesterone receptor so that progesterone will have a larger impact during the luteal phase. In that sense, at least some of progesterone's overall effects are can be indirectly attributed to estrogen. Regardless, once I've looked at the effects of both estrogen and progesterone and consider the interactions (along with the spike of estrogen before ovulation), the overall structure of the menstrual cycle should make some logical sense in how it is trying to prepare a woman's body for the potentiality and eventuality of pregnancy. Estrogen There are actually three primary estrogens which are estrone (E1), estradiol (17 beta-estradiol or E2) and estriol (E3) which have slightly different effects. Each tends to predominate at different times in a woman's life with estrone most relevant during pregnancy and estriol most relevant after menopause. Since I am discussing the normal menstrual cycle here, I will exclusively focus on estradiol or simply use the term estrogen generally. Estrogen is produced primarily by a woman's ovaries although it can be produced elsewhere, generally by the conversion of other hormones such as testosterone via aromatase. As I mentioned above, estrogen has its own specific receptor and there are two subtypes called estrogen receptor alpha and estrogen receptor beta. These are found in varying levels in different tissues in the body which not only explains how estrogen can have differential effects in different places but also why certain drugs that target specific receptors can be used to treat such diseases as breast cancer. In this case, it is estrogen receptor alpha that is primarily at play and drugs that specifically block that receptor allow estrogen to work in other tissues that express estrogen receptor beta while still treating the disease itself. In the same way that testosterone is responsible for the development of male secondary sexual characteristics, estrogen has a primary effect on the development of female secondary sex characteristics. Estrogen is critically involved in the deposition of breast fat and contributes both to women's increwased overall body fat levels and her lower body fat patterning. In men estrogen can have the same effect, for example and some males develop gynecomastia, the development breast tissue, under some conditions such as puberty or testosterone abuse. High levels of estrogen can also cause water retention. Estrogen causes the growth plates of bones to close and this is part of why women are typically shorter than men; at puberty their bones fuse and stop lengthening. Critically, estrogen is a major player in increasing bone density although it is not the only factor here. Estrogen also plays a role in cognition and 14 mental function and there are endless other effects that I won't detail here (6). Since is is the primary topic of this book, I will focus primarily on the impact of estrogen on processes related to fat loss and fat gain with a brief mention of some of its effects on exercise, training and muscle growth (discussed in detail in Volume 2). Recall again that estrogen dominates during the follicular phase (first half of the cycle), starting at a low level and gradually increasing to a surge prior to ovulation. It then drops before rising gradually during the luteal phase, reaching a level about half of that of the peak during the follicular phase. Since estrogen has typically been blamed for a woman's issues with body fat, let me start with its effects in this regard. It turns out that estrogen has both positive and negative impact on fat metabolism, fat cells and fat loss although, in the aggregate most of its effects are positive (this may come as a surprise to many readers). I should mention that one very confusing issue regarding the role of estrogen and body fat in women is that it has different effects in different parts of the body. This is a large contributor to the typical fat patterns in women. I'll talk about some of the implications of this later in the book. Discussed more below, estrogen can impact negatively on thyroid levels which can have an indirect effect on body fat by lowering metabolic rate; estrogen also has a number of other potential negative effects on body fat. I haven't talked about how fat stores or mobilizes fat yet but estrogen does increase the levels of a specific receptor in fat cells (the alpha-2 receptor) that inhibits the release of fat from fat cells by decreasing the fat mobilizing effect of hormones released during exercise (7). These receptors are found to a greater degree in women's lower body and this is one way that estrogen may at least indirectly impact on body fat levels in the lower body. In contrast, estrogen does not impact on the levels of the alpha-2 receptor in the upper body and increases the fat cell's sensitivity to fat mobilizing hormones (8). Estrogen thickens the connective tissue in the skin and fat matrix in the lower body and this is the primary cause of cellulite (9). What is happening is that excess body fat pushes through the connective tissue and you can think of it as a holiday ham pushing through the mesh it comes wrapped in. Cellulite is not a different type of fat and it doesn't respond to nearly any of the supposed treatments for it short of some invasive almost surgical treatments (fat loss generally improves its appearance). The presence or absence of cellulite seems to be partially genetic (perhaps due to elevated estrogen levels Beyond that, most of estrogen's other effects are relatively positive in terms of body weight and body fat levels. First, there is an enzyme in fat cells called lipoprotein lipase (LPL) which breaks fatty acids off of what are called chylomicrons (produced after fat is eaten) for storage. Unless levels are very low, estrogen inhibit the activity of LPL in lower body fat cells inhibiting fat storage in that area (9a). Estrogen also inhibits the storage of visceral fat which is at least part of why it is protective against heart disease. While LPL was long-considered the singularly important enzyme for fat storage, there is a second, far more important enzyme responsible for fat storage in fat cells called acylation stimulating protein (ASP) that plays a far larger role. While progesterone (discussed next) affects ASP, estrogen does not. Estrogen also increases LPL activity in muscle cells which causes fat to be stored there as Intramuscular Triglyceride (IMTG). These provide a quick source of energy during certain types of exercise and women's higher levels of estrogen cause them to store more IMTG than men (10). Estrogen also increases the level of fatburning enzymes in skeletal muscle along with activating a compound (called AMPk) which enhances the use of fat for fuel. Although many of estrogen's effects clearly limit fat gain in women, there is no doubt that many aspects of its metabolism clearly function to increase lower body fat. This fat exists primarily to provide energy during pregnancy and breastfeeding and given the large calorie requirements of both, it makes logical evolutionary sense to store calories there (11). Demonstrating this is an oddity of female metabolism where, during pregnancy, normal fat storage and mobilization patterns effectively reverse. The normally easy to store and difficult to mobilize hip and thigh fat becomes the easiest to mobilize as it is being used to provide energy for pregnancy and breastfeeding. Despite it's potentially negative effect on body fat, and especially lower body fat, estrogen also has a number of significant positive effects helping to regulate appetite, body weight and body fat levels (12). Demonstrating this is the fact that postmenopausal women who do not go on Hormone Replacement Therapy (HRT) gain significant amounts of weight and body fat (with a shift in distribution) and this is prevented if estrogen levels are maintained with HRT. And at least some of this is mediated by the interaction of estrogen with the Hormone leptin (discussed briefly below and in much greater detail later in the book) which acts to regulate appetite, body weight and body fat. Among its many other effects, leptin acts to improve the brain's response to other hormones that help to regulate appetite and body weight. Simply, when leptin is low (as occurs during dieting), these signals don't work as well and this is part of the overall adaptation to dieting that I will discuss in detail in Chapter 9. 15 Not only does estrogen increase leptin production from fat cells, it also increases leptin sensitivity in the brain causing leptin to send an even stronger signal. Estrogen also sends it's own leptin-like signal in the brain further increasing the effect (13). The end result of this is that estrogen helps to control hunger, with the largest effect occurring during the follicular phase and hunger being lowest in the 4 days or so before ovulation when estrogen surges. As well, falling levels of estrogen after ovulation contribute to the increase in hunger and cravings that are seen during the luteal phase (14). At least one way that estrogen does this is through it's effects on the brain chemicals (effectively hormones in the brain) serotonin and dopamine. Serotonin is involved in overall mood and appetite (especially carbohydrate cravings) and low levels can contribute to depression. Dopamine is part of the reward system and low levels can drive cravings for high-calorie/high-sugar foods. When estrogen is high during the follicular phase/before ovulation, serotonin and dopamine levels will be higher; when estrogen levels decrease, the levels of both serotonin and dopamine will as well, increasing hunger and cravings. Estrogen has many other positive effects on metabolism. One is that it increases insulin sensitivity. This means that, when estrogen is the dominant hormone, the body will burn more carbohydrates for fuel and this has implications for both diet and exercise. Insulin also inhibits fat release from fat cells and this change is yet another way that estrogen can impact on body fat levels. Finally, the surge in estrogen during the second half of the follicular phase causes a woman's body to retain more sodium; on a high-salt diet this will cause her body to retain water. While training will be discussed in detail in Volume 2, let me briegly address estrogen's overall positive effects here. Estrogen prevents inflammation, limits free radical damage, may limit muscle damage itself (reducing soreness) and acts to help remodel and rebuild skeletal muscle (15). At the same time, estrogen may negatively impact on tendon strength and this has enormous implications for the risk of knee injury in women that I will discuss in Volume 2 (16). While estrogen is most commonly blamed for all aspects of woman's body fat (and especially lower body fat problems), it's clear that the picture is more complicated than that. Certainly estrogen has some effects in fat cells, especially in the lower body. that can be considered negative. At the same time, estrogen also has a number of positive effects on fat metabolism, body weight regulation and appetite control. In the aggregate, estrogen probably has a more overall positive than negative effects in this regard. I'd add that the impact of estrogen on any aspect of fat gain or fat loss interacts with the diet. Many of estrogen's negative effects are primarily seen when combined with a high dietary fat intake. Regardless, while estrogen receives much of the blame, the fact is that progesterone causes far more problems. Progesterone Progesterone is the second primary reproductive hormone in women and is released from the corpus luteum that develops after release of the egg at ovulation. While it has a tremendous number of roles in the body most of them aren't that relevant to this book and I will once again focus on fat loss, fat gain while briefly addressing training. As a steroid hormone, progesterone has a structure similar to many other hormones in the body and can actually be converted to aldosterone (involved in water balance), cortisol (a stress hormone), and the androgens. Due to that structural similarity, progesterone can bind to four different receptors. The first is the progesterone receptor itself where a normal signal will be sent. At the cortisol receptor, progesterone only sends a weak signal, weaker than cortisol itself. Progesterone is also an antagonist at the androgen and aldosterone receptor, not only blocking the effects of the hormones that would normally bind there but sending an actual negative signal. This cross reactivity not only explains many of progesterone's effects but is critical for the discussion of birth control in Chapter 3. As I described above, progesterone remains low during the follicular phase of the menstrual cycle and has little to no effects at that time. During the luteal phase, following ovulation, progesterone starts to increase gradually, reaching a peak halfway through the cycle before decreasing again prior to menstruation. And as I'll describe, as much as estrogen tends to get the blame for so many aspects of women's fat loss issue, progesterone is of far more importance. Not only does it have its own profoundly negative direct effects, it also acts to cancel out estrogen's many positive effects, essentially doubling its negative effects in this regard. Before discussing progesterone's negative effects, let me examine its one potential benefit in terms of fat loss. I mentioned above that the increase in progesterone is the cause of an increase in women's body temperature after ovulation and that Basal Body Temperature (BBT) has long been used to indicate when ovulation has occurred. Along with changes in vaginal mucus (due to estrogen), this can be used to determine a woman's peak fertility. Along with this increase in body temperature comes an increase in energy expenditure and resting metabolic rate. This is another effect that is variable between women with 16 increases ranging from 2.5-10% over normal which might amount to roughly 100-300 extra calories per day burned. In premise this should benefit weight and fat loss. This is counteracted by the fact that, during this time, both hunger and cravings are increased, adding to the fact that women are more prone to food cravings already (16a). Women also show an increased attention/notice of tasty, high-calorie foods (16b). This can make controlling food intake more difficult and, on average, women's calorie intake increases more than their metabolic rate. The increase in hunger during the luteal phase occurs for several reasons. As I mentioned above, a primary one is the drop in estrogen from before ovulation which causes both serotonin and dopamine levels to go down. While progesterone by itself does not appear to increase hunger, it does so in the presence of estrogen, which describes is the hormonal profile at this time (17). Progesterone causes some degree of insulin resistance (described below) and one consequence of this is that blood sugar may become unstable. This can cause blood sugar levels to fall, also stimulating hunger. Finally, although leptin levels go up during the luteal phase (which should help to control hunger), leptin resistance also develops so that this effect is blunted. Overall, the above effects result in increased hunger and cravings, especially for high-sugar/high-fat foods (chocolate is the most commonly reported craving although other foods are often craved). When diet is uncontrolled, an increase in food intake of 90-500 calories has been observed during the luteal phase and this can readily exceed any small increase in metabolic rate that occurs. There are supplements that can potentially help to avoid this that I will discuss in Chapter 23. In premise, so long as food intake can be controlled during this time, the increase in metabolic rate during the luteal phase can be used to enhance fat loss. If a woman is able to avoid an increase in calorie intake, the 100-300 calorie/day increase in metabolic rate with an average duration of 10 of the 14 days of the luteal phase would burn ~1000-3000 extra calories. This amounts to somewhere between 1/3rd to just under one pound of extra fat lost. Alternately, food intake could be increased slightly during this phase which could increase diet adherence for those women seeking fat loss (I will discuss this again in Chapter 19). I mentioned above that progesterone can bind to the aldosterone receptor, which is involved in water retention in the body. Since it blocks aldosterone from binding and causing water retention, progesterone causes women to be less likely to retain water during the first half of the luteal phase. As progesterone drops during the late luteal phase/PMS week, there is a rebound effect which can cause water retention. As with the surge in estrogen before ovulation, this drop in progesterone changes how the body handles sodium and women on a high-sodium diet during this time may show extreme amounts of water retention. Dietary strategies to limit this will be discussed later in the book. The above are the potentially good effects of progesterone on a woman's body weight and body fat levels. Now let me look at the large number of bad effects. Like estrogen, progesterone increases LPL activity in women's lower body fat, breaking fatty acids off of chylomicrons for storage (17a). This is compounded by the fact that progesterone also activates ASP (mentioned above) which is not only one of the key enzymes in storing body fat but has been described as the most potent enzyme for the fat storage process. ASP is found preferentially in subcutaneous fat (which women have more of to begin with) and, for all these reasons, one researcher has called ASP "A female fat storing factor" (18). All of which is important as, unlike estrogen, progesterone potently activates ASP (ensuring storage of the fatty acids made available by its effects on LPL). Within the context of the menstrual cycle this sets up a sequence of events where the surge in estrogen before ovulation not only sensitizes the progesterone receptor but also increases the number of the fat loss inhibiting alpha-2 receptors. Increasing progesterone levels then activates LPL and ASP ensuring that excess calorie intake (driven by increased hunger) is stored as body fat. As well, progesterone opposes estrogen in that it impairs insulin sensitivity meaning that the body doesn't utilize carbohydrates as well. This can be good or bad depending on the situation. In the context of a high-carbohydrate, low-fat diet, insulin resistance is not a good thing as there will be an overproduction of insulin. In contrast, insulin resistance can be beneficial on a lowered or low-carbohydrate diet (19). The practical implications of this, discussed in Chapter 19, is that a higher carbohydrate/lower fat diet will be superior in the follicular phase while a lowered carbohydrate/higher fat diet will be superior in the luteal phase. As mentioned above, this insulin resistance also causes blood glucose levels to become more unstable, affecting energy levels, mood and potentially hunger. Perhaps confusingly, the insulin resistance that develops during the luteal phase causes a decrease in carbohydrate use and an increase in fat utilization both at rest and during aerobic exercise. While this sounds beneficial, most of the additional fat being burned is from the IMTG stored within the muscle so this does not impact the visible subcutaneous fat stores directly (I will discuss this in detail in Chapter 10). The combined effect is that progesterone increases the storage of fat in a woman's fat cells while increasing the use of fat stored in her muscles. 17 Taken as a whole, the effects of progesterone are to ensure and enhance not only general fat storage but fat storage specifically in a woman's lower body. Following ovulation, in preparation for pregnancy, the increase in progesterone will not only block estrogen's beneficial effects but will cause her to store more fat in her fat cells while burning more in her muscles. Although metabolic rate may be up slightly, hunger and cravings will increase and this tends to increase calorie intake far above the small increase in metabolic rate. If calorie and fat intake is too high during this phase, there will be increased fat storage. In contrast, if calories can be controlled, the changes in metabolic rate can be harnessed to potentially increase fat loss. Looking briefly at training, progesterone's overall effects are quite negative. First and foremost, progesterone binds to the androgen receptor which would normally bind testosterone. This not only prevents testosterone from binding and having a positive effect on muscle but progesterone acts as a receptor antagonist, inhibiting any effect that might be seen. Due to this, progesterone tends to decrease tendon strength and decrease the ability to build muscle (recall that estrogen directly improves a woman's muscular remodeling and growth). For endurance athletes primarily, the increase in body temperature during the luteal phase can cause problems with thermoregulation during exercise in hot or humid conditions. High-intensity endurance performance may also be impaired as the use of carbs for fuel is lowered because of the insulin resistance that develops. An Overview of Menstrual Cycle Changes With the above discussed, let me summarize the changes that are occurring. Early and Late Follicular Phase After menstruation, during the early follicular phase, estrogen and progesterone are both fairly low although estrogen will start to increase and have the dominant effect overall. Insulin sensitivity will be high with a woman's body using more carbohydrate for fuel at rest. Appetite and hunger will be stable and controlled, especially in comparison to the previous luteal phase. Blood glucose levels will remain stable as well. Her metabolic rate will be normal and fat storage will be normal and/or lowered at least relative to the luteal phase. Estrogen will be exerting anti-inflammatory effects and have a positive effect on muscular remodeling from training. Early in the follicular phase, water retention will be low after any retention from the previous late-luteal phase has disappeared and this is when women will show their lowest bodyweight. For the most part all of the above will hold in both the early and late follicular phase with one or two exceptions. Due to the surge in estrogen, appetite will be reduced significantly in the 3-4 days prior to ovulation. This same surge can cause water retention, especially if a woman is on a high sodium diet. Early Luteal Phase During the early luteal phase, most of the above reverses or at least starts to reverse. Body temperature increases slightly and with this will come a small increase in metabolic rate. Hunger and cravings will tend to go up both due to the fall in estrogen after ovulation along with increasing progesterone levels. This can cause an increase in food intake that can easily overwhelm the small increase in metabolic rate. Due to the impact of progesterone on both LPL and ASP, fat storage will be higher than in the follicular phase and this is compounded by estrogen's effects both on anti-fat mobilizing receptors and it's sensitizing of the progesterone receptor. Since progesterone causes insulin resistance, a woman's body will use more fat for fuel (and less carbohydrates) both at rest and during exercise but the fat comes from within the muscle. Blood sugar becomes unstable and this can cause energy and mood swings along with hunger. Binding of progesterone to the aldosterone receptor will cause a loss of body water and there tends to be little water retention during this phase. Due to the negative impact of progesterone on muscle along with it's blocking of the androgen receptor, muscle growth and remodeling are negatively impacted. The increase in body temperature may harm endurance performance especially in the heat. Late Luteal Phase/PMS/PMDD Moving into the late luteal phase, estrogen and progesterone continue to drop and this has a large number of effects on a woman's body. Blood sugar levels often become even more unstable during this time period and this can cause women to experience low blood sugar (hypoglycemia) negatively affecting energy, mood and hunger. Just as with earlier in the luteal phase, cravings for high-fat and high-sugar foods are usually high here, related both to the drop in serotonin and dopamine levels. Falling dopamine levels also cause levels of the hormone prolactin to increase, causing breast tenderness. The same basic pattern of fat storage and fuel utilization seen in the early luteal phase will be maintained. As progesterone 18 drops, there is a rebound effect with water retention typically being the worst at this time with the effect being increased for women on a high-sodium diet. While not frequently mentioned, sleep is often interrupted during the late luteal phase as well. Melatonin, a hormone predominantly involved in sleep, is more inhibited by the impact of even small amounts of light, and this can prevent women from sleeping well (20). There are a number of strategies including avoiding light late at night, sleeping in a dark and cool room that can help with this. A sleep mask may also be useful and I will discuss specific sleep supplements in Chapter 23 that can be used. Finally, in that proportion of women that experience it at all, the late luteal phase is also when Premenstrual syndrome (PMS) or Pre-Menstrual Dysphoric Disorder (PMDD) will typically occur (21). Again, there are different patterns here with some women experiencing symptoms in the early luteal phase or having those same symptoms continue through the first days of menstruation. Women may suffer from headaches, joint or muscle ache, digestive problems, issues with coordination and many others. Exhaustion, irritability, anger, problems with concentration and mood swings are common as well (as I mentioned, in PMDD, this may reach the extreme of depression, anxiety or suicidal thoughts). Antidepressants have shown a benefit for PMS and PMDD which further suggests that serotonin levels are involved with some of the typical PMS symptoms As well, due to low estrogen levels, a large percentage of women suffer from hot flashes, identical to what is seen after menopause (21a). As mentioned, some women experience none of the traditional effects whatsoever. Treating the symptoms of PMS have always been of great interest (for what should be obvious reasons) and many approaches have been tried or shown to be beneficial with an equal number of often claimed remedies having zero effect. Regular exercise appears to reduce the symptoms of PMS but this may be problematic if the presence of PMS or PMDD makes it difficult for women to maintain an exercise program (21b). A number of dietary supplements, including specific vitamins and minerals along with others may help to alleviate many of the typical PMS symptoms as well and I will provide recommendations in Chapter 23. Finally, in preparation for shedding the uterine lining and the start of menstruation, cramps are common here and these may continue into the early stages of menstruation as bleeding starts. Related to falling levels of progesterone, the cause of the cramping are prostaglandins, short lived chemical messengers, that cause the uterus to contract. This can generally be treated with Non-Steroidal AntiInflammatories such as aspirin, Naproxen Sodium, Ibuprofen and others. Although the reason is unclear, approximately 18% of women are resistant to their effects and may require medical treatment (22). As the late luteal phase ends, menstruation and the next cycle begins. Summary of the Menstrual cycle I've summarized the primary general changes that occur in a woman's physiology that occur across the menstrual cycle and this chart, or a variation on it, will appear multiple times throughout this book. As you can see clearly, there are changed in fuel utilization, fat storage, hunger, appetite, water retention and others that occur due to the impact of either estrogen, progesterone with estrogen and progesterone typically having roughly opposite effects in the body. From a fat loss perspective, both estrogen and progesterone have positive and negative effects although it's arguable that the late luteal phase when progesterone is dominant is truly the problem time. Day 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 Phase Early Follicular Note Menses (3-5 days) Dominant Hormone Estrogen Estrogen Progesterone Progesterone Insulin Sensitivity High High Lowered Lowered Fuel at Rest Carbs Carbs Fat Fat Fuel During Exercise Fat Fat Fat (increased) Fat (increased) Fat Storage Lowered Lowered Increased Increased Metabolic Rate Normal Normal Increased Increased Hunger Lowered Lowered Increased Increased Blood Glucose Stable Stable Unstable Unstable Water retention Lowered Increased Lowered Increased (highest) Muscle Growth Increased Increased Decreased Decreased Early Follicular Early Luteal Late Luteal Ovulation 19 PMS (4-7 days) Hopefully the above chart makes it clear just how much more complicated a woman's physiology is compared to a man's. There are major changes occurring at least every 2 weeks and in some cases even more frequently than that. These changes interact, overlap and often reverse completely (in contrast, men are basically the same daily). Addressing women's needs for diet or fat loss means taking those changes into account, taking advantage of the positives while minimizing the negatives. Other Hormones While I have focused on the differences in the reproductive hormones in this chapter in order to describe the menstrual cycle, there are other differences between women and men's hormones that are important or worth addressing at least briefly and I'll round out the chapter by looking at a few of them. One hormone that will not be discussed here is cortisol as it will be discussed in detail in Chapter 13. Growth Hormone (GH) Growth hormone is involved in many processes in the body but a primary one in adults is the mobilization of fat. Women show higher levels of GH than men along with an larger increase in response to exercise. This is probably part of why women use more fat for fuel under some conditions. Insulin The hormone insulin is one about which there is a great deal of confusion and mistaken information. While all too frequently blamed for being the cause of fat gain it's better to think of insulin as a general storage hormone. It impacts on fat metabolism, stimulating fat storage and inhibiting fat mobilization and burning but it also increases the storage of carbohydrate in skeletal muscle and liver and is involved in skeletal muscle growth. In general, women have lower levels of insulin along with better insulin sensitivity than men. This is more pronounced during the follicular phase when estrogen increases insulin sensitivity and is decreased in the luteal phase when progesterone causes some degree of insulin resistance. Thyroid I would imagine that almost all readers are familiar with thyroid hormone although, like so many things, there is a lot of confusion and misinformation about it. Among it's other effects in the body, thyroid hormones are a primary controller of metabolic rate, interacting with other hormones (such as the catecholamines discussed below). Thyroid also impacts on fat cell metabolism (helping to mobilize fat out of the cells) and skeletal muscle. There are two primary types of thyroid hormone called T4 (thyroxine) and T3 (triiodothyronine) which are released from the thyroid gland in a ratio of about 80% T4 to 20% T3. T4 is primarily a storage hormone which is converted to the more active T3 in other tissues in the body, especially the liver. The conversion of T4 to T3 is important as this process goes down while dieting, leading to lower levels of T3 and a lowered metabolic rate; I'll talk about this more later in the book. Reproductive hormones interact with T3, estrogen decreases levels of active thyroid hormone while progesterone increases them. This may be part of the metabolic rate increase during the luteal phase. T3 can also be converted inside of tissues to T2 which has it's own metabolic effects there (23). Women are significantly more likely to be hypothyroidal (meaning that their thyroid gland releases insufficient levels of thyroid hormones) having more issues when iodine intake is insufficient. They are also three times as likely to suffer from thyroid cancer as men and this is probably related to the direct effect of estrogen on thyroid hormone metabolism (24). Women are also more likely to suffer from depression and, while far from the only cause, low thyroid levels are an often undiagnosed cause. While beyond the scope of this book, Hashimoto's thyroiditis, an autoimmune condition, is far more common in women than men (women are more likely to suffer autoimmune disease in general). In the case of Hashimoto's the immune system attacks the thyroid gland and can cause swings from low to high thyroid. All medical cases of hypothyroid are treated with thyroid hormone to replace the hormone that is not being produced normally. Either T4 or a combination of T4 and T3 are typically given and some women report better results from a type of thyroid called Armour (25). Determination of hypothyroid of any sort must be made through blood tests and medication should be used under the care of a health practitioner. Of more relevance to this book, in addition to their already generally lower levels of thyroid hormones, women's levels can be impacted significantly by certain diet and training practices, this effect being both large and rapid. Women's overall dietary choices can also negatively impact on thyroid hormone levels (and by extension their metabolic rate) and I will discuss these later in the book. 20 The Catecholamines The catecholamine hormones are refers to adrenaline and noradrenaline in America and epinephrine and norepinephrine everywhere else in the world Adrenaline (epinephrine), as it's name suggests is released from the adrenal gland into the bloodstream and has effects throughout the body. In contrast, noradrenaline (norepinephrine) is released from nerve terminals and only has effects very locally where it is released. Many readers may have heard these hormones referred to as the fight or flight hormones although, as I'll discuss in Chapter 13, they appear to have slightly different effects in women. Released in response to a variety of stressors, the catecholamines raise heart rate, blood pressure, mobilize fuel for energy and have many other functions. In general women show lower levels of the catecholamines at rest along with a generally lower response to exercise compared to men. While women initially increase levels during exercise to the same degree as men, they rapidly adapt to exercise and no longer match men's levels. As with the differences in GH described above, these differences are probably part of why women's overall nutrient utilization patterns at rest and during exercise are what they are. The catecholamines also interact with thyroid hormone in controlling metabolic rate along with fat mobilization. Thyroid hormones sensitizes the catecholamine receptors (meaning that the hormones send a more potent signal) while the catecholamines stimulate conversion of T4 to T3 in the liver. Like thyroid, levels of the catecholamines drop during dieting and these two factors are a large part of the overall decrease in metabolic rate. Leptin I mentioned leptin briefly above want to examine it equally briefly here (I will discuss its effect in detail in later chapters). Leptin is a hormone released primarily from fat cells and it's discovery in 1994 changed the field of obesity research forever. Not only did it indicate that fat cells were far more than just an inert storage space but would lead to the realization that they produced numerous hormones involved in regulating the body's metabolism (26). Relative to body weight and body fat specifically leptin helped to fill in a number of gaps in previous research in terms of how body fat and body weight were regulated. Early research had suggested that there was some way that the body or brain could "know" how much fat an animal was carrying or how much it weighed so that metabolism and food intake could be adjusted. Although the system is much more complicated, leptin provided a mechanism for how this could occur. Leptin levels turn out to be related to two primary factors. The first is the amount of body fat someone is carrying with higher levels of body fat leading to higher leptin levels. The second is the amount someone is eating over several days with leptin levels rapidly changing in response to alterations in food (especially carbohydrate intake). In the early days of leptin research, it was thought that leptin acted to prevent obesity but this is now known to be false. Rather, leptin acts primarily as an anti-starvation signal with decreasing levels (in response to food restriction or fat loss) signaling the body to lower metabolic rate, increase hunger/appetite along with other effects. (this will be discussed in detail in Chapter 9). Decreasing leptin levels is also involved in menstrual cycle dysfunction, discussed in Chapter 12. Perhaps surprisingly, women turn out to have higher levels of leptin than men. While some of this is simply due to having more total body fat, women's fat cells also produce more leptin than men's due to the effect of estrogen. While estrogen acts to generally sensitize the brain to leptin as I mentioned above, women do tend to show relatively more leptin resistance than men overall, meaning that it cannot send as potent of a signal. As well, although leptin levels increase during the luteal phase (possibly in response to the surge of estrogen right before ovulation), leptin resistance increases further. Finally, in response to dieting and exercise, women's bodies show a different response than men's and it looks like women's brains may respond differently to changing leptin levels than men's. All of this adds to a woman's general physiological tendency to store and hold onto body fat as it means that women's bodies may fight back harder and adapt more quickly to dieting or exercise, slowing fat loss to a greater degree than would be seen in men. I will discuss this in detail in Chapter 9. 21 22 Chapter 3: Hormonal Modifiers Having examined the normal menstrual cycle in detail in the last chapter in terms of the major hormonal changes that occur and how it affects a women's physiology, I want to next look at some commonly experienced situations that will change a woman's physiology from that of the normal menstrual cycle (this will not include explicit disease states which are outside of the scope of this book). I will refer to these as hormonal modifiers throughout this book and each will alter a woman's physiology in a very specific and often dramatic way with implications for her nutritional or fat loss guidelines. While each hormonal modifier is distinct in its own way, there are commonalities and this will allow me to group them somewhat going forwards. Effective Hormonal States In the previous chapter, I discussed the three primary hormones which impact on a woman's overall physiology which are estrogen, progesterone and testosterone. Each has its own distinct effects and, when one or the other is relatively dominant in a woman's body, it generally results in a fairly similar physiology. So regardless of the specific hormonal modifier present, two women with an estrogen-like, progesteronelike or androgen-like physiology will be considered to have a similar physiology in terms of the nutritional and other recommendations I will make later in this book. Each effective hormonal state will be related to the menstrual cycle itself meaning that I will consider the estrogen-like physiology to be the equivalent of the follicular phase physiology described in the last chapter and a progesterone-like physiology to be the equivalent of the luteal phase physiology. The androgen-like physiology is slightly more complicated in that it may generate an estrogen-like or progesterone-like physiology depending on other factors. Amenorrhea/Oligomenorrhea While there is no truly "normal" menstrual cycle, in that the variation between two women (or within the same woman) can be extremely large, it is still possible for the cycle to become extremely disrupted. While there are less severe disruptions I will mainly focus on amenorrhea and oligomenorrhea here. As both will be discussed in some detail in Chapter 12, I will only look at each briefly here. Amenorrhea refers to the absence of a menstrual cycle and is defined clinically as a lack of menstruation for 90 days or more with less than three total cycles in a year (some women will not menstruate for extended lengths of time). Strictly speaking, amenorrhea can occur under many different situations. This can include a woman who has begun to menstruate at all (called primary amenorrhea), pregnancy (where menstruation stops due to not being needed) and birth control (which deliberately shuts off the normal cycle although some bleeding may still occur). There can be numerous medical causes for amenorrhea but none of these represent the type of amenorrhea that I will discus in this book. Rather, I will focus only on Functional Hypothalamic Amenorrhea (FHA). As the name suggests, FHA originates in the hypothalamus (a structure in the brain I will discuss later) which will shut down the menstrual cycle under certain circumstances. Within the context of this book, these tend to be stress related including the stress of dieting, the stress of exercise, mental stress or some combination of the three. Physiologically, in amenorrhea, estrogen drops to about 33% of normal levels and progesterone drops to roughly 10% of normal. The normal cyclical changes are also lost and hormonal levels of both are effectively a flat line. The release of LH and FSH, which I described briefly in the last chapter, also disappears such that the follicle never matures or implants, the corpus luteum doesn't develop and there is no uterine lining to shed (hence the lack of bleeding). When amenorrhea develops, a woman's physiology changes enormously and I will discuss those changes in detail in Chapter 12. Oligomenorrhea refers to an infrequent or delayed menstrual cycle and is defined clinically as a cycle that only occurs every 35-90 days (recall that the normal menstrual cycle occurs within 24-32 days). In contrast to amenorrhea where a woman's primary hormones drop to low levels and show no cyclical changes, in oligomenorrhea those hormones are lowered but are still changing. On some days hormone levels may be identical to the normal menstrual cycle but on others their levels will be random. Like amenorrhea, oligomenorrhea can occur for many reasons. This includes some types of birth control (where light bleeding may occur) and a variety of medical conditions (including PCOS, discussed next). There are two types of oligomenorrhea that I will consider in this book. The first is part of the continuum of adaptations to dieting that can lead to amenorrhea that I will discuss in Chapter 12. While all women were originally thought to have this type of oligomenorrhea although it's now known that there is a subgroup of women who are oligomenorrheic due to elevated androgen/testosterone levels. 23 Hyperandrogenism I want to look next at the situation where testosterone levels in women are elevated which is referred to as hyperandrogenism. Here I am combining two slightly different hormonal situations which are absolute and relative hyperandrogenism. Absolute hyperandrogenism will refer to any situation where a woman's testosterone levels are elevated above normal. Relative hyperandrogenism will refer to a situation where testosterone levels are not elevated above normal but estrogen and progesterone levels have decreased so that androgens are relatively higher. In this section, I only want to look absolute hyperandrogenism, when a woman's testosterone levels are elevated above their normally low level along with the implications that has. Given the effects of testosterone, overall the effect of absolutely hyperandrogenism is some degree of masculinization or virilization of a woman's body with an increased prevalence of male secondary sexual characteristics along with other potentially negative effects. This includes increased body and facial hair, oily skin, acne, and an increased risk of hair loss. Hyperandrogenic women also tend to store more fat in a "male" like body in that they have narrower hips and tend to carry relatively more of their body fat around their midsection. Beyond this, women with elevated testosterone levels often show greater amounts of muscle mass along with potentially improved sports performance and an ability to respond to training. I'll come back to this below and discuss it in detail in Volume 2. Probably the most common cause of elevated testosterone levels in women, and the one I suspect most readers are at least passingly familiar with, is Poly-Cystic Ovary Syndrome or PCOS. PCOS has been estimated to occur in between 6-20% of women and is one of the most common disorders of the endocrine system. PCOS is often associated with either oligomenorrhea, amenorrhea or infertility (due to a lack of an egg being released). In fact, roughly 15-20% of women who are infertile are diagnosed as having PCOS. PCOS is often associated with weight gain and obesity with more fat being carried around the midsection. Here even small amounts of weight loss of 5-10% of current body weight drastically improve health markers and fertility (1). Interestingly, while PCOS women often report having trouble losing weight, research shows no difference in weight loss between PCOS and non-PCOS women, at least within tightly controlled research (1a). In Chapter 23, I'll discuss a number of supplements specifically for PCOS symptoms. PCOS is clinically diagnosed according to the Rotterdam criteria and requires that two of the following three symptoms be present: multiple cysts on the ovaries, clinical or biochemical signs of hyperandrogenism, and either oligomenorrhea or anovulation (an egg is not released). Practically this means that there are four distinct types of PCOS. A woman could have all three symptoms or any combination of two of them (i.e. cysts+hyperandrogenism, cysts+oligomenorrhea/anovulation, or hyperandrogenism+oligomenorrhea/anovulation). When PCOS is present, hyperandrogenism is the most common symptom and this will usually manifest with oily skin, acne, central fat distribution or hair loss), the PCOS women's testosterone levels may be 2.5-3 times a normal woman's levels. While this is still well below even the low normal range in males, women's greater sensitivity to androgens means that this will have a profound effect on her overall physiology. PCOS women also have lower levels of Sex-hormone Binding Globulin (SHBG) which results in more free testosterone (the biologically active type) being available. Perhaps the most commonly seen metabolic dysfunction in PCOS is insulin resistance, an inability of the body to properly respond to insulin. Not only does this have numerous health consequences, it acts to maintain elevated androgen levels as elevated levels of insulin affect adrenal metabolism so that it produces even more androgens. Insulin resistance is extremely prevalent in PCOS and is estimated to occur in 60-80% of women with PCOS. This may increase to 95% if obesity (specifically fat around the midsection, called central obesity) is present. The impact of obesity appears to be significant as lean women with PCOS show a lower rate of insulin resistance. While I don't intend to cover every possible medical situation a woman might encounter, I do want to mention that women with PCOS are also 3 times more likely to suffer from thyroid disease (especially Hashimoto's thyroiditis, mentioned last chapter) than non-PCOS women (2). 24 The second situation where a woman might show elevated testosterone levels is currently referred to as subclinical hyperandrogenism. Finally, there is an extremely rare condition called congenital adrenal hyperplasia (CAH, which can also occur in males). For complicated reasons, individuals with CAH produce adrenal androgens at an enormously elevated level. Since it is so rare, I won't discuss it further in this book and only mention it for completeness. Hormonal Birth Control Formerly referred to as oral contraception (OC, a term no longer accurate due to other modes of application), hormonal birth control (BC) is used by a great percentage of women and is likely to represent the most common hormonal modifier readers may encounter. As they have no effect on a woman's physiology, I will not include barrier methods of birth control in this discussion. When I use the 25 abbreviation BC it should be taken to only refer to hormonal forms. As the name suggests, a primary use of BC is for its intended purpose which is to prevent unwanted pregnancy but there are other situations where BC is used. One is simply to control or regulate the menstrual cycle. This may be necessary to treat endometriosis, regulate irregular cycles (as in oligomenorrhea), to control heavy bleeding or excessive acne, or in cases of severe PMS/PMDD. For reasons I will explain below, BC is also used to treat PCOS as well. Finally, BC is often used by female athletes to regulate the timing of their cycle. Many women experience changes in performance across the menstrual cycle and it can be disastrous if an important competition falls during the part of a woman's cycle when her performance is decreased. By using BC, this can be avoided by either controlling or eliminating the changes in her physiology that would be occurring. While a potential benefit, there are also drawbacks to BC for athletes; this will be discussed in Volume 2. Of all the hormonal modifiers I will discuss in this book, the discussion of BC is likely to be the most complicated with it being somewhat incomplete. The difficulty here is that there is no single type of BC. There are multiple general categories of BC but they may differ in the types of hormones present, how they are applied and the ultimate effects that they have on metabolism. New forms and combinations of BC are constantly being developed and there is simply little to no data on their specific effects in most cases. To try and keep the individual variance to a minimum, I will be dividing the different types of BC into distinct categories in terms of their general metabolic effects and will focus only on the issues that might be relevant in terms of diet and fat loss. Types of BC Since their introduction, all forms of BC have been based around synthetic versions of estrogen and progesterone. And regardless of how it is taken or used, in the most general sense, BC can be divided into combination BC (containing synthetic estrogen and progesterone) and progestin only BC (containing only synthetic progesterone). The synthetic estrogen ethinyl estradiol (EE) has been used almost exclusively in BC for decades. In early forms of the pill, doses were very high with 150 micrograms of EE. Newer forms of BC typically have 15-30 micrograms of EE on average with the reduction improving safety and reducing side effects (higher doses of EE are only used for emergency contraception). In contrast, there are at least 8 different types of progestins (synthetic progesterones) with newer types being developed. While both EE and the progestins act very similarly to estrogen and progesterone in the body they are not identical to the hormones that a woman naturally produces. EE is significantly more potent than a woman's natural estrogen in many ways and can impact on a woman's physiology depending on how it is taken (i.e. orally vs. any other method). Progestins are even more complicated and differ in how well or poorly they bind to the progesterone, androgen, cortisol and mineralocorticoid receptor and this has an enormous impact on their overall effect in the body. The progestins are generally grouped into one of four generations based on when they were developed. They may also be distinguished by their chemical structure and what hormone they are synthesized from but the details of this are unimportant here. The development of newer types of progestins was driven by the desire to improve menstrual cycle control while reducing the side effects that were commonly seen with earlier progestins (5). The first three generation progestins were all derived from testosterone due to the similarity in chemical structure [illegible]. For example, while natural progesterone is anti-androgenic, blocking the normal signal at the androgen receptor, testosterone-derived progestins are androgenic, sending a positive signal. The androgenic effects of synthetic progestins [illegible] side effects such as oily skin, acne and body hair in addition to other effects described below. [illegible]

[illegible]

athlete isn't tired although high-level athletes may do this in order to ensure technique stays stable when they fatigue in competition. Technical training sessions are typically fairly limited in duration; the time spent on any one drill is also usually limited as endless repetition tends to cause athlete's to lose focus. Performing shorter technical sessions more frequently is generally superior for this reason. Alternating between different drills back and forth tends to have better results as the athlete has to think and focus more when they switch from one drill to another and back again. 38 Warming Up Let me next discuss warming up; any activities done before a workout and has as it's primary goal preparing the body for the workout session. Most typically a warm-up routine would include some type of low-intensity activity to generally raise body temperature (and this is more important when exercise is being done in the cold) which might be followed by some amount of stretching (if needed), technical training (if needed) and then progressively more intense exercise as the workout itself begins. The specific types of warm-up activities done depend very heavily on the type of workout being done with the importance of the warmup increasing as the intensity of the workout goes up. A low intensity aerobic workout may need no specific warm-up while a maximum sprint workout might require 45-60 minutes before the actual workout begins. Warm-ups for weight training workouts can vary enormously depending on the type of workout being done. Very heavy training tend to require the most warm-up which is usually done by performing multiple sets of the same exercise with progressively heavier weights. Specific technical drills may be done in certain types of activities as well. For more traditional muscle growth or general fitness training, less warm-up is generally needed. Although I'm not aware of any research on the topic, anecdotally women seem to require more warmup than men for high-intensity activities, especially those that are highly technical. This could be due to differences in their nervous system and some have suggested that differences in the muscles and ligament themselves may be responsible. Regardless, women involved in high-intensity activities such as strength/power or sprint training may need to experiment with their warm-ups, performing more total work at progressively increasing intensities until they determine how much they individually need to perform at their best. Cooling Down Conceptually related to warming up is cooling down and the goal here is to facilitate the body's return to normal after an intense workout. This occurs through a number of mechanisms including allowing heart rate to return to normal levels (primarily for aerobic training), to help clear waste products from muscle (for high-intensity aerobic, HIIT or weight training workouts) and, importantly to lower body temperature back to normal. Women differ here from men, taking longer for their body temperatures to decrease to normal following a workout. For women exercising in the heat, and especially during the luteal phase and/or for women on certain types of birth control (when body temperature is elevated to begin with), avoiding excessive heat buildup during training along with bringing it back down more quickly is important for recovery. I will discuss this in more detail in Chapter 22. Typically a cool-down will consist of 5-20' of very low intensity aerobic activity (130 heart rate or lower) as this type of active recovery brings heart rate down gradually while helping to remove waste byproducts from muscles. This may be followed by light stretching if needed as this can help the body generally relax so that it can start the recovery processes. The amount of cool-down necessary depends on the intensity of the workout. Low-intensity workouts may require little to no cool down as the workout itself is at a recovery heart rate while a HIIT workout might require 10-20' of low intensity activity. General Training and Diet Goals While I suspect a large majority of women reading this book will have changes in body composition (specifically fat loss) as their primary goal, I want to look at some of the individual goal or sport categories that women might be interested or involved in. For each goal/sport I'll first describe what it represents before looking at the typical combination of types of exercise that might or might not be done. For the individual sports categories, I will primarily be grouping them based on their requirements for strength, power, muscle size, endurance, etc. along with the types of training that are most commonly done. Since I can't include every possible sport in this chapter, readers will have to compare the primary training they perform for their sport with what I have described. One type of sport I won't describe is pure skills sports such as archery or pistol shooting where the training is almost exclusively of a technical nature. Bone Health Due to the importance of bone health (bone mineral density or BMD) to women I want to discuss it both first and separately from the other goals. As I will discuss later in this book, women have until roughly their mid-20's or so to develop peak BMD. Most of the gain occurs during puberty but this isn't the target readership for this book and there's little that can be done in hindsight. At most parents of young girls who are reading this book can ensure that everything is being done after puberty to maximize BMD in 39 terms of activity and nutrition. Past the mid-20's, there is typically a slow loss of bone density that accelerates at menopause, especially if hormone replacement therapy is not undertaken (the effect is due to the lack of estrogen which is also a contributor to the loss of BMD in amenorrhea). The increased rate of loss in women along with starting at a lower peak BMD is part of why osteoporosis is much greater risk for women than men (differences in average life span is also important here as men typically die before their bone density drops far enough for it to be an issue). I say typically above as emerging evidence finds that proper exercise and nutritional intake (discussed in Chapter 20) is able to increase BMD even past the mid 20's. Perhaps more importantly, some research suggests that even postmenopausal women can either slow/eliminate the age-related loss of BMD or gain small amounts of BMD outright. The effect is smaller in this population: while the post-pubertal female may gain 2-5% BMD per year, the gain is only 1-3% per year past the mid-20's and into menopause. This is still significant in that even avoiding the typical loss of BMD helps to avoid problems later in life: a postmenopausal woman who gains 1.5% BMD instead of losing 1.5% BMD is still 3% ahead. The key factors in developing or maintaining BMD are activity and proper nutritional support including adequate calories, calcium, Vitamin D and others. Since I will discuss nutrition in detail in Chapter 20, I will focus on the exercise component here. In short, the primary requirements for exercise is that it generates high peak forces and is brief and intermittent in nature. Higher levels of activity are generally associated with higher BMD but the type of exercise done makes an enormous difference in the overall effect of BMD, both in terms of the change and where that increase is seen (researchers typically focus on the lower leg, hip, spine and wrist). Studies have found that athletes in sports with a large amount of jumping such as gymnastics, volleyball and basketball, tend to have the highest BMD (at least in the lower body). Athletes who lift weights have the next highest BMD with explosive lifters such as Olympic lifters having higher BMD than powerlifters who lift more slowly. Sports with less of a high-impact or explosive component tend to have lower levels of BMD. Surprisingly, endurance athletes, especially those who's sport generates no impact forces (i.e. cycling, swimming, cross country skiing and others) often have lower BMD than sedentary individuals with running being slightly higher. These observations, combined with a number of direct studies has led to the conclusion that the best types of exercise for improving BMD are weight training and jumping activities which are high-intensity, generate high peak forces and are done intermittently (5,6). In contrast, running or walking, which generates low peak forces and is done continuously is not as effective in improving BMD except perhaps in postmenopausal women who have very low BMD to begin with. Sprint running does improve BMD but, as above, requires good technique to be done safely. Weight training, which can be used to load all bones of the body is superior in some ways to jumping which only stresses the lower body (jumping may also have no effect on BMD in postmenopausal women). So far as weight training, a key aspect is that the weights be heavy enough and put stresses on the bone directly or in unusual ways. Studies finding a benefit suggest that loads higher than 80% of maximum (roughly 8 repetitions to fatigue) are required in younger women while slightly lighter weights (12-15 repetitions to fatigue) are sufficient after menopause. Amazingly, one study in postmenopausal women used one set of upper body and one set of lower body exercises for 10-12 and 12-15 repetitions to fatigue twice weekly and that alone (perhaps 5 minutes of training) improved BMD. Since it is high peak forces that improve BMD, gradually moving to faster lifting speeds may be beneficial but it must occur gradually and be done safely. A recent study used extremely heavy loads (sets of 5) in post-menopausal women and found amazing improvement in BMD but this must be worked up to gradually. While a recent study found that jumping was beneficial for older women, most has shown that it is mainly effects before menopause. Perhaps most surprisingly is how little it takes. As few as 10-20 maximal vertical jumps (a jump where the knees are bent, the person jumps as high as possible and lands) with 15-30 seconds between repetitions done 3-6 times per week has a significant impact on BMD. This means that as little as 5-10 minutes of activity three times per week can improve BMD, at least in the legs. Given the differences in the effect of exercise and BMD in different age groups, choices of exercise should be population specific. For pre-menopausal (but not very young) women a combination of heavy, full-body weight training along with small amounts of jumping seems to be ideal. Postmenopausally, running (if it can be done safely) and heavy weight training would be an optimal combination. If a postmenopausal woman already has low bone density, the amount and intensity of training must be brought up gradually to avoid overstressing the already weakened bones. It's unclear how the other hormonal modifiers impact on this. I mentioned that birth control may negatively impact on BMD and the research on PCOS seems to have an overall beneficial effect and I would expect exercise to add to this. 40 General Fitness and Health While the reality is that many, if not most, women are interested in weight or fat loss at most points in their life, there may be some female readers who are simply interested in improving their overall health and fitness. This includes improving their overall quality of life, improving bone density (or at least limiting it's loss), avoiding age-related muscle or function loss and others. Achieving these goals can be done with roughly three hours per week of exercise which should include a minimum of two days per week of resistance training along with at least three days per week of aerobic training. HIIT is optional but may be useful for variety. Premenopausal women would want to add a small amount of jumping for bone health and older women should add flexibility training. Even when changes in body composition are not the primary goal, they often happen in the early stages of beginning an exercise program. Improving Body Composition/Appearance While athletes of varying types frequently want to improve their body composition (this will be defined in detail in the next chapter), here I am talking about the woman who is not an athlete and who has no goal of competing in any sporting activity but who wants to improve her appearance to one degree or another. In the most general sense, her training will look very similar to what I described for the woman seeking general health and fitness although she will probably be performing proportionally more exercise overall. Three to four days of proper resistance training with as many (and potentially more) days of some type of aerobic work would be common; HIIT could be done for one or two sessions per week. Stretching has little impact on appearance but can be done as desired. While it doesn't really impact on appearance, jumping should be done by premenopausal women to improve BMD. More specifically, changing body composition entails two primary goals which are gaining lean body mass (LBM, defined in more detail in the next chapter) and losing body fat. Gaining LBM occurs in response to proper resistance training (discussed in Chapter 28 of this volume and in detail in Volume 2) along with sufficient dietary protein. While a slight caloric surplus maximizes gains in muscle mass, beginning trainees often find that they gain small amounts of LBM while eating at maintenance or losing fat. Since women in this category don't generally desire enormous gains in LBM, the need for an explicit muscle gain phase would be unlikely to be included in this goal. While gaining LBM (or at least not losing it) helps to improve overall shape and appearance, losing body fat always has a much more profound effect on lowering BF% (I will show why this is the case in Chapter 7) and realistically will be a more typical goal for most women looking to improve body composition. The obese/PCOS woman may wish to lose small amount of fat to improve their overall health and/or fertility as well. As I will discuss in Chapter 8, fat loss is primarily driven by the creation of a long-term imbalance between calorie intake and energy expenditure. Calories can be reduced, activity can be increased or the two can be used together. Which approach is taken tends to depend on several factors such as how much exercise is being done along with how much fat must be lost. I will discuss these specific situations later in the book. Physique Sports This category includes women's bodybuilding, physique, figure, and bikini. All are judged primarily on appearance with factors such as overall muscularity, symmetry and body fat levels playing a role in competition results. Posing is a critical aspect of competition as well. Each subcategory of the physique sports has its own requirements for either muscularity or leanness. Typically the amount of muscularity required goes down from bodybuilding to physique to figure with bikini requiring the least. Similarly, the requirement for leanness decreases with bodybuilding/physique requiring the lowest levels, fitness being somewhat variable but generally being slightly higher and bikini usually requiring the least. I will give more specific numbers in the next chapter. The physique sports are unique in that the primary type of training that is done is not performed in the actual competition (posing is the only component has any relevance to the competition). For all physique sports, weight training tends to be the primary training modality with some amount of aerobic exercise (cardio) being done. Less aerobic/HIIT work is done when increasing muscularity is the goal while proportionally more will be done when dieting. Posing practice is typically ignored until the contest diet has begun and, even there, it may not be done in large amounts until fairly close to the contest. 41 Strength/Power Sports This category includes sports where the competition is geared around maximal or near-maximal, often single, efforts lasting no more than a few seconds). This includes powerlifting, Olympic lifting, and some of the throws (shotput, discus) in track and field. Due to the similarities in terms of training for the former sports, I'd include female strongman competition here as well although it's events are typically longer (60-75 seconds) and it might realistically be included in the next sport I will discuss. All pure strength/power sports are predicated on some degree of muscle size, strength and power production along with technique. Endurance (outside of the ability to complete long workouts) is not required. As such, the training for these sports revolves almost exclusively around strength/power training, explosive training and technical work. The types of resistance training that is done can vary but typically includes a large amount of heavy/low-repetition training (heavy weights for sets of 1-5) with some amount of higher repetition work to increase muscle mass as needed. Explosive training may include jumping exercises along with others such as medicine ball work or explosive lifting. Technical work tends to vary by the sport and will depend heavily on the sport in question. In powerlifting and Olympic lifting, the weight room is the sport although specific technical work may be done as needed. Female strongman competitors perform some combination of traditional weight room work along with practicing with the implements while throwers typically lift and throw and lift and throw and then lift and throw some more. As it can hinder the development of strength and power, low intensity aerobic work outside of the lowest intensity activity (i.e. brisk walking) is almost never done. At most what is often called work capacity or general physical preparation (GPP) is done and might involve sled dragging or light or performing barbell complexes (a series of exercises done continuously). HIIT would be universally inappropriate (except perhaps for female strongman) and true sprinting would make more sense for athletes in these sports. At the same time, the technique requirements are high and the impact can be a danger for heavier athletes. Several sports in this category have weight classes which means that athletes cannot continue to gain weight unless they intend to compete in a higher class. With one exception, weight class athletes tend to maintain a reasonably but not excessively low BF% as this lets them carry more muscle at any given body weight. Depending on their weight class and how far away they are from their competition, athletes may actually be cutting their weight within a fairly small range (about 5% of total body weight), the total amount of fat that needs to be lost is decreased. The lack of true aerobic work in strength/power sports along with the fact that weight training burns proportionally fewer calories than other types of training can make fat loss relatively more difficult since it's hard to increase calorie expenditure without harming performance. This means that reducing calorie intake/adjusting the diet itself tends to be the best approach for fat loss. The exception to the above is weight class sports where a woman has a specific goal weight to make. Athletes in this group often carry a significant amount of body fat as it often improves their leverages and allows them to eat enough to support their training. Fat loss is rarely a goal until these athletes retire from competition. High-Intensity Performance Sports Although all sports tend to require a high-intensity in both training and competition, I am using the term to refer to sports that still have a large requirement for strength, power and explosiveness but which is lower than the pure strength/power sports. Speed is often a requirement and for certain events, there may be some endurance component although it is not very large. Some of the sports that might be included here are the 100m/200m sprint in track and field, some track cycling events (i.e. match sprint), sprint swimming events (50-100m) along with others of that rough duration. Other sports where the competition event falls between roughly 20 seconds and 1 minute would be included here. Cheerleading figure skating, and gymnastics also fall into this category. While the duration of the competitions tends to be longer (i.e. the free skate program is 4.5 minutes and gymnastics routines vary in length) the requirements for strength and power are more similar to the other sports in this category than not. Generally they revolve around strength, power and explosiveness and alternate between near maximal explosive efforts (i.e. a jump in figure skating) and relatively easier recovery. As described above, athletes in these sports often show the highest levels of body fat due to the explosive nature of their sport. In addition to a usually staggering amount of technical work, sports in this category tend to revolve around strength, power and explosiveness than much else. Many of these sports have a high requirement for maximum speed and this makes up a large amount of training as well. While relatively more endurance is require for these sports, the amount of true endurance training tends to be at least somewhat limited or done in very specific ways. Track sprinters almost never perform true endurance work. Cyclists may perform 42 specialized workouts for both general and speed endurance. Track cyclists often ride their bikes at low intensities for an hour a few days per week but this is for recovery (or possibly to reduce body fat slightly), is non-specific and it doesn't represent recovery or higher improvements in the event specific. For example, all cyclists simply enjoy riding their bikes. For most of the described sports, true HIIT is rarely done although pure sprint training is par and parcel of the training due to the requirements for a high top speed. Cheerleaders and figure skaters may do minimal true aerobic work as much of their conditioning comes from practicing routines/skills; HIIT may also be done. Depending on the sport, increasing amounts of muscle mass can be relatively more or less beneficial; the same goes for reducing BF%. Track cyclists and sprinters will tend to carry the most muscle since their body weights are being supported and, within limits, BF% is often somewhat higher. In contrast, excessive muscle mass can potentially slow down a track sprinter although their body fat levels tend to be very low. Sports such as figure skating or gymnastics have an aesthetic component that can't be ignored and excessive muscle mass and body fat can be detrimental to performance. At the same time both sports have a large strength and explosiveness component which needs to be trained. Typically a fairly large total amount of training is done in these sports and that alone is often sufficient to keep women who do them fairly lean. If fat loss is desired, it may be possible to add low-intensity work (i.e. extending warm-up and cool-downs) to burn extra calories while adjusting diet slightly. I'll mention and will discuss again that athletes in many of these sports have already habitually undertrained and if diet is adjusted at all, it may actually be to increase food intake. Mixed Sports I will use the term mixed sports to refer to activities that require a relatively even balance of strength/power along with endurance. Explosiveness and speed are often important as well. The majority of team sports such as basketball, ice hockey, netball, field hockey, volleyball and softball and others fall into this category but individual sports such as the middle distances (400-800 m) in track and field or many swimming events along with mixed martial arts or boxing would be included here as well. Athletes needn't be as strong as athletes in the strength/power or high-intensity performance sports but need more endurance than either (but less than endurance athletes discussed next). Given the nature of these sports, the training tends to be far more balanced in terms of the different types of training that are required. Strength and power work, explosive training, sprint training and general aerobic/anaerobic work may all included and they all tend to be performed at some point in the week. Alternation of higher intensity days with lower intensity days is a common pattern and there is often also the need for technical work along with tactical training, especially in the team sports. Generally, different aspects of training are emphasized at different parts of the year moving from more general conditioning to more specific competition work as the season approaches. In the team sports especially, competitions may occur weekly with the competition season lasting for several months at a time. Developing optimal muscularity along with maintaining a reasonable BF% is also typically an aspect of these sports. As with some strength/power sports, MMA and boxing have weight classes which may require explicit dieting and/or water manipulation to make the weight limit. To at least some degree, the sheer amount of training being done tends to keep these athletes lean but losing fat may require some changes to training (such as increases in aerobic activity can burn significant calories. Since the diet is only source for fat loss. Endurance Sports (Finally) are the endurance sports which are defined as events where the competition lasts 4 minutes or more (most competitions are much longer than this). Examples include running, cycling, mountain biking, rowing, the longer swimming races, triathlon, cross country skiing, race walking and others. Events can last from just over 4 minutes (in the 1500m in track and field) to 6-7 minutes (rowing) up to several hours (the marathon/ultra-endurance running, cycling and triathlon). There are even ultra-endurance events where the athlete may be active for hours or more a less continuous movement for many hours at a time and women actually outperform men in ultra-endurance running events longer than 52km (32 miles). As the name suggests, the primary determinant of performance is aerobic endurance although there are others such as lactate threshold (this has other names but can be thought of as the maximum speed which can be maintained for an hour), efficiency and technique. Top speed is also important either to 43 improve speed at lower intensities or to give the athlete the ability to catch a competitor or sprint at the end of the race. Even in the shorter events such as the 800m (which may last no more than 2 minutes), the predominant type of training (up to 80%) done in endurance sports is relatively low- to moderate-intensity aerobic work complemented by relatively small amounts (perhaps 20%) of HIIT of varying durations. Small amounts of true sprint work may also be done. Aerobic work may be done most days per week (one per day) with HIIT typically done no more than twice per week (especially for runners). Some endurance sports such as swimming and rowing are highly technical and specific technique workout are often done. With the possible exception of rowing, which requires a good deal of strength at the start of the race, weight training does not typically make up a large amount of training for endurance sports. Excessive muscle mass can be detrimental in most cases although swimmers and rowers tend to be more muscular than other athletes as their sports require proportionally more power and they don't have to go up hills. For this reason, it would be almost unheard of for an endurance athlete outside of a rower or swimmer to explicitly try to gain muscle mass. It's actually not unheard of for endurance athletes to want to lose muscle mass, especially in muscles that don't contribute to their sport (for example, upper body muscles in running or cycling). While some research has found that weight training and jumping may improve performance (especially in running), both types of training tend to be relatively de-emphasized for most endurance athletes. As it most is generally used in the of-season, especially for athletes who live in wintery areas and can't train easily. Once the competition season approaches, it is eliminated from training as often as not. While this is logical in a purely competitive sense, it is actually a large problem in terms of developing or maintaining optimal BMD. Only running puts any type of impact stress on the bones (and then only in the lower body) but it is not a high peak force and the effect is not large. Swimming and cycling not only have no impact forces but the body is supported and they may show poorer BMD than sedentary individuals. One study even found that cyclists lost bone density during their 4-month competition season despite the inclusion of weight training and jumping. All of these factors, when combined with issues such as menstrual cycle dysfunction or outright eating disorders, has the potential to not only harm these athlete's BMD in the short term but set them up for problems much later in life. Not only are endurance athletes not building BMD during the critical years, they may be losing it. For this reason, the inclusion of weight training with some jumping in at least the off-season of training (coupled with proper nutrition as described in Chapter 20) should be considered mandatory for all female endurance athletes. This may not be optimal in the sense that it is only done for part of the year but the realities of high-level competition are that compromises have to be made. If possible, it would be ideal to maintain at least some amount of that type of training during the competition season but that may not be realistic due to the competition demands and amount of training that must be done to meet them. With few exceptions, endurance athletes tend to want to either lose body weight and body fat levels. Body fat is, in a very real sense, dead weight that they are carrying and serves no purpose. Any excess body fat is simply extra weight to move (especially up hills) without contributing to performance. Runners are typically the leanest of all as they have to project their bodies across gravity with cyclists carrying slightly more fat due to being supported on the bike. Swimmers often carry slightly more body fat than cyclists. Not only does this not harm performance it may help because it makes the athlete float more easily which means that less of their energy goes to staying on top of the water. Cold water swimmers (another sport where women outperform men) tend to carry more fat as well, not only to help them float but because it acts as insulation. Rowing is unique among endurance sports in that it has two weight classes and female rowers may have to actively cut weight to make the weight cutoff. Since dehydration tends to harm performance, it cannot always be used and this means that fat loss may be the only approach to make the weight cut off with some women potentially being unable to realistically reach the lower weight class. From an energetic standpoint, with the possible exception of ultra endurance events, body fat is never limiting for an endurance event and in the sense that it is effectively deadweight, reducing it within limits does tend to improve performance. At the same time, reaching extreme levels of leanness and what is required to do so can cause a number of problems. These will be discussed in Chapter 12. When fat loss is desired, endurance athletes have the benefit of already burning a large number of calories through their training. Adding more activity may not be possible without being counter-productive and this means that reducing food intake is often the major factor. A small increase in low-intensity work (i.e. lengthening warm-ups or cool-downs for harder workouts) is often possible but adjusting the diet is usually the only realistic approach in most cases. 44 Chapter 5: What is Body Composition? Having examined the normal menstrual cycle and the most common hormonal modifiers, I want to discuss the topic of body composition for the next several chapters. There are two primary reasons I want to do this. The first is that the differences in body composition between women and men (detailed below) tend to underlie many of the differences in sports or perhaps more accurately the dietary or training differences that exist between them. Hormonal differences (and the changes that occur) clearly play a role but the difference in body composition tends to explain a great deal of the differences that are seen. The second has to do with a topic that will take up a large portion of this book which relates to dieting and what I will for now call weight loss. I mentioned that women are generally more likely to be dieting than men and this is true whether the general population or athletic population is being examined. There are still many long-held misconceptions and simply poor ideas about dieting and many of them relate to a misunderstanding of the differences between body weight, body fat and body composition. Because while many may pursue dieting to try to lose body weight, it isn't generally speaking body weight that they actually care about losing. Looking at body composition and what it means is critical for understanding why it is that weight loss isn't the goal but rather fat loss. Weight loss isn't always the goal. Since it's also the goal for many who are not dieting to lose weight or are not dieting at all. While I will be talking primarily about fat loss in this chapter, there is more to it than that and weight loss is not the goal. Rather, the goal is to change or improve the body composition. Which is not to say that the scale doesn't have its uses or that weight is irrelevant in all situations (i.e. weight class athletes who must reach a specific weight). But for everyone else, there are a number of potential problems with it by itself. To nobody's surprise, there are a set of issues that women face in this regard that men really don't. Understanding body composition, what it means, along with the differences between body weight and body fat, are a key aspect of improving women's results in everything from dieting in general to improving their athletic performance. Women's Body Composition So what is a woman's body actually made of? The answer is a whole bunch of different things including bones, skeletal muscle, organs (heart, liver, kidney, brain, etc.), water, stored carbohydrate, blood, minerals and of course there is body fat. For simplicities sake, these different components of the human body are typically divided into two categories. The first is fat and includes, well, all of the different types of fat that I will discuss in some detail below. Everything that is not fat will be called lean body mass (LBM) and you'll sometimes see this called Fat Free Mass (FFM). For all practical purposes they are interchangeable and I will use LBM throughout this book. What is LBM and What is it For? While many, especially in the athletic community, tend to equate LBM with muscle, this isn't really accurate. Rather, LBM refers to everything that isn't fat and this includes a number of distinct tissues which are structurally very different. The brain has a very specific structure as do the various organs (including a woman's reproductive organs). Bone is it's own tissue as is skeletal muscle. Water, minerals and carbohydrate are not tissues at all but are all part of LBM in the body even if they are not integral parts of a specific tissue. Bones provide the physical structure of the body along with providing a physical framework, skeletal muscle generates force for movement, reproductive organs exist for reproduction, etc. All are important although, as you'll see later in the book, some are relatively more important than others in terms of short-term survival. They are all important but as I will talk about below, only a few are really that relevant in terms of what can or cannot be impacted on by diet (or training) and what is really worth paying attention to in the short term. While the amount of bone, or rather how dense bones are, is critical to women's health, the only type of LBM that is important in terms of altering body composition is skeletal muscle. Other types of LBM can change, water and glycogen for example, but changes in the amount of muscle are key here. Skeletal muscle is made up of a number of different types of tissue. What most people think of as muscle is a protein (Myosin) that are basically the muscle fibers that are made of protein but this is only about 25% of the total in muscle. The rest is a combination of water, minerals, stored carbohydrate (called glycogen), intra muscular triglyceride (IMTG, fat stored within the muscle itself) and the various cellular machinery involved in muscular metabolism. What is Body Fat and What is it For? In contrast to LBM which is made up of a number of very distinct types of tissue, body fat tends to be fairly similar in its chemical structure with one exception. The technical term for body fat is adipose tissue and most types of body fat are made of white adipose tissue (WAT) although it's really more of a beige color. Whether they know it or not, when people want to lose "weight" or improve their 45 appearance, it's WAT that they want to lose. All WAT is made up primarily of stored triglyceride (TG), and this makes up 85-90% of the total fat cell (the rest is water and cellular machinery). A TG is the combination of three fatty acids attached to a glycerol molecule. When people talk about saturated or unsaturated fats they are actually referring to the chemical structure of the fatty acids chains. The fat found in food is nothing more than TG and I'll talk about how women's bodies handle dietary fat in Chapter 10. The exception to the above is what used to be called brown adipose tissue (BAT) but is now thought to be brite or beige adipose tissue in humans (the distinction isn't that important here and I'll call this BAT as well). Sort of a reddish color, BAT stores very little triglyceride and exists to burn other fuels for energy and to produce heat (1). It's currently not clear how much of a real world impact on calorie expenditure BAT has at this point. As well, since BAT tends to be primarily activated with exposure to cold, which most in the modern world try to avoid, the relevance of BAT is questionable. So what is body fat for beyond making people unhappy about their appearance? The earliest ideas held that body fat was nothing more a relatively inert place to store energy and clearly that is certainly one of it's primary purposes. During certain types of exercise or when there is insufficient food (as in dieting or starvation), stored fat is mobilized to provide energy to the body. While carbohydrate can also provide energy, fat stores are especially useful for this as it tends to provide 9 calories per gram while carbohydrates only provide four. As, importantly, the storage of carbohydrate requires a large amount of water with 3-4 grams of water being stored for every gram of carbohydrate, while fat does not. A fairly lean individual might store 100,000 calories of fat, enough to sustain them for weeks or months without any food. To store that same amount of energy as carbohydrate would be impossible and the actual stores of carbs (as glycogen in the muscle and liver) is fairly limited. For women especially, it's clear that lower body fat provides a fuel source during pregnancy and for breastfeeding. As I mentioned in Chapter 2, women's hips and thigh fat is actually used preferentially for this purpose. This may seem (and be) problematic for women but it is a biological requirement. Related to this, at least part of a woman's body fat distribution is probably related to sexual selection and attraction, providing the curves and other female characteristics that are found to be sexually appealing. But there is far more to body fat than storing energy. Body fat turns out to be crucial in both immune system function and inflammation with both too little and too much body fat causing problems. Too little fat means that the immune system may not function as well as it could while too much means that the immune system is overactive. Excessive body fat also can cause an inflammatory state. Body fat may also play a role as a physical cushion in the body and for certain types of athletes, a higher body fat could protect them against injury. Body fat is also a place where the body stores glucose; it becomes impossible for the body to store incoming carbohydrate in muscle and fat cells can take up the slack at least for a little while. As I mentioned in Chapter 2, fat cells are also a place where local metabolism of hormones can occur. A great deal of women's estrogen is actually made from the conversion of testosterone within the fat cell (in postmenopausal women, almost all of her estrogen is made this way). Fat cells also can impact on cortisol (a stress hormone) metabolism, converting active cortisol to inactive cortisone and vice versa and there are other numerous effect occurring with more being discovered almost continuously. Perhaps one of the most newly recognized aspect (newly here means since the mid 1990's) is that fat cells, produce a host of chemicals and hormones that drastically impact on physiology. Leptin, which I mentioned in Chapter 2 and which I will talk about in great detail later, was the first to be discovered and the list continues to grow almost weekly. My point here is primarily to point out that, as much as people dislike body fat (for appearance reasons) and while excesses certainly cause health problems, fat cells are critical for overall health and function. Too little can be just as bad as too much and in older women it can cause problems (low estrogen levels are a potential issue). This is simply an issue when people where makes no fat cells, a number of health problems crop up. So it's simply an issue that thinking of fat cells as "good" or "bad" is mistaken (3). Types of White Adipose Tissue While the above applies to all types of WAT generally, it turns out that fat stored in different parts of the body can act very differently. Different distributions of fat (i.e. upper versus lower body) can impact on overall health and there are large differences in the rate of blood flow through the fat cell which will impact on how fat gets in and out of the fat cell along with how easily or not they release that fat back into the bloodstream. There are also clear gender differences that I will discuss in a later chapter. For now let me look at the different types of fat. 46 The first type of fat is essential fat, in the body that is essential for both life and normal function. This includes fat around the brain, around the internal organs (different from visceral fat, discussed next), in the nervous system (sheath around nerves are made of fat) and in the brain. In general, essential fat is taken as 3-4% for men and 10-12% for women with the difference being attributable to what is called sexspecific fat (breast tissue is included here). You can't lose essential fat and if you did, you'd be dead. Visceral fat, which many readers have probably heard of, refers to a type of fat found primarily in the gut that surrounds the organs (it is different from essential fat, though). Visceral fat is highly metabolically active meaning that, while it stores fat fairly easily, it also releases fat easily. This is probably to provide a rapid source of energy to the body but excess amounts of visceral fat is associated with insulin resistance and increased heart disease risk (4). Visceral fat is deep within the body and not really visible outside of making the stomach stick out (and often feel quite hard to the touch). When it is lost, while the stomach may be flatter or easier to suck in) there is no major change in appearance. Testosterone tends to promote visceral fat accumulation and between having low testosterone and elevated estrogen, women do not generally store much. However women with PCOS/hyperandrogenism or who become overweight tend to store visceral fat. After menopause, visceral fat levels increase if HRT is not begun which contributes to the increased risk of heart disease seen in women under those conditions. Subcutaneous fat is fat found underneath the skin which makes it visible in a way that essential and visceral fat is not. Whether they know it or not, when people talk about losing fat (or even weight to some degree), they are really talking about losing this type of fat. While subcutaneous fat used to be considered a single type of tissue, it's now known that fat in different parts of the body are physiologically distinct (I will discuss this in more detail in Chapter 6). Upper body fat is more similar than not and represents everything above the waist including fat on the face, shoulders, chest (except breast fat), upper and lower back and abdominal area (which can be further subdivided into deep and superficial and upper and lower). Lower body fat refers to everything below the waist including the glutes/hips, thighs and calves. Since they have less visceral fat, women tend to carry more subcutaneous fat with more of that found in the lower body (except for PCOS/hyperandrogenism, obesity and the postmenopausal woman on HRT tend to carry more upper body fat). Relative to visceral fat, subcutaneous fat is less metabolically active than visceral fat which means that it tends to have less of an impact on disease risk. Carrying more fat in the lower body, which is particularly metabolically inactive, lowers heart disease risk which is why women are typically protected until after menopause. What is Body Composition? With the above background, I can finally address what body composition represents. Fundamentally it refers to the ratio of all of the different tissues in the body that I mentioned above. So assume we could determine how much of a woman's body was made up of every kind of tissue in the body: muscle, fat, bone, water, stored carbohydrate. This would represent her total body composition. Perhaps she might find that she had 40% muscle, 25% body fat, some percentage of brains, liver, kidneys, reproductive organs. Bones would make up some other percentage, water, minerals, stored carbohydrate another percentage. When all of this was added together, it would add up to 100%. Let's also assume that we could determine the weight of each tissue. If we took the weight of each tissue and added them together, it would add up to her total bodyweight. So if our woman weighed 150 pounds total and we know she was 40% muscle, 25% fat or 34 lbs would be fat, and the same would hold for every other tissue in her body. And that breakdown, the percentage of her body that was represented by every type of tissue present would be her body composition. Essentially, it describes how I moved an entire tissue at a time. However, here I go to this level of detail and it's more common to delineate the body into fat and everything else (LBM). This is important for a number of reasons not the least of which is that weight may remain the same even if body composition is changing. Of more relevance to the next section, it's important because weight gain or loss can occur over different time frames and in response to the gain or loss of a variety of different tissues. Losing and Gaining Weight: What is Being Lost or Gained? Although it has been changing in the last decade or so, the reality is that most people (women or men) tend to focus only on changes in body weight. If weight goes up, that's usually bad and if weight goes down, it's usually good. Even the most athlete trying to lose weight (or gain it) worry about it. Take the example, let's say someone starts a diet and a few days later their scale weight has dropped by a few pounds. Most would consider that a success but I would ask the following question: What was lost? Was it water, stored carbohydrate, muscle, fat, bone, organs? Perhaps the person just had a big bowel movement. 47 The scale can't answer this question in any meaningful fashion and this presents a rather large problem. And this becomes an enormous practical problem as many people, especially if they are dieting, not only focus solely on the scale to track their progress but often obsess over the changes (or lack thereof). They may weigh multiple times per day (or before and after going to the bathroom) and then freak out over completely to small day-to-day changes in bodyweight. A frequent pattern is that as weight goes down, it's time to celebrate. Bring on the cake. But if weight goes up, it's time to reduce food intake even more and add an extra hour of exercise to the gym. As you'll see below, these types of shortterm changes are relatively meaningless overall although daily weighing can still be useful so long as it is approached correctly. Women, primarily the normally cycling female who must deal with a regular monthly changes in body weight, have a much more difficult problem here that I will discuss below. When looking at weight loss or weight gain, there are usually some safe assumptions that can be made about what is being gained or lost. Surprisingly, there can actually be some small changes in organ size but these are impossible to measure, happen rapidly and probably don't represent much total weight in the first place. Bone density also changes but only in both directions but those changes tend to be fairly slow over time and aren't anything that can be measured quickly. During weight loss, bone loss is at most 1-6% of the total loss and gains in bone density are highly variable ranging from as little as 0.5-2% in younger individuals. Even so, it's only with very large changes in the realm of 2-3% over 6-12 months with proper training and nutrition. Since the changes are so small and over any reasonable dieting time frame and can't be measured easily (only one method of doing that) discussed in the next chapter can measure BMD), this usually isn't worth worrying about either. Practically this means that the only body tissues worth worrying about are water, the carbohydrate stored within muscle (called glycogen), body fat and muscle. Even the amount of undigested food moving from the gut through the colon before excretion can actually make up 3-7 lbs (~1.5-3 kg) depending on the diet (high-fiber diets tend to produce more food residue) and this can be a significant portion of a woman's total bodyweight in some cases. But over most realistic time frames (i.e. the months that most diets will last), those are really the only tissues that need to be worried about. And the basic bathroom scale can't differentiate between them (a special kind of scale discussed in the next chapter attempts to do this). Two pounds of water loss and two pounds of fat loss will show up identically here in terms of weight and for the majority of practical situations, with the exception of weight gain where an increase in scale weight can't give any indication of what is being gained. That said, there are some general comments that can be made regarding the relative contribution that changes in food, water, carbohydrate, muscle and body fat may be making changes in body weight and over what time frame. Water/Glycogen/Food Residue Almost without exception, very short-term changes in scale weight tend to represent changes in water, glycogen or food residue (a good bowel movement can cause a 1-2 pound weight loss in some cases). Even small changes in the diet can cause scale weight to change pretty significantly in a fairly quick period of time. Someone on a low-sodium diet who eats high-sodium meal may bloat up for a day or two, gaining several pounds of water weight. Chronic stress can also cause water retention due to the increase in cortisol which, as I mentioned in Chapter 2, binds to the receptor involved in water retention. Dietary carbohydrate intake can enormously impact on water weight. Through a variety of mechanisms, when carbohydrates are lowered, the body tends to lose a lot of water; losses of 1-15 pounds in a few days have been seen in studies of low-carbohydrate diets. Many diet books use this to their advantage. By lowering or eliminating carbohydrates from the diet, body weight drops enormously in a few days and this can be very rewarding to the dieter (5). It can also backfire when dieters get frustrated that the rapid losses don't continue indefinitely. They may lose 5 pounds in the first week due to water loss and then lose "only" 1-2 pounds per week after that. This works in the opposite direction as well: someone who has been on a low-carbohydrate diet who eats a lot of carbs for one reason or another can see their weight spike fairly significantly (large individuals may gain 7-10 pounds in one or two days). Every gram of carbohydrate stores 3-4 grams of water with it which explains the big increase in body weight. As I mentioned in Chapter 4, this partially explains why women often "feel bulky" when they start weight training. Their muscles start storing more carbohydrate and this causes them to store more water. But it goes away within about a week. While all of the above is true for both women and men, it is only women who have to deal with the added issue of the menstrual cycle (recall that some forms of birth control can cause water retention and PCOS 48 women may experience issues at nearly random times). Most women are familiar with how wildly their body weight may change throughout the month. I discussed the hormonal reasons for this in Chapter 2 but the late follicular and late luteal phase tend to be the worst and body weight changes of 5-10 lbs (~2.5-5 kg) are not unheard of. This tends to generally drive women crazy (even women well versed with body composition can be affected psychologically by this) but it gives women an additional factor in tracking changes that men don't have to deal with. What matters is that these water weight changes, small or large, don't mean anything meaningful in terms of reaching their goals. Rather, given the actual rate of change in levels of body fat and muscle mass, these rapid changes can only represent the gain or loss of water, carbohydrate, food in the gut, etc. As I will discuss in a later chapter, storing a pound of actual body fat takes about 3,500 calories over maintenance which makes a true loss or gain of 4 pounds of fat a physiological impossibility as it represents 14,000 calories of energy. To eat that many excess calories or burn that many calories through exercise in a day would be impossible. The above should not be taken to mean that body weight is meaningless or that the scale is useless. Rather, it must be understood that short-term changes in bodyweight don't represent anything meaningful in terms of what people are trying to lose (or gain). As well, the scale can still have its place to track progress, it must simply be used appropriately. As well, in at least one case, body weight does matter. This is for those weight-class athletes who have to reach a specific weight for their competition. In this case, manipulating water, glycogen and food residue it useful but with the understanding that it is only a shortterm change to achieve a specific goal. I don't know that this represents a majority of this book's readership and the reality is that, under most circumstances, the focus of dieting (or weight gain for athletes) should be on the changes in either body fat or LBM (specifically muscle). Fat and Muscle As short-term changes in body weight always represent water, glycogen, etc. they don't particularly count in terms of actually changing body composition. Certainly it may change a little bit with water (or glycogen and food residue) changes but overall changing the ratios of body fat and skeletal muscle is how body composition changes. When trying to lose (or gain) weight, individuals should really be focused on the changes in either fat or skeletal muscle but the goal of fat loss is always to lose as much fat as possible while the muscle gain should ideally come with as little body fat gain as possible (it's usually impossible to avoid it completely). I'd note that the scale still can't differentiate changes in fat and muscle either; they do occur over a much longer time scale than changes in water, glycogen or food residue. So while a 2-3 lb (~1-1.5 kg) change in body weight over a day will assuredly represent nothing more than water weight or food in the gut, that same change over multiple weeks is far more likely to represent a "real" change in body composition. Even that conclusion can be problematic depending on when body weight is actually measured. If someone only measures once per week (i.e. on Monday) the change from the first to the second measurement might represent a real change or might not. I'll talk about tracking in the next chapter. For now I just want to emphasize my primary point which is that short-term changes in body weight represent water or glycogen or food residue weight, etc. while the long term changes are far more likely to represent real changes in the amount of fat or muscle someone is carrying. That said, when females want to improve their body composition, it is usually in terms of fat loss, muscle gain or some combination of the two. Body Fat Percentage While the overall concept of body composition has to do with the fact that the human body is made up of different amounts of different tissues, the goal should really be something that most people can understand: body fat percentage (BF%). This represents the percentage of someone's total weight that is made up by fat (by definition everything else is LBM). If someone has a BF% of 30%, the other 70% is LBM. If their BF% is 20%, they have 80% LBM. I'll present some representative numbers later but this percentage can range from a lower limit of 10-12% in the leanest woman up to 5060% in cases of extreme obesity. And while body composition in the most general sense is more associated with appearance and health than weight per se, BF% is arguably even more related to both. A woman at 25% body fat will not only have a different appearance but is likely to be healthier than one at 45% body fat I'd mention again that the distribution of body fat is also relevant here and a more typical female fat 49 patterning (gynoid or pear shaped with body fat in the lower body) is metabolically healthier than the typical male fat patterning (android or apple shaped). Even if someone gains a small amount of muscle, their health will improve even if their body weight per se is unchanged (6). Within the context of this book, perhaps the larger attention to be more worried about BF% than body weight (with the exception being athletes who have to worry about a specific weight). Two women at the same weight with a different BF%/body composition may look totally different. So consider two women who both weigh 130 pounds. The first is an athlete at a relatively lean 18% body fat while the second is inactive with 28% body fat. Their weight is the same but their body composition is different and the athlete will be visibly more muscular and leaner than the inactive woman. And while body weight can change due to shifts in water weight or food residue without any real change in body composition, it's entirely possible for body weight to stay the same while body composition is improving. So consider a beginning exerciser who gains 2 lbs of muscle while losing 2 lbs of fat. Her weight will be unchanged although her body composition will have improved. Or consider a more extreme example where a woman starts at 130 lbs and 25% body fat and 6 months later she is 130 lbs at 20% body fat. She may look completely different while weighing the same. Related to this, the predicted weight loss (or gain) may be lower than expected due to changes in body composition. If a woman gains 2 pounds of muscle while losing 4 pounds of fat, she will have only lost 2 pounds on the scale although her fat loss is double that (this will come up again in Chapter 11). Her weight will only go down by two pounds but the impact on her body composition, health, appearance and how her clothes fit may change significantly. Finally, this can work in the opposite direction as well and body composition can worsen while weight is stable. I mentioned an example of this in Chapter 3 when I discussed how some forms of hormonal birth control can cause a slight loss of muscle and gain in fat despite no real weight change. The same occurs at menopause without hormone replacement therapy, even if a woman's body weight doesn't change much, she will start to lose LBM and gain fat (along with a shift in fat distribution). Weight is unchanged but body composition is worsening. For athletes or those actively trying to gain weight, the above concept works exactly in reverse. Here the goal is usually to gain as much muscle as possible while gaining fat or reducing the gain of body fat. Here too, the scale can't tell the whole story and increases in body weight indicate that either muscle/LBM or fat is being gained. In general, when people try to gain weight it's in an attempt to gain LBM while limiting the amount of fat that is gained and tracking changes in body composition is more meaningful than changes in weight per se. There are exceptions, those recovering from true eating disorders or after an extreme diet, where weight gain must come with an increasing amount of body fat (along with regaining lost LBM). Because regardless of what is or isn't happening to body weight it's the changes in body composition that matter. And this may mean losing fat, gaining muscle or both. And outside of those weight class athletes who have to reach a specific goal weight, it's those long-term changes in body composition that really matters. Having a measurement or estimate of BF% is important for tracking changes over time and a variety of calculations can also be done with these numbers and I will present them throughout this book. As well, some aspects of diet such as protein intake are better set relative to LBM so being able to calculate or at least estimate this is important. Finally, these measurements and calculations allow someone to much more accurately track what is changing in their body than they would be able using body weight alone. Let me note that for the rest of this book, I will refer almost exclusively to BF% as it is the value with the most relevance. 50 Chapter 6: Measuring and Tracking Body Composition Continuing from the last chapter, I want to now look at some of the practical aspects involved in both measuring and tracking changes in body composition or body fat percentage (BF%). I will be discussing a number of methods but it's important to realize that none provide more than an estimate. Every method has pros and cons and while I will use that estimate to determine many aspects of diet setup later in the book, from a tracking standpoint, changes are more important than absolute numbers. Since they can also be relevant, I will also be looking at some non-body composition methods of tracking progress in this chapter and will make recommendations for combinations of those methods that can be used. I'll also examine the issue of what "good" BF% might be for different situations, introduce the diet Category system I will use in this book and look at some female specific issues relevant to the topic. True Body Composition Measurements The first set of methods I want to discuss are true body composition measurements in that they measure (or estimate) some aspect of actual body composition. They vary in their accuracy, difficulty of use and availability and I will describe them more or less in order from least to most complex. Body-Mass Index (BMI) The BMI is a fairly old measurement which relates an individual's body weight to their height. More technically BMI is defined as weight in kilograms divided by height in meters squared but this can be converted to inches and feet for Americans. For decades BMI has been used to indicate general health or some kind of ideal weight and insurance companies use some version of it to determine how much to charge you per month. A high BMI tends to correlate with health risk and a BMI greater than 25 kg/m 2 is defined as overweight and a BMI greater than 30 kg/m2 is considered obese. Very low values are equally problematic with a value below 18.5 kg/m2 is considered unhealthy or malnourished (possibly indicating an eating disorder or wasting disease). Between 18.5 and 25 is considered optimal. It's critical to note that these are only averages and it's been established that individuals with a high BMI can be healthy while those in the optimal range may be unhealthy. Part of the reason for this is that BMI is not strictly speaking a measurement of BF% and doesn't indicate body composition or how much fat or LBM someone is carrying. Two females who are 5'7" tall and who weigh 150 pounds have the same BMI. If one is an athlete with 20% body fat and the other is inactive at 35% body fat, not only is their body composition different but so are their relatively health risks. It's also not uncommon for active individuals, generally males, to be told that they are overweight due to a high BMI score although they are relatively lean and simply carry more muscle mass. This leads to active individuals to often suggest that BMI should be thrown out for being useless but this is an over reaction. BMI was never meant to be used in an athletic population. In the general public, it is simply not that common to find people with high BMI who also have a low BF% although people with a low BMI often have a fairly high BF% (they are often called skinny fat). It's also possible to have a high BMI and be metabolically healthy or a low BMI and be unhealthy (1). But no body composition method is perfect and BMI is not useless, it's limitations simply have to be acknowledged. For that same group, BMI will give at least a rough indicator of general health risk along with giving a fairly easy way to track changes from diet and exercise (technically since height is not changing, tracking body weight would provide the same information). And while BMI has primarily been used to track overall health trends, it turns out that it can give a rough estimate of BF% (2). A calculator to determine BMI can be found here: That BMI value can be used to estimate BF% here: Due to its easy of use, I have used this method in my books for over a decade. And while inappropriate for athletic individuals, I think it is probably the easiest approach for people first starting out. It's quick and easy, provides a good starting point and can be used to track changes over time. Once someone has been working out consistently for 6+ months, I would not consider BMI to be accurate and they should use another of the described methods. 51 Tape Measure/Circumference Measurements Although decidedly low-tech, it is actually possible to get a decent estimate of BF% with nothing more than a tape measure. The military has often developed a lot of these equations since they need to be able to measure a lot of people quickly and easily. There are online calculators that estimate BF% in this fashion that generate results that are at least similar to more complicated methods which can be found here: Even if they are not used to track body composition per se, tape measure measurements still provide another way to track general progress while dieting (or attempting to gain muscle). During a diet, a decrease in circumference measures (i.e. diameter of the hips or arms) generally indicates body fat loss and it's not uncommon to see this occur even in the absence of much weight loss. Muscle is denser and takes up less space than body fat so someone gaining some muscle while losing fat should still see a reduction in their tape measure measurements. Taking a variety of measurements including arms, bust, waist, abdomen, hip and thighs can provide a general indication of whether fat is being lost and specifically from where. Even a single trouble spot (i.e. arms or thighs) could be tracked in this fashion. Whether used for BF% estimation or just as a general tracking method, the tape measure is not without problems. First and foremost it's critical to always measure at the same spot, around the largest part of the bust or halfway down the thigh or what have you or the values can't be compared to each other. This is not always easy and even slight differences in where the measurement is taken can make them inaccurate. It's also important to at least try to pull the tape measure to the same tension every time, neither too tight nor too loose. This can also be very difficult to do and there are tape measures with a spring on the end such as the Gulick II which will improve the accuracy of measurements. Waist/Hip Ratio (WHR) The waist/hip ratio is exactly what it sounds like, that is the ratio of the waist (measured with a tape measure at the narrowest part) and the hips (measured with a tape measure at the widest part). Technically the WHR ratio is not a measure of body composition but a measure of body fat distribution and health risk. On average, women tend to have a lower WHR than men but WHR can go up with menopause, PCOS/subclinical hyperandrogenism and in obesity. A WHR calculator can be found here: As with circumference measures, it's important to not overtighten the tape measure. Skinfold Calipers Possibly the most commonly used method of BF% measurement are skinfold calipers, a small plastic device that is used to squeeze fat at different parts of the body. The measurements go into an equation that then estimates BF%. A variety of sites ranging from 3 to 7 (or more) can be used and numerous different equations exist. Typical sites for women's measurement are the back of the arm, chest, iliac crest (above the hip) and thigh. Calipers give a BF% estimate that is usually close to much more high-tech methods at least in the hands of a trained user. Trained is a key word here as using calipers correctly takes a good deal of practice and many do not have it (this can be a big problem at commercial gyms with a high employee turnover). My general experience is that most trainers are hesitant to grab as much fat as they should. Many women have a thigh skinfold that is nearly impossible to measure accurately in many cases. This can lead to drastically underestimated BF% values. While generally accurate when used properly, calipers do have an inherent error of about 2-3% in either direction (note that all methods have some degree of inherent error). This means that they may not be able to pick up smaller changes in BF% to begin with. Fat cells do store water and changes in water retention (that will usually show up as changes in scale weight) can impact on skinfold measurement. The equations can be problematic as well. A host of assumptions are being made about bone density (which differs between women and men, can vary with training, etc.) which can cause them to give some strange values, even if the skinfold measurements are accurate. The equations will occasionally put women well below the 10% lower limit for essential fat, men have been estimated at 1-2% and, due to differences in bone density, black male athletes are occasionally given a negative number. Although it doesn't give an estimate of BF%, some recommend just tracking the skinfold changes. If the thigh skinfold goes down from 25mm to 22mm, fat has been lost. 52 There are many different calipers on the market ranging from very cheap one site click types (which I do not recommend as they are very inaccurate) to $400 clinical calipers used in research. The best one I've found in terms of the price to accuracy ratio is the Slimguide caliper. At $29.99 it's as accurate as the more expensive models and only a little more than the cheaper models which can be incredibly inaccurate. They are indestructible and any reader who wants to be able to track at least some of their own skinfolds could pick up a set and practice on themselves. Underwater Weighing/Bod Pod Underwater weighing is an older method of estimating BF% and often considered the "Gold Standard" for measuring body composition in that it was thought to give the most accurate value (other methods were usually compared to it in research). Underwater weighing requires getting dunked into water in a bathing suit and is essentially based on the rather simple concept that fat floats. First the person is weighed on land and then again underwater and this allows BF% to be estimated. While reasonably accurate, underwater weighing facilities are not commonly found outside of research laboratories and can be expensive. Similar to underwater weighing, a fairly new technology is the Bod Pod; it measures how much air a person displace and represents a similar concept to the above. It is done on land and in clothes but the machine is very expensive and not commonly found. There is also some recent question as to the Bod Pod's overall accuracy and I mention it only for completeness. Due to the cost and difficulty of access, it's unlikely that either underwater weighing or the BodPod will be particularly useful in most situations readers of this book might encounter. Tracking fat loss or muscle gain requires that measurements be made at some reasonable frequency and using either method frequently is somewhat unrealistic. One approach that could be used would be to get an estimation via underwater weighing and compare it to a simpler method such as calipers or the taper measure. If two measurements 4-8 weeks apart can be obtained, it will indicate how close the simpler method is to the supposedly more accurate method both in terms of absolute values and changes. Then only the simpler method would be used going forwards. Bioelectrical Impedance (BIA) I mentioned in the last chapter that some scales at least attempt to estimate BF%, rather than only measuring body weight, and BIA scales represent the primary approach to this. BIA works on the basic concept that fat and LBM have different amounts of water. It works by running an electric current through the body and this can be foot to foot (scales), hand to hand (little handheld devices) or hand to foot (specialized equipment) and using the speed of measurement to estimate body water and BF%. While quick and easy, BIA is extremely sensitive to water balance in the body. Drinking a large glass of water or having a large urination can change the values and some devices have an athlete and non-athlete mode that will give different BF% values for the same person measured at the same time. If hydration status is controlled extremely well, BIA might have some role but this tend to be uncommon outside of research. Change's in body weight and water throughout the menstrual cycle would make this method almost impossible due to shifting amounts of water weight although women with one of the hormonal modifiers would have less issue in this regard. Overall I do not recommend BIA. Infrared Reactance/Bodymetrix/Skulpt Infrared reactance (IR) is an old method where a device was put against the biceps (front of the arm) and measured how quickly a beam bounced back as this was meant to indicate how much muscle and fat was present. Not only is measuring at that single site not very useful as it has no relationship to the rest of the body, the method was originally developed to get a rough estimate on cattle for farmers. It's not very accurate and I do not recommend it. There are two related devices which are the Bodymetrix and Skulpt which claim to measure skinfolds without having to use calipers or pinch the person. Both work by bouncing a beam through the fat and back to determine its thickness and this is used to estimate BF%. Both are kind of cool and high-tech and use multiple measurement sites. The devices are somewhat expensive and I doubt that any but the highest end commercial gyms would have one. I also haven't seen any validation of their accuracy. They are worth watching but at the current time I can't recommend them. Dual-Energy X-Ray Absorbitometry (DEXA) DEXA, relatively speaking, is both one of the newer and higher tech methods of measuring body composition, using some fairly high technology to make a head to toe toe full body scan. In doing so, and 53 in contrast to the other methods I've discussed, DEXA can determine a person's body composition beyond just fat and LBM. Most importantly to readers of this book, and this was the purpose for its original development, DEXA can measure bone mineral density (BMD). Currently it is the only method that is able to do this. While DEXA machines have been expensive and relatively difficult to find in the past, their availability is increasing while the the cost of a measurement is decreasing. While the ability to measure DEXA is reason enough to consider it, it will also estimate BF%, killing two birds with one stone. While it gives an overall measure of BF%, DEXA is unique in that it will provide a BF% for the upper and lower body separately. So it might tell a woman that her upper body is 20% body fat and her lower body is 27% body fat. While this can be useful to track the changes in regional body fat levels (i.e. upper body might drop by 3% but lower by only 1%), for the purposes of this book, only the whole body BF% will be important. I do need to make an important point about DEXA relative to other methods as it pertains to how information later in this chapter and book will be presented. In many cases, I will recommend specific aspects of diet set up or other issues be based on starting BF%. And the values I have traditionally used came from older methods such as calipers or rough visual assessment. This is important as DEXA seems to give systematically different numbers than those older methods, in the realm of 3-6% higher (3). Demonstrating this, a number of top physique competitors have been measured via DEXA in contest shape and invariably the values are at least 3% more than what calipers would put them at (i.e. a woman might be calipered at 10% but DEXA would say she is 13% or higher). I don't honestly know what is responsible for this difference and I don't think it matters in a practical sense. It simply needs to be recognized. When I provide some rough BF% for different goals below and present my Category system I will provide both the older method values along with adjusted DEXA values. While the price is decreasing, DEXA does cost more than other methods and would be generally inappropriate for regular tracking of a diet or muscle gain program. Measurement simply have to be made too frequently to be cost effective or practical under most conditions. Which doesn't mean that DEXA shouldn't be considered, due to its ability to track changes in BMD. As BMD changes far more slowly than body fat, an annual DEXA measure would probably be more than sufficient. When it is done, it could be correlated with a simpler method such as calipers or the tape measure with the simpler method being used for most short-term tracking. Other Tracking Methods While having some initial estimate of BF% is important for many topics in this book, and is important to track on some level, there are other methods of tracking progress on a diet (or when trying to gain muscle). None estimate body composition per se but in conjunction with one of the methods above, they have their use. Tape measure measurements are one of these but were discussed earlier. The Scale Hopefully after reading the last chapter, you understand that short-term fluctuations in scale weight don't represent anything real (in terms of the gain or loss of actual body fat or LBM). I should mention one major exception. For women who are carrying a significant amount of body fat (what I will call a Category 3 dieter, defined below), assuming even the most basic exercise and nutrition program is in place, almost all weight lost will be body fat outside of the initial drop in water weight. In this case, the scale may be all that is necessary to track progress. This is especially true as many of the true BF% methods tend to become inaccurate at the extremes of high or low body fat. For women not in this situation, the scale can still provide useful information but only if the person using it can accept that day-to-day changes are meaningless and they should not make poor lifestyle choices based on those changes. In recent years, there has been a good bit of backlash against the scale, especially for women. Some of this is based on what I've discussed already in terms of it not representing changes in body composition or what have you, that the day-to-day changes make the practice useless. While I have made similar arguments above, the key to weighing is to do it daily and use those measurements to create a 7-day rolling average (this just means that every new day's number will replace the value from 7 days ago). This approach smoothes out the daily fluctuations (one higher day is offset by a lower day) and creates a trend line that is either flat, downwards or upwards representing no change or a real loss or gain of some type of body tissue (the scale still can't say if it's fat or muscle). It also eliminates the inaccuracy of measuring once weekly where a single day's fluctuation can give a very inaccurate picture of what is actually occurring in the body. 54 Recent research has found that daily weighing actually helps people to adopt new health habits, probably by focusing their attention to those goals (4). If a woman weighs daily, it acts as an immediate reminder that she is attempting to change her eating or activity habits and that along helps with adherence. Daily weighing and feedback was also shown to help college aged women avoid the normal freshman year weight gain that tends to occur by giving them better feedback (5). Regular monitoring of body weight has also been shown to help with long-term weight maintenance, a topic I will discuss more in Chapter 31 (6). While short-term fluctuations should still be ignored, a true increase in weight of perhaps 3-5 pounds should indicate that the dieter is backsliding and that more focus needs to be placed on eating and activity patterns (7). Regarding psychological stress, while some women might be impacted, overall the practice of daily weighing has not been shown to cause psychological problems (8). While body weight can be tracked with a spreadsheet, there are apps such as Happy Scale and others that can do it. Even some recent bathroom scales will keep a built-in rolling average of weight. Whether dieting or attempting to gain weight, it is ideal to weigh at the same time of day under the same conditions. This usually means in the morning, preferably after using the bathroom. Weight can be taken naked or clothed although naked will give more consistent values (clothes can weigh 1-2 lbs or 0.5-1 kg or so). Clearly if any given woman finds that daily weighing is causing her psychological stress, it should be abandoned. But overall, so long as the scale is used properly, it is a useful tool with daily weighing being the superior approach overall. I would mention that even this approach to using the scale can be a problem for the normally cycling woman as the weekly changes in body weight will make the averages inaccurate. I will discuss this issue below along with how to take it into account/work around it. The Mirror/Pictures Along with the tape measure and the scale, another non-body composition method that can be useful to track changes is to simply use the mirror or pictures. The reality is that most people (women or men) who want to lose weight/fat or change body composition do it primarily to look better (specifically to look better naked). This is actually critical for physique athletes who are judged on their appearance but even performance athletes who want to alter body composition for their sport are often concerned with their appearance as well. This is especially true for women due to the societal pressures that are present here. The mirror or pictures can be useful in this regard but there are some issues that I want to address. One problem is that the mirror or pictures can lie. Or, perhaps more accurately, the person's brain can lie to them about what they are seeing. This is most pronounced in many eating disorders where an extremely skinny individual will still "see" a fat person in the mirror (males tend to see a skinny body even while heavily muscled). Even outside of that extreme, most people will tend to focus on what they perceive as their specific trouble spots or simply see a different body that is actually there. As I'll discuss somewhat later in the book, it's extremely common for dieters who have reached an extremely low level of body fat to see themselves as fat even when they are at a BF% that is fairly low. There is an additional issue which is that there are often differences in mirrors or lighting that can impact drastically on a person's visual appearance. In general, very bright lights tend to worsen appearance as it washes everything out and slightly darker lights (within limits) improve it. Every gym seems to have one particularly magic mirror that makes people look significantly leaner or more muscular than they actually are. Using the same mirror under the same lighting will avoid this problem. Similar to the mirror, pictures can provide not only a way of tracking but a record of the changes that have occurred. In some ways, they may be better than the mirror in that they are taken less frequently. Over short periods of time, unless someone is extremely lean (here I am talking about certain types of athletes), visual changes just don't occur that quickly. Looking in the mirror daily, someone is highly unlikely to see any visual changes occurring and this can be demoralizing. Even if those changes are occurring, they tend to be too small on a day-to-day basis to be noticeable. In contrast, taking pictures every 4-8 weeks will tend to show more visual changes, especially if they are compared side by side. As with all aspects of tracking changes, it's critical to take pictures under the same conditions. This means that the same clothing (or something very similar should be worn), the same lighting, distance from the camera, etc. should be used. If this is not done, the pictures will not be comparable to one another. It's a dirty little secret in the fitness and diet industry that many of the before and after pictures being used to sell a product may be taken on the same day. Changing the lighting from harsh to lowered, tanning, having the person wear a more flattering outfit, change their posture, go from frowning to smiling makes a staggering difference in no time at all. If you look closely the person may be standing slightly differently, twisted at the waist to narrow it, etc. 55 Clothing Fit Similar to the use of the tape measure and circumference measurements, another approach to tracking progress is to go by the way clothes fit. In some ways this is actually superior to the tape measure in that the issue of measuring the exact same place is eliminated. If a specific piece of clothing is fitting more loosely, body composition is improving and if that same piece of clothing is fitting more tightly, it may be worsening. I say may as those athletes focusing on gaining muscle may still find some clothes fitting more tightly due to the increases in muscle mass. Even after the dieting is done, when long-term maintenance is the goal, clothing be used to catch problems before they get out of hand (this is just another method of monitoring like using the scale). If a specific piece of clothing starts to get tight again, weight regain may be occurring and the individual will know to become more focused on their eating and exercise habits. Choosing From the Different Methods Having looked at a variety of ways of either estimating BF% or tracking progress while attempting to alter body composition (whether losing fat or gaining muscle) I want to make some more specific recommendations about how to integrate them. Once again, every method has its pros and cons, benefits and drawbacks. None are perfect and most are best used to track progress over time rather than being considered as a one-time measurement. Regardless of the specific measurement, if it is changing in the goal direction (i.e. down for fat loss, up for muscle gain), that is what matters most. As well, except for some of the methods I explicitly stated were inaccurate (i.e. BIA, Infrared), the differences in the values given by most methods should all be fairly close to another. If one method gives a BF% of 22% and another a BF% of 25%, that's simply not meaningful. It certainly won't significantly impact on most of what I will present in this book. Perhaps the best way to offset any given method's cons is to use some combination of methods rather than relying exclusively on one (perhaps the lone exception to this is the woman carrying significant fat for whom weight loss will almost always indicate fat loss). That way, any changes that are not picked up by one method will become apparent by another. So consider a woman who we know lost 2% body fat at (roughly 2.5 lbs/1.2 kg for a 130 lb/59 kg woman). The error inherent in calipers might not be able to measure that but a 7-day rolling average of her scale weight certainly would. If she were very lean, she might also notice visual changes in the mirror. I think you get the idea. In terms of specifics, despite their limitations, scale weight (combined with some measure of BF%) will be required in order to do the calculations that I will present in this book. Again, a 7-day average or at least measuring at specific times of the month along with ignoring small daily variations are the key to making this method useful. Used properly, calipers are surprisingly accurate but tend to have problems at the high and low extremes of body fat. Paying attention only to changes in the values can be useful here although this doesn't provide a BF% estimate. As well, without a helper, only a handful of sites can be measured unless someone is a contortionist. BMI is quick and easy and can either be used as a general indicator of health or to estimate BF%. It is not appropriate for athletic or well-trained individuals. Tracking the WHR ratio may be useful for women who are carrying large amounts of fat around the midsection since it indicates the loss of visceral fat which indicates an improvement in health. The tape measure can be surprisingly accurate to estimate BF% as well as tracking changes in inches overall. To one or more of those methods that tracks or semi-tracks some aspect of body composition, the mirror or pictures (or a test piece of clothing) can be added. Basically the combination of a BF% estimate, possibly another method that tracks regional changes in body composition (WHR or tape measure) and at least one non-BF% method of tracking progress will probably give the best combination in terms of providing enough different data points to truly track changes. Some may not even need all of those and I can't cover every possible circumstance to make recommendations. I'd only reiterate, at the risk of beating a dead horse, is that short-term changes in any of these measurement methods tend to mean very little in the big scheme. Focusing on larger changes is the key to making any of them work. Let me finish by making an often under appreciated point which is that changes in body composition or even weight are generally not only slow but rarely happen in a constant or linear fashion. Weight or BF % may drop, then remain unchanged for a week or two before dropping significantly (seemingly overnight), etc. The same tends to occur for those trying to gain weight with weight going up, stalling, going up some more. There are a number of reasons for these plateaus and stalls that I'll discuss in Chapter 25 but women must understand that a lack of change for a week or two means nothing. 56 Body Composition Numbers Having looked at the concept of body composition/BF% in the last chapter and methods of estimating/tracking it above, I want to address the question of what a good or appropriate BF% might be. The answer to that question depends entirely on the situation. The optimal BF% for basic health will differ than that for optimal athletic or appearance goals. While healthy levels BF% have been thrown around, it's now becoming clear that it is possible to be healthy while carrying significant body fat or to be unhealthy while being lean. Much of this comes down to activity levels and active individuals with more fat are often more metabolically healthy than those who are lean but inactive. Which isn't to say that there isn't a general relationship between increasing BF% levels and health, simply that it is not universal. While excess body fat tend to be highly associated with health risks, it's equally possible to carry too little body fat for optimal health. At the lower extremes of BF% (10-12% for women) as seen among some athletes or in anorexia, a woman's physiology is severely negatively impacted in terms of her hormone levels, menstrual cycle function, etc. This will be discussed in great detail in Chapter 12. This just means that there is some happy medium to be had between too little and too much in terms of health status. I would mention that, even in those cases where a woman might not be able to achieve a supposed "healthy" BF%, even a 5-10% fat loss from her current level drastically improves health and fertility. Moving to sports performance, what represents an optimal BF% can vary enormously depending on the sport and I discussed at least some of this in Chapter 4. Some sports, especially those that require the body to move across or against gravity tend to be both lighter with a lower BF% than those that don't. Runners tend to be lighter/leaner than cyclists who are lighter and leaner than swimming while rowers are larger and may carry more fat overall. In some sports, shotput for example, higher levels of body weight and BF% may improve performance through a variety of mechanisms. Athletes in weight class sports tend to be relatively lean as this allows them to carry more muscle at any given bodyweight; many have a Superheavy weight class where weight is unlimited and the athletes often carry significantly more fat. In some of these sports, it's common to train at a higher body weight and BF% before reducing body fat and manipulating water weight at the last minute to make their class. A large number of women's sports such as gymnastics, ballet and figure skating tend to have an aesthetic component emphasizing extreme thinness and BF% tends to be low in those sports. The physique sports of bodybuilding, physique, figure and bikini have requirements that range from the lowest limits of women's BF% to slightly below the average/healthy range. I would note that even in sports where a lower BF% is common or where losing body fat may improve performance, this does not imply that lower is always better. As I'll discuss in detail in Chapter 12 and touch on throughout this book, a woman can develop hormonal, metabolic, physiological and other problems from the dietary and exercise requirements to get super lean and this can have the contradictory effect of harming performance. Outside of those sports, such as the physique sports, where a specific BF% is required, performance athletes should strive to reach an optimal BF% rather than a minimal one. In terms of non-athletic appearance goals, the types of bodies that are often held up as societal ideals are perhaps 18-20% for women or a bit higher, generally with less muscularity. To put this into perspective, women tend to get visible abdominal muscles (the 6-pack) around 15-17% or so and this is leaner than many want to be outside of specific athletic subcultures. While many skinny and/or light women might appear lean, they often have a high BF% which is called being-skinny fat. I've presented some relatively average values for BF% for different situations below and you will see two values. The first represents numbers derived from older BF% methods while the second is their DEXA adjusted equivalents. Situation Older DEXA Lower Limit 10-12% 16-18% Extreme Obesity 50%+ 56-58% Average 21-28% 27-34% Recommended for "health" 18-25% 24-31% In Shape by Mainstream Media Standards ~18-20% ~23-26% Physique Athletes* 10-18% 16-26% Performance athletes Varies Varies *Physique represents bodybuilding, physique, figure and bikini and the degree of required leanness varies between them. 57 Readers who want a rough visual idea of what the above numbers represent can see examples of what women at different BF% look like here (note that the listed percentages are based on the older methods rather than DEXA): In addition to giving some context for the numbers in the above chart, the pictures at that link can provide somewhat of a reality check for the estimated or assumed BF% a person may have. It's not uncommon for online fitness forums to give people body fat estimates based on visual assessment and many are being given bad information in that they are being told that they are 14-16% body fat when they are really 25%. For physique athletes especially, this causes enormous problems as women underestimate their dieting time and get nowhere close to reaching their goals. Diet Categories While estimating BF% is important for many reasons, at least one of those is for readers to determine what dieting category they are in. This is a delineation I have used for many years now and is based around the fact that a woman's physiology is changing to one degree or another based on her current BF%. I won't detail those here but those physiological changes impact on many practical aspects such as how much protein she might need while dieting, her relative risk of menstrual cycle dysfunction, how rapidly she will be able to lose weight or fat, her relative risk of muscle loss and others. When I look at the research relating to women's body fat issues, it will be important to recognize what category the subjects being studied are in. I've presented my categorization system using both older BF% methods and adjusted DEXA values below. Category Older Methods DEXA 1 24% and lower 27-30% and lower 2 25-34% 28-40% 3 35%+ 38-41%+ While I have presented the table with rather discrete cut off points, please realize that this is a continuum. My cutoff points are based on underlying physiology but it's not as if physiology changes completely from one category to the next. A woman at 35% body fat (41% via DEXA) is far closer to a Category 2 female physiologically than a woman at 45%. Similarly, a Category 2 dieter at 25% is essentially the same as a Category 1 dieter. But some sort of delineation is required to make the system work and I'll only suggest that women at the very low end of one Category should consider themselves in the next lower category in terms of the rest of this book. So a woman at 35% body fat (41% by Dexa) should consider herself in Category 2 in terms of her diet set up, etc. Some Category Comments Before looking at some last practical issues, I want to make some brief comments about the Categories above and who might typically be found within them. In general, the Category 1 female tend to be involved in some degree of training. They could be in one of the physique sports, a performance sport or be what I will call a serious trainee (possibly workout out intensely 5-6 times per week but not competing). In many cases, either for performance, competition or appearance, these women will want to lose some amount of body fat As frequently they may want to gain muscle. The physique athlete may wish to bring up weak bodyparts and many performance sports benefit from increased muscle mass. I'd note that there can be a Category 1 female who is not active or training. They frequently are genetically lean although they still want to lose weight. Weight gain is a rare but very occasional goal. Category 2 tends to span the broadest range of possible situations since it matches up with relatively average BF% for women to begin with. Athletes may be in this group as many sports do not require extreme leanness, many recreationally exercising women may also be in this group. It's possible for a physique athlete who let their body fat get away from them in the off-season to be here although that can cause a lot of problems when it's time to diet down for a contest. The serious trainee might fall here if here diet isn't set up correctly such that, despite all of the training, she is not losing body fat or maintaining a lower body fat effectively . It's just as likely for women to be in Category 2 who are sedentary or only minimally active. Fat loss is likely to be the primary goal in this group. 58 In general, Category 3 women are the least likely to be involved in any sort of training program or sports although there are certainly exceptions. As I've mentioned, in some sports such as the throws in track and field or the super-heavyweight classes of some strength/power sports, athletes benefit from being heavier and athletes may carry a significant amount of body fat despite being involved in intensive training. While it's atypical for them to want to lose fat until they are done competing, they frequently desire to increase their muscle mass to improve performance. Overall, it is far more likely for the Category 3 women to be relatively sedentary. In this case, fat/weight loss and/or improving health and fertility is likely to be the exclusive goal. A Female Specific Issue for Tracking Body Composition With the understanding of what body composition/BF% is and how to measure and track it, I want to address a female specific issue that goes unconsidered by most that the normally cycling woman must contend with (women with a hormonal modifier have less of an issue with this). That issue is the often considerable changes that occur throughout the menstrual cycle in water retention. This not only impacts on scale weight but can also impact on other methods of tracking (9). Both calipers and the tape measure measurements can readily be altered during different weeks of the month and, depending on its degree, water retention can make women feel or look puffy in the mirror or in pictures or make clothing fit differently. As I described in Chapter 2, the late follicular and late luteal phase (Weeks 2 and 4 respectively) tend to be the worst in this regards with the early follicular (Week 1) generally showing the lowest body weight and early luteal (Week 3) being somewhere in-between. This causes several problems. The first is that it can exacerbate the normal issues many have with the scale. The woman who is already fixated on the small day-to-day changes can be driven mad by the weekly changes as she will be adhering to her diet and exercise program and almost over night, her weight spikes by several pounds or kilograms. Hopefully readers will avoid at least this issue now that I have pointed out what those types of fluctuations mean but I've even known female trainees who, despite knowing full well the difference between body composition and body weight, still getting affected by these types of weight shifts. Even taking a rolling average as I recommended above doesn't eliminate this because the average value and trend line will be shifting up and down each week. Within any given week, the rolling average will be useful but from week to week it will not be. An added issue is that it makes tracking changes more difficult for the normally cycling woman compared to women with most of the hormonal modifiers or men since comparing different weeks of the cycle to one another won't give any accurate indication of what is happening in response to her diet or exercise program. To better illustrate this, I've shown a hypothetical month of average weekly body weights and how they might change in different weeks of the cycle along with month to month. These numbers are for illustration only and any individual woman may see smaller or larger changes from week to week. Phase Month 1 Month 2 Early Follicular 143 lbs 141 lbs (-2) 140 lbs (-1) Month 3 Late Follicular 147 lbs 145 lbs (-2) 144 lbs (-1) Early Luteal 145 lbs 143 lbs (-2) 142 lbs (-1) Late Luteal/PMS 150 lbs 148 lbs (-2) 147 lbs (-1) You can see that average body weight is changing from week to week during the month with the lowest value occurring in the early follicular phase and the highest in the late luteal phase. I might go so far as to suggest women avoid any measurement during the last week of the cycle due to the large increase that can occur which can be extremely psychologically stressful. In practice that would mean only tracking for three weeks out of the month. Perhaps the bigger point of the table is that comparing any individual week of the cycle to any other individual week is rather pointless due to the water weight shifts that are occurring. Weight goes up from the early to late follicular phase, goes down to a different number in the early luteal before increasing again in the late luteal phase. The week-to-week shifts in hormones and body weight make any comparisons useless. The same holds for other body composition methods. At the same time, you can see that it is possible to compare one week of the month to the same week of the following month. Bodyweight or BF% could be compared between the early follicular phase of Week 1 and the early follicular phase of Week 2 and this will give some indication of what is actually happening over time. The same would hold for the late follicular to late follicular, early luteal to early 59 luteal and late luteal to late luteal. So from Month 1 to Month 2, body weight goes down 2 lbs in each week of the phase. The numbers are all still different from each other but the absolute change is the same. I've shown a similar result from Month 2 to Month 3. The changes might not be this consistent in the sense that every week might not show the same 1 or 2 pound loss and I'd expect the late luteal phase to be the most variable. But overall, comparing only like weeks of the cycle to each other will give a much better indication of what is happening than trying to compare weeks within the same month. This does raise the question of what a woman's "real" weight or BF% both for her own peace of mind as well as within the context of the calculations that will appear late in this book. That is, which week's numbers should a woman use when setting up her diet or protein intake or what have you? In one sense it doesn't matter so long as the same week of the month is used to make any changes. In another sense, since any increase in water weight from week to week isn't "real" in the sense of representing a true change in body composition, measuring in a week where water retention is known to occur makes no sense. As water retention is likely to be at its lowest during the early follicular phase, I'd generally recommend using the average body weight, BF% estimate, from that week. Usually weight will be at its lowest roughly 3-4 days following menstruation and this would give the best indicator of a woman's true weight. There is another reason that using the early follicular phase to set up a diet is important related to when it's best for the normally cycling woman to actually start her diet that I will discuss in a later chapter. How Often Should Measurements be Taken? The final question I want to address regarding tracking body composition is how frequently measurements should be taken, either for general tracking purposes or to know when some aspect of the diet may need to be adjusted. In general, outside of the daily weighing/rolling average I described, most people probably take measurements too frequently. Even with the scale, this is true and people will weigh when they wake up, before and after they go to the bathroom, with and without clothes, in the evening with the goal of getting the lightest weight possible (this is of course the correct value). At any gym you can see dieters weighing before and after the workout to see how much they've lost. Even with other methods, people go a little bit nuts. They'll break out the calipers or tape measure daily or multiple times daily and just drive themselves crazy by doing so. Measurement error and the small day-to-day changes I described make this pointless and even actual body composition changes are far too slow for this to be useful. The only possible exception to the above is the very lean Category 1 female who is nearing the end of her diet where appearance and even skinfolds may be changing very rapidly. This will never happen in the Category 2/3 female and the reason has to do with the total amount of fat being lost relative to how much is left to lose. A 130 lb female at 14% body fat with 10% essential fat only has 4% fat or 5 pounds of fat that she can lose. A half-pound fat loss represents 10% of that value and the measurable or visible changes my be profound. In contrast, a 200 lb female at 40% body fat with 10% essential fat has 60 lbs of fat that she could potentially lose. A 2 pound fat loss is only 3% and simply won't be visible or measurable. But outside of that singular population (lean Category 1 females), obsessive and constant measurement only adds to the inherent stress of dieting (stress is discussed in Chapter 13). At the other extreme, it is possible to measure too infrequently. Whether trying to lose fat or gain muscle, if the diet and training program are set up effectively, changes should be occurring within some reasonable time frame (even if the normally cycling woman has to wait a month to accurately judge it). Waiting endless months before realizing that no progress is being made is wasted time. At some point, some aspect of the diet or exercise program has to be changed if nothing is happening. Somewhere between those two extremes is a happy medium and I've provided some general guidelines based on the dieter's Category (since that will impact how rapidly significant changes occur). These values should be applied to every form of tracking except for daily weighing which is, by definition, done daily. Since the normally cycling woman may have to wait a month to gauge if changes are occurring, she should use 4 week multiples. Women with any other hormonal modifier can measure anywhere within the recommended range. Category Frequency of Measurement 1 Every ~2-4 weeks 2 Every 4-8 weeks 3 Every 8-12 weeks 60 As the Category 1 dieter is generally an athlete or is on a specific time schedule to reach their goals, they will need to measure the most frequently to ensure that they are not falling behind in their progress. The changes here tend to be small (i.e. fat loss may be no more than 0.5 lbs/0.22 kg per week) but still must be tracked. Technically speaking, if a woman is normally cycling she will still be having shifts in water weight that mean she can only realistically compare changes every 4 weeks. If a woman loses her menstrual cycle (assuming it was present to begin with), which is likely to happen if she diets to the lower limits of Category 1, this will cease to matter. With the development of amenorrhea, the normal cyclical changes in hormones will disappear and measurement can be made as often as necessary. As a woman enters Category 2 or 3, the duration between measurements increases for reasons already mentioned. While changes may occur proportionally faster here (a woman who is heavier or carrying more fat can often lose more quickly), it's still important to avoid too frequent assessment of body composition as there may not be a sufficient enough change to maintain motivation. But that duration can't be too long or a complete lack of results might be missed. Once again, daily weighing really eliminates this problem as an average trend downwards in weight over time will indicate if the diet or exercise program is effective. It's simply that measurable changes in BF% or visible changes are unlikely to occur that rapidly. A Basic BF% Calculation Wrapping up the discussion of BF%, I want to present an equation that will serve as the base calculation for many different aspects of this book (other calculations will be shown in later chapters). That calculation is how to determine, based on body weight and some estimate of BF%, how many actual pounds (or kg) of fat or LBM a person is carrying. Since I will be referring to her throughout this book, I will use a sampler dieter who weights 150 pounds at 22% body fat (Category 1). Step 1: Convert BF% into a decimal To do this take the BF% value and divide by 100. 22% / 100 = 0.22 Step 2: Determine Total Pounds of Fat Next multiply bodyweight by the value in 1 to get pounds of fat. 150 pounds * 0.22 = 33 pounds of fat Step 3: Determine LBM Subtract the total pounds of fat from weight to get pounds LBM. 150 pounds total weight - 33 pounds of fat = 117 pounds of LBM. So she weighs 150 pounds with 33 pounds of fat and 117 pounds of LBM. This same calculation can be used in reverse to determine BF% based on their LBM and fat mass. I've shown this in the box below for a woman with 30 lbs of fat and 120 lbs of LBM Step 1: Determine Total Weight Add the total pounds fat to total pounds lBM 120 lbs LBM + 30 lbs fat = 150 lbs total bodyweight. Step 2: Determine BF% Divide total bodyweight by pounds fat and multiply by 100 to get 30 lbs fat / 150 lbs total weight = 0.20 * 100 = 20% body fat It's fairly uncommon to have information on the total pounds of fat or LBM to do the above calculation. Rather, the first equation is generally used to determine the starting point on fat mass and LBM while the second is used to see how changes in either will alter BF% or body composition. 61 62 Chapter 7: Altering Body Composition With an understanding of what body composition represents along with various methods of measuring/tracking it and some representative numbers for body fat percentage (BF%), I want to look at some specifics related to altering body composition. Certainly this won't be the goal of every reader (and I will address general health and fitness later in the book) but, realistically, most women do want to change their body composition either for performance or appearance. I'd note again that there are times when manipulating water and body weight may be relevant for specific athletes or situations but here I will focus on making "real" changes to body composition or BF% in terms of altering the amount of LBM (here referring to muscle mass) and body fat someone is carrying. In this chapter, I will be looking at a variety of different topics. I will start by looking at how alterations in LBM or fat mass can potentially alter BF% before looking at specific situations where someone might want to alter how much of either that they have. I'll also look at the physiological underpinnings of each process. The Best Way to Alter Body Composition Technically, any given reader of this book could have one of two primary goals. The first would be to gain muscle. While the process may be difficult in terms of what is required in terms of training or nutrition, changes here are relatively simple in the sense that gaining muscle will cause muscle to be gained. Losses of body fat may alter how someone looks for any given muscle mass but cannot increase the total amount of muscle mass that someone is carrying. Generally speaking, here the goal is to gain muscle without gaining excessive amounts of fat. In contrast, since it represents the ratio of fat to total bodyweight, gaining or losing LBM or fat can impact on overall BF% although in slightly different ways. If someone gains LBM without gaining fat, BF% will go down as they are now heavier with the same total amount of fat (only the BF% has changed). Even if some fat is gained, so long as more LBM is gained than fat, BF% still goes down. If fat is lost, BF% goes down almost without exception and in this case a woman will have less total pounds/kg of fat. If fat is lost with no muscle loss, BF% will go down. Even if some muscle is lost (and women are less likely to have this occur), so long as more fat than muscle is lost, BF% will go down. Finally, if someone gains LBM while losing fat, BF% will go down. This works identically in reverse in terms of BF% going up and body composition worsening. If LBM is lost with no change in total fat, BF% will go up due to fat making up a larger proportion of total weight. If fat is gained either without a change in LBM or in excess of LBM gains, BF% will also go up. In the case where LBM is lost while fat is gained (as occurs in some diseases, with certain drugs and with some types of birth control), BF% will go up and this may be true even if weight doesn't change. Since it is rare outside of a few specific situations for someone to want to increase their BF%, and since dieting is a far more prevalent goal, I will be focusing primarily on lowering BF% here. And the reason that I am discussing this is due to an oft heard suggestion that, rather than dieting and focusing on fat loss per se, the goal should be to focus on increasing LBM. This idea generally revolves around two primary claims. The first is the fact that muscle burns calories even at rest and that increasing the amount of LBM will raise metabolic rate. Old studies suggested that a single pound of muscle could burn 40-50 calories per day but this is drastically incorrect with the real value being closer to 6 cal/lb or 2.7 cal/kg (1). For perspective, a pound of fat burns roughly 2 cal/lb or 0.9 cal/kg meaning that three pounds of fat will burn the same number of calories as one pound of muscle. The consequence of this is that only the most extreme gains in muscle mass have even the potential to raise energy expenditure meaningfully. Consider that, over the first 6-7 months of training might gain 3-4 pounds of muscle which amounts to 18-24 calories extra per day burned. Two different studies have found that women show perhaps a 30 calorie per day increase in resting metabolic rate when they gain 4.5 pounds (2 kg) of muscle over 12-24 weeks (1a,1b). A gain of 10 lbs of LBM has the potential to burn 60 calories per day and a massive 20 pound gain in muscle might burn an additional 120 calories per day. Every bit adds up but, in the shortterm especially, gains in LBM have no meaningful impact on energy expenditure. It does take energy to synthesize muscle but even there the relatively slow rate of muscle gain in women makes this fairly insignificant. It takes roughly 2,700 calories to synthesize one pound of muscle so a woman gaining one pound of muscle per month might burn ~100 calories extra per day. Any actual increase in energy expenditure from the process of gaining muscle will primarily come from the training involved but longterm increase in metabolic rate from muscle gain are more or less irrelevant under all but the most extreme circumstances. 63 The second idea behind gaining LBM to lower BF% revolves around the mathematical fact that BF% will go down if pure LBM is gained and body weight goes up. As described above, here the total amount of fat a woman is carrying will not change but, since her total weight has increased, the relative percentage of fat will go down. While this is certainly true, as I'll show in the chart below, the effect of gaining LBM pales in comparison to the process of losing fat in terms of its impact on BF%. I will be starting with a sample dieter who weighs 150 lbs with a BF% of 22%. The first calculation in the last chapter can be used to determine that she has 117 lbs of LBM and 33 lbs of fat. All I will be doing below is to manipulate the amounts of LBM, fat or both and recalculating BF% (using the second equation from the previous chapter) for each change. All I've done here is recalculate BF% by dividing the total weight by the total amount of fat. I will be making one simplifying assumption which that 100% fat is being lost or 100% LBM is being gained. While this isn't always the case, it makes the math simpler and the differences in the results don't change that meaningfully without that assumption. First I'll look at moderate changes of either a 5 lb gain in LBM or a 5 lb loss of fat with no other change. I'll also look at what happens if someone gains 5 lbs of LBM while losing 5 lbs of fat (this isn't common and I'm showing it mainly to make a point). I'll also look at the extremes of gaining 20 lbs of LBM (roughly a woman's maximum potential) or losing the same 20 pounds of fat. Finally, just for illustration, I'll show a 10 lb loss of fat. Fat LBM Weight BF% Change (lbs) (lbs) (lbs) Starting Point 33 117 150 22.0 N/A +5 pounds muscle 33 122 155 21.3 -0.7 -5 pounds fat 28 117 145 19.3 -2.7 -5 lbs. fat/+5 lbs. muscle 28 122 150 18.7 -3.3 + 20 lbs muscle 33 137 170 19.4 -2.6 - 20 lbs fat 13 117 130 10.0 -12.0 - 10 lbs fat 23 117 140 16.4 -5.6 The primary message of the above chart is that, in every case, compared to gaining LBM, losing the same amount of fat has a far more pronounced effect on lowering BF%. In the first case, gaining 5 lbs of LBM only lowers BF% by 0.7% while losing the same 5 lbs of fat lowers it by 2.7%, nearly four times as much. Gaining 5 lbs of LBM while losing 5 lbs of fat generates a larger result (3.3% vs. 2.7%, basically the individual results added together) but the major effect is still from losing fat. At the extremes, gaining 20 pounds of LBM only reduces body fat by 2.6%, almost the same as losing only 5 pounds of fat. But losing 20 pounds of fat reduces BF% by 12% from 22% to 10% (the lower limits of what a woman might achieve). Even a 10 pound fat loss causes over twice the reduction in BF% (- 5.6% vs. -2.6%) than gaining 20 pounds of muscle. Half as much fat loss as LBM gain has twice the impact on BF%. Hopefully the above shows that the impact on BF% by gaining muscle isn't even close to that of actually losing fat. The fact that even smaller amounts of fat loss have a greater effect than enormous gains in LBM shows that; when the numbers are equal, fat loss may have four times the overall effect. Even if this weren't the case, there is an additional issue that must be considered which is the time frames involved. Because in almost all situations, the fact is that gaining muscle is a grindingly slow process, even moreso for women than men. In contrast, fat loss can occur relatively quickly. So while gaining even 5 pounds of muscle might take 6 months of effort (and might come with a small amounts of fat gain), that same 5 pound fat loss might take only 5-10 weeks. As the numbers get larger, so do the differences. A 10 pound gain in muscle might take a woman a year or more. The same 10 pounds of fat loss might take 1020 weeks. Gaining the extreme of 20 lbs of muscle is a career goal for most women and might take 3+ years if it is achieved at all. That same 20 lb fat loss might take 6 months for a lean female and less than that for someone in my Category 2 or 3. Even if it took a full year to lose that 20 lbs of fat, it's still one third of the time it would take to gain the same amount of muscle with far greater impact on BF%. I'll finish this section by noting that everything discussed above works the same in the opposite direction. That is, losing LBM tends to have a relatively small overall impact on increasing BF% while fat gain always has the much more profound effect. Without putting it in chart form, if the sample dieter loses 5 lbs of LBM with no change in her total fat her BF% only increases from 22% to 22.7%. If instead she gained 5 pounds of fat with no change in LBM, her BF% would increase from 22% to 24.5%. Regardless, increasing LBM or preventing it's decrease is still important for other reasons, discussed next. 64 Gaining and Losing LBM Irrespective of the fact that gains and losses in LBM have minimal effects on either metabolic rate or BF%, there are still many reasons for women to be concerned about how much LBM they are carrying either in terms of increasing it or preventing its decrease. For general fitness and health, carrying some amount of muscle mass along with some degree of increased muscular strength tends to improve health, strength and overall physical function (i.e. the ability to carry heavier objects). Even small increases in muscle size, especially in women's proportionally weaker upper bodies, can have a huge impact on function and the importance of increasing LBM and strength has finally been recognized by most authorities. While this is important for all women, it becomes especially critical with aging as there is often a loss of LBM that harms both health and function. I would mention that much of this age related loss is related primarily to changing activity levels. Studies of female master's athlete show a significant retention of LBM compared to their sedentary peers. Women have the additional factor of menopause where LBM loss accelerates significantly, especially if they decide not to go on HRT. This often sets up a vicious cycle where a loss of muscle leads to a loss of function/decrease in activity which causes more muscle to be lost. For the physique athlete, muscular size is part and parcel of their competitive requirements although the amount of muscle required depends on sport (decreasing from bodybuilding to physique to figure to bikini). Symmetry and balance among muscle groups is key and frequently these athletes only need to gain muscle in specific areas. For performance sports, gaining muscle can improve strength and power production, improving performance in many sports. In others, too much muscle mass can be detrimental. A weight class athlete may not be able to realistically make their weight class if they carry muscle mass beyond a certain point although they may simply move into the next higher weight class. Many endurance sports have performance harmed if excessive muscle is carried, especially in muscle groups not relevant to the sport (i.e. the upper body for runners). I would mention that, due to their generally reduced ability to gain muscle mass, female athletes don't have nearly the concern here as male athletes (who often get a bit overzealous in the weight room in the off-season) but there may be situations (discussed below) where muscle loss is actively sought. LBM loss is, in general, not good although there are occasional situations where it may be actively desired. Outside of the normal aging process and menopause, probably the most common situation a woman will encounter where LBM loss is a risk is during a diet. Again I'm focusing only on actual muscle mass loss as the early water and glycogen loss is technically LBM. The relative risk of LBM loss depends on a few factors. A primary one is body fat percentage with leaner dieters being at greater overall risk. Category 3 dieters may lose zero actual LBM while Category 2 dieters are at a slightly higher risk and Category 1 female dieting to the extremes the most risk. Other factors play a role here, with the exercise program and diet playing a major role in whether or not LBM is lost. I'd point out again that women are much less likely to lose LBM than men and studies I will describe near the end of the book found that female physique competitors who were performing resistance training and eating sufficient protein lost essentially zero LBM while reaching the lower limits of BF%. There are a number of reasons to limit LBM losses while dieting. One is that even if BF% is decreasing, the loss of LBM can lead to less than hoped for visual improvements. Dieters end up being slightly smaller versions of their previous self. Maintaining LBM or even increasing it slightly has a profound impact here providing the much sought after "toned" appearance that represents sufficient muscle size coupled with a reduction in BF%. Weight training, in addition to both increasing muscle mass or preventing its loss, can indirectly improve fat loss as well. This is discussed in Chapter 14. A second reason is that avoiding LBM loss while dieting helps to limit the normal drop in metabolic rate (discussed later in the book) that can occur (2). Additionally, it's recently been found that LBM sends a signal to the brain that can increase hunger; losing LBM while dieting results in more hunger than would otherwise occur. The main risk here is for the Category 1 dieter. So long as they are active and eating sufficient protein, there is little to no problem but I described a subgroup of lean Category 1 females who are inactive who are often attempting to lose weight. As they tend to diet without exercise (especially resistance training) or sufficient protein, they often lose LBM. This leaves them with more rebound hunger than they would otherwise experience which puts them at the risk for rebounding and ending up at a higher BF% than they started at (3). There are rare occasions where LBM loss may be accepted or even desired. I mentioned one above which is the endurance athlete who may be carrying excess muscle, especially in non-relevant muscle groups, that is harming their performance. Losing that muscle reduces body weight and this often improves their performance. Outside of that there are two primary situations where actively losing LBM or at least 65 avoiding further increases may be appropriate. The first is in PCOS or Category 3 women (and remember that the two are often linked) who has gained a large amount of undesired LBM as they gained weight. Losing that extra LBM during a diet may be an appropriate goal either from an aesthetic point of view or to allow bodyweight to be reduced to the most significant degree. Similarly those women with PCOS/subclinical hyperandrogenism who are not interested in sports performance (and who tend to put on LBM at a slightly faster rate than other women) may want to explicitly avoid the types of training that tend to increase LBM to the greatest degree. There are still benefits to weight training such as improved health but the program will have to be modified in this situation. But outside of those very few exceptions, the majority of women should put at least some effort into either increasing their LBM or at least preventing its loss and this is true whether fat loss or simply general health is the goal. I'd note that the type of training that best accomplishes this turns out to be the type of training that either increases or prevents/slows the loss of bone mineral density. The Physiology of Gaining and Losing Muscle Without getting into unnecessary complexity, I want to look briefly at the process of either increasing or maintaining muscle (the overall processes tend to be more or less identical). Muscle fibers can technically increase in number (called hyperplasia) or size (called hypertrophy). The latter represents the majority of the increase or decrease in muscle and I will focus only on it. In the simplest sense, subjecting skeletal muscle to certain types of stress causes it to adapt. In the case of muscle gain that generally means weight training (discussed in Chapter 4) where it is forced to work against a heavy weight for relatively short periods of time. This stimulates growth and now amino acids (from dietary protein) must be provided in sufficient quantities as the building blocks of new muscle tissue. Sufficient calories are also required and the best muscle gains will always occur when proper resistance training is combined with sufficient dietary protein and a slight calorie surplus (4). Since their rate of growth is relatively slow, women never need a large calorie surplus and I will give specific recommendations in a later chapter. The amount of muscle someone can gain is based on a number of factors. There are genetic factors but arguably the most important is the level of reproductive hormones. Testosterone is the key player here although recall that estrogen plays an important role in muscular remodeling as well. Progesterone has a negative effect on muscle growth through various mechanisms. Due to their lower testosterone levels, women start out with less muscle mass, gain it more slowly, and have less potential in terms of the total amount of muscle that they might gain. In response to training, at least in beginners, women do gain a similar percentage of muscle as men, the amount is simply smaller in an absolute sense due to starting out with less. Women with elevated testosterone levels due to PCOS/subclinical hyperandrogenism do tend to have more muscle, gain it relatively more quickly and have a higher potential gain that they might achieve. Quite in fact, differences in testosterone levels between women directly predict the amount of muscle and strength that they gain from training (4a). The process of losing muscle, called atrophy is effectively the opposite of gaining it. Here the muscle fibers are broken down to provide amino acids and energy to the body. While this can happen in a number of disease states, the most common situation for readers of this book will be dieting. Hormones such as cortisol increase to mobilize fuel and one of its impacts is to break down muscle tissue to provide energy to the body. Excessive aerobic exercise can cause this as well. This breakdown of muscle is especially prevalent if the diet has insufficient protein and no resistance training is present to "tell" the body to maintain the muscle that it has. For the record, while it was long felt that extreme caloric deficits caused more muscle loss, this is generally untrue if the diet is set up appropriately. Most very low calorie diets did not use exercise and contained far too little protein and that, rather than the calorie intake per se, was the problem. There are other situations that muscle loss can occur under as well. The loss of estrogen signaling after menopause is one (although resistance training and sufficient protein can at least off set this) and I've mentioned several times that certain forms of birth control can cause this. Why Gain or Lose Fat? In addition to the fact that changes in the amount of body fat have a more pronounced effect on changes in body composition and BF% than changes in LBM, the reality is that a large percentage of women are either currently dieting or have dieted at some point in their life. And while their goal may have only been to lose weight, hopefully it's now clear that the goal should actually be losing fat. As I also expect the majority of the readers of this book to be interested in fat loss, for whatever purpose, I will spend proportionally more time on it. 66 First let me look at the issue of gaining body fat. Certainly body fat gain is, for most people, not an explicit goal but rather a consequence of their lifestyle and environment. The modern environment has been termed obesogenic, meaning that we are surrounded by readily available, high-calorie palatable foods that are easy to over eat with a lifestyle that requires very little activity for most people. That said, there are a small handful of situations where someone might have fat gain as a goal. Fat regain would be a more accurate description as, outside of some specific disease states beyond the scope of this book, it will generally only be a goal after someone has lost a significant amount of fat to begin with. This would include the Category 1 female who had dieted to or near the lower limits of female BF% (~10-12% by older methods). Whether it was done for physique competition or performance, she will need to regain body fat to some level to normalize her hormonal status, physiology, menstrual cycle function, etc. There might also be the occasional performance athlete who had reduced their weight or fat to the point that it harmed performance or health for whom regaining some amount weight and fat might actually improve both. Finally are women with eating disorders that cause them to achieve extremely unhealthy body weight and body fat levels and for whom regaining both weight and fat is part of their recovery. This must be medically supervised and I include it only for completeness. In terms of sports performance, there are a handful of situations where carrying more body fat can be useful. One is in the types of strength/power sports such as throwing or Olympic lifting (in the unlimited class) where overall body size plays a role in performance simply due to the increased body mass. Since there is a limit to how much LBM can be gained, typically gaining fat is the only real way to increase overall size and weight at some point. Gaining fat not only increases their leverages but allows these athletes to eat enough to support heavy training. Even athletes who have to make a weight class often train at a higher body weight and body fat level and then reduce fat and manipulate water before a competition. They may diet down slightly for a competition before deliberately raising weight and body fat to optimize their training before the next competition. Moving to the topic of fat loss, there are numerous reasons why a woman might want to lose body fat. Generally, carrying excess body fat tends to be unhealthy although, as I mentioned in Chapter 6s, this relationship is not as cut and dry as many think. Body fat distribution plays a role and I mentioned that carrying more fat in the lower body is healthier than carrying more in the upper body. It is also possible to carry more fat and be metabolically healthy or carry less and be metabolically unhealthy. Activity levels are a major factor here (i.e. being fatter but active may be healthier than being leaner but inactive) but, in general, carrying excess body fat is associated with a variety of health risks. Both insulin resistance and fertility problems are common with obesity (and PCOS which is often associated with it) and even losing small amounts of fat ends to drastically improve these health parameters. Arguably the most common reason that women will want to lose fat is for appearance reasons. Many people will pay lip service to wanting to improve their health (and this is often true when the issue of fertility is a concern) but, right or wrong, the reality is that most want to look better. In some cases, and this is especially true for women who are fixated on body weight rather than body composition, the goal is to reach some (usually) arbitrary goal weight or lose some (usually) arbitrary amount of weight. If it does nothing else, hopefully this book will demonstrate that body composition is the far more important end goal than body weight per se and that attempting to lose some number of pounds or reach some specific goal weight is not the best way to approach things. For athletes, body fat reduction may be a goal for performance reasons, appearance reasons, or both. As I mentioned in Chapter 6, reducing body fat can improve performance at least up to a point. Beyond that point, between the hormonal and physiological changes that occur, along with the amount of food restriction and exercise required, performance is often harmed (along with other changes I will discuss in Chapter 12 when I talk about menstrual cycle dysfunction). As I've mentioned, the physique sports have appearance as a primary goal and, whether healthy or not, reducing BF% is part and parcel of competition. There are also some performance sports with an aesthetic component, where the athletes are expected to look a certain way and may be penalized if they do not meet the appropriate requirements. Even for those performance sports without an explicit appearance component, female athletes are often still under pressure from an appearance point of view due to wearing skin-tight outfits in competition or simply those common social pressures that exist for women (but rarely men). Gaining and Losing Fat: Introduction In Chapter 5 I mentioned that fat cells are primarily made up of stored triglyceride (TG, three fatty acid chains connected to a glycerol molecule) with a bit of water and the cellular machinery involved in fat 67 cell metabolism and hormone production. Since the glycerol part of the TG isn't that relevant to the discussion, going forwards I will only be focusing on the metabolism of the individual fatty acids (FAs). I am also only going to describe the system generally here as gender differences are discussed in detail in Chapter 10. At any given time, fat cells are both storing fatty acids within the fat cell (called lipogenesis) or releasing them (called lipolysis). It's a fairly continuous process and while this might seem wasteful, it gives the body the ability to rapidly adjust to changes in energy needs. That is, if fatty acids are needed for energy, they are already available for use rather than having to be mobilized first. In terms of fat gain, it is the balance of these two processes that determines what happens to a fat cell. If more fat is being stored than released (and burned for energy), fat will be gained. If more fat is being released/burned for energy than is being stored, fat will be lost. There are actually two ways that fat can be gained, through an increase in fat cell size or an increase in fat cell number (this runs contrary to old ideas that said fat cell number was set at birth and never changed). When fat is gained, fat cells can increase either in size or number. In contrast, when fat is lost, fat cells will only decrease in size. This is important as, when Category 2 women and men were both overfed, upper body fat cells increased in size while lower body fat cells increased in number (5). Short of surgery, the new fat cells can't ever be lost again. Both fat gain and fat loss are more generally controlled by what is called energy balance, the difference between calorie intake (from food) and calorie expenditure (from a number of different factors). It is more complicated than this with the balance of nutrients (protein, carbohydrates, fat and alcohol) all having slightly different effects. Since protein is rarely if ever stored as fat and alcohol has only indirect effects on fat gain, I will be focusing almost exclusively on carbohydrates and fat metabolism below. With that basic background, I want to look in some detail at the physiology of both gaining and losing fat in the same way as I looked at the physiology of gaining and losing muscle. Given that it is relatively more complicated, and given the general focus on fat loss for women (along with some gender specific issues), I will spend much more time on fat loss than fat gain. Gaining Fat As I mentioned above, in the most general sense, fat gain occurs when calorie intake exceeds calorie expenditure. As more calories are available than are required, any that can't be used for immediate energy are stored for later use. This can actually include increased carbohydrate storage within muscle or liver as glycogen or fat within the muscle as Intra-Muscular Triglycerides (IMTG) but at least some will go into fat cells. And in this regard, there are a number of gross misconceptions and ideas about what causes fat gain. Since fat cells are made up of the exact same chemical structure found in the fat in food, many assume that eating fat will lead to fat gain. In recent years, the idea that carbohydrates are the cause of fat gain has become popular (usually based on the increase in the hormone insulin). And it turns out that there is truth to both of ideas in that both carbohydrate and fat can contribute to fat gain; they just happen to do it through different mechanisms (6). Dietary fat contributes to fat storage directly and any that isn't burned for energy or stored as IMTG will be stored in fat cells (under extreme circumstances fat can be stored in places such as the pancreas or liver). In contrast, dietary carbohydrates cause fat gain indirectly by affecting how much fat the body is burning overall. When more carbs are eaten, the body burns more carbs for energy which means it burns less fat. Whatever dietary fat is being eaten is likely to get stored. Similarly, if less carbohydrates are eaten, the body burns more fat for fuel which means less fat may be available for storage. An often heard claim is that excess dietary carbohydrate is converted to fat and stored (a process called De Novo Lipogenesis or DNL) but this is a process that happens very rarely in humans. Generally it takes massive intakes of carbohydrates (700-900 grams per day) for multiple days to stimulate DNL. Under most circumstances, DNL contributes minimally to fat gain (7). I want to make it clear that this in no way means that a low-carbohydrate diet automatically prevents fat gain. Since they are typically associated with higher total fat intakes, the overall effect may be no different than a higher carbohydrate/lower-fat diet. I would mention only briefly that protein is essentially never stored as fat and alcohol acts very much like carbohydrate in terms of its effect on fat storage. When alcohol is consumed, it must be burned off for energy so anything else that is consumed is more likely to be stored. If alcohol is consumed with fatty foods, this can readily contribute to fat gain. All of that said, an actual net gain in fat can only occur if calorie/nutrient intake exceeds requirements. While the full details of how fat is stored is incredibly complex, I only want to focus on two key enzymes (both mentioned in Chapter 2) that are involved in storing dietary fat. After it has been eaten, dietary fat goes through complex metabolism in the stomach whereby it is repackaged into something 68 called a chylomicron. These go into the lymphatic system and eventually move past the fat cells. Here an enzyme called Lipoprotein Lipase (LPL, also found in muscle) breaks fatty acids off of the chylomicron for storage within the fat cell. While LPL was thought for decades to be the only key player in this process, it is now known that Acylation Stimulating Protein (ASP), which I mentioned in Chapter 2 is a far more potent stimulator of fat storage, especially in women. For years, it was thought that storage of ingested dietary fat was the only way that fat cells could store fat but there is actually another which is being called the direct pathway (8). Here, fatty acids that have been released from fat cells and which are floating through the bloodstream can be stored directly in fat cells and this occurs without the activity of LPL. More importantly, fatty acids released from fat cells in one place in the body can be stored in fat cells in a different part. I'll talk about the implications of this for women in Chapter 10. There are a tremendous number of factor which determines how likely or not someone is to store (or lose) fat although many of them are outside of our control. Both the type and amount of exercise play a role here and there are clear genetic differences with some people being more prone to store fat than others. Hormones are key players with insulin playing a primary role in both stimulating fat storage and inhibiting fat mobilization although I'd mention that eating pure dietary fat will stimulate fat storage (via ASP) without the need for insulin to increase. Other hormones tend to play a more modulating role here. Clearly both estrogen and progesterone have an impact on both how easily and where fat is stored. Cortisol, which I have not discussed but will detail in Chapter 13, has an odd impact on fat metabolism in that small pulses of cortisol increase fat mobilization but chronically elevated levels can increase fat storage (especially if insulin levels are high). The hormonal modifiers can also play a role. I mentioned that birth control can play a role although it seems to be small overall along with the effect depending heavily on the specific type of BC in question. PCOS/hyperandrogenism can contribute to fat gain along with impacting on where that fat is stored. The general aging process along with a shift in women's hormones as she ages also plays a role with menopause causing both weight and fat gain along with a shift in fat patterning unless HRT is begun. While all of these play clear mediating roles in whether or not fat gain occurs, the primary factor is still the balance between calorie intake and expenditure which are about the only two factors that most people have any sort of control over. Losing Fat: Introduction As I realistically expect most readers of this book to be primarily interested in fat loss, I will be spending proportionally more time on the topic. Once again, here I will discuss primarily general concepts as female specific issues/gender differences are discussed in detail in Chapter 10. Just as fat gain occurs when more fat is being stored than mobilized in a fat cell, fat loss occurs when more fat is mobilized and then burned for energy that is being stored. And, just as with fat gain, the primary cause of this will be when there is a long-term deficit between calorie expenditure and calorie intake. In that situation, the body must find an alternative source of energy (or adapt metabolically, discussed in detail in Chapter 9) to make up for the deficit. That means using fuels already stored within the body and that includes stored body fat. If that deficit is maintained for a sufficient time period, the net effect will be that fat is mobilized from fat cells and "lost". I would note again that while fat gain can occur through increases in fat cell size or number, fat loss always occurs through a decrease in fat cell size. Outside of surgery (i.e. liposuction, cryolipolysis), the removal of fat cells (a process called apoptosis by which they actually die) generally only occurs under very extreme conditions. While the above paragraph more or less sums up fat loss, I want to delve a bit further into the physiology involved as this will be important when looking at some of the gender differences that occur in fat distribution and fat loss itself. To do so I will ignore many of the more minor or less important aspects of the fat loss process and focus only on three: mobilization, transport and oxidation (the actual burning of fat). Let me now look at each in sequence. Mobilization/Lipolysis While all of the steps in fat loss are important, arguably the single most critical is that of the actual mobilization and release of fatty acids (called lipolysis) from within the fat cell. I say most critical as, short of surgery, if the fat is never mobilized from the fat cell, it can never be lost. Lipolysis describes the process by which the stored TG is broken down into three FAs and a molecule of glycerol by a variety of enzymes. Those fatty acids and glycerol are released from the fat cell into the circulation around the fat cells and how easily or not this occurs depends on a number of different factors. 69 One is the actual size of the fat cell. Larger fat cells release FAs more easily than smaller fat cells and this is part of why fat loss becomes more difficult as people get leaner. This actually means that, contrary to belief, people carrying more body fat have an easier time using fat for fuel during a diet (this is part of why they lose less muscle as well). Their fat cells are typically more full and there may be more of them in total and this adds up to an easier time mobilizing fat for energy. One odd factor is the actual type of fat stored in a given fat cells. Here I am talking about saturated and unsaturated fats (this is just a description of their chemical structure and I will discuss them more in Chapter 18). Saturated fats are more difficult to mobilize than unsaturated and it turns out that lower body fat is more likely to store saturated fats. This often make it hard to the touch compared to fat in other parts of the body which is softer or squishy. The primary enzyme in fat cells that is responsible for lipolysis is Hormone Sensitive Lipase (HSL), which breaks fatty acids off of its glycerol backbone for release into the bloodstream. There are other enzymes of importance but HSL ultimately controls whether or not fatty acids are mobilized. When HSL activity is high, fatty acid mobilization is high and when HSL activity is low, fatty acid mobilization is low. While there is more complexity to the system, HSL activity is ultimately controlled by hormone levels which can either increase or decrease the amount of HSL present or increase or decrease its activity. There are three primary hormones and multiple secondary hormones that play the major role in either stimulating or inhibiting fat mobilization (9). The secondary hormones here are growth hormone (GH), cortisol and the reproductive hormones, testosterone, estrogen and progesterone. GH directly stimulates lipolysis but the effect is delayed, taking about 2 hours after a surge in levels to stimulate fat mobilization. Women do have higher GH levels than men both at rest and in response to exercise. I mentioned cortisol above and will repeat that it's effects depend on whether or not it is released in short pulses (which mobilizes fat) or is chronically elevated (which may cause fat storage). I detailed the reproductive hormones in Chapter 2 and will only say here that their effect tends to be somewhat indirect, modifying the level of enzymes or receptors that impact on fat metabolism. The three primary hormones, in that they have direct effects, involved in regulating lipolysis are insulin, the catecholamines and a relatively new hormone called Atrial Natriuretic Peptide (ANP). The first two have been known for decades to have an impact but ANP was not only discovered relatively recently (in 2000) but stimulates lipolysis through a completely different biochemical pathway than other hormones utilize (10). ANP will come up later in the book not only because there are gender differences in how levels respond to exercise but because it has potential to help women with lower body fat loss. Insulin is arguably the single most important hormone in terms of fat mobilization in that even small increases it can inhibit lipolysis by up to 90%. Even the amount of insulin present in the bloodstream after an overnight fast inhibits lipolysis by 50% from its maximal levels. This prevents an excessive release of fatty acids, as might be seen in Type I diabetes, that can be damaging to the body. In addition to its effects on inhibiting lipolysis, increase in insulin also stimulates fat storage so its effect is two-fold in terms of the fat cell (note that insulin is a general storage hormone also increasing carbohydrate and protein storage elsewhere in the body). Insulin has this effect by binding to the insulin receptor which both activates LPL and inhibits HSL. Prior to the discovery of ANP, the catecholamines, adrenaline and noradrenaline (or epinephrine and norepinephrine) the fight or flight hormones, were the only hormones thought to directly affect lipolysis. As I mentioned in Chapter 2, adrenaline is released from the adrenal gland and works throughout the body while noradrenaline is released from nerve terminals and only works locally where it is released. Levels of both increase in response to various types of stress although exercise is the one that we tend to have the most control over. The type, amount and intensity of exercise determines how much of each hormone is released with adrenaline primarily being released at lower intensities and noradrenaline not being released in significant amounts until much higher exercise intensities are reached. The catecholamines have their own specific receptors called adrenoceptors (or adrenoreceptors) which are found almost everywhere in the body including the heart, skeletal muscle, liver, fat cells, etc. Here it gets slightly complicated as there are two primary types of adrenoceptor which are called alpha- and beta-adrenoceptors which have roughly opposite effects in the body. There are also multiple subtypes of each such as alpha-1, alpha-2, beta-1, beta-2 and beta-3 which are found in different levels in different parts of the body. The simplest way to think of beta-receptors is as accelerators of some physiological process. When catecholamines bind, this causes an increase in heart rate, blood pressure, or fat mobilization. In terms of fat cell metabolism, only the beta-2 receptor is relevant and when it is activated, HSL activity will increase, mobilizing fat. In contrast, think of the alpha-adrenoceptor as a brake. When activated, it slows some process, lowering heart rate, blood pressure, calorie burning or decreasing lipolysis. Only the alpha-2 70 receptor is relevant to fat cells and when it is activated, it will decrease the activity of HSL, decreasing lipolysis. While this may seem needlessly complex, I am discussing it as fat cells in different parts of the body can have different ratios of beta-2 to alpha-2 receptor and this has an enormous impact on the relative ease or difficulty of mobilizing fat. Fat cells with more alpha-2 receptors than beta-2 are more difficult to mobilize than fat cells with the reverse distribution. This actually means that the catecholamines can technically stimulate or inhibit lipolysis depending on the receptor ratio and I will discuss this more below. In terms of their overall effect on lipolysis, insulin and the catecholamines basically oppose one another. Insulin inhibits HSL activity, inhibiting lipolysis, while the catecholamines (broadly) stimulate HSL activity and stimulate lipolysis. Generally speaking, when insulin is high, as after a meal, the catecholamine levels will be low with nutrient storage being stimulated. In contrast, when the catecholamines are high, as between meals or during exercise, insulin is low with fat mobilization being stimulated. In the rare situations where both insulin and the catecholamines are elevated, insulin's effects will dominate and fat cell lipolysis will be decreased. Finally there is ANP which, bizarrely, is released from the heart and which is primarily involved in regulating blood pressure and water balance but also stimulates lipolysis. As stated it works through a different overall pathway than insulin and the catecholamines although the end result is still to increase HSL activity. A number of factors regulate ANP with exercise being the one that we can most control. Of some interest ANP can maintain lipolysis even in the face of high insulin and there is some indication that its effects may depend on the specific region of body fat (i.e. upper vs. lower body). Transport While the mobilization of fatty acids is the critical step for ultimately losing fat, it isn't sufficient to guarantee fat loss. Quite in fact, a large number of fatty acids that are released turn right back around to be stored within the fat cell a gain, a process called re-esterification. Fat cells can't burn fat for energy which means that any mobilized fatty acids must be carried somewhere in the body that can. The critical step here is the rate of blood flow past the fat cell. If it is high, mobilized fatty acids will be carried away from the fat cell. If it low, they will not and are more likely to be stored again. As it turns out, the same basic factors that control lipolysis tend to control blood flow with insulin, the catecholamines and ANP playing the major role. Insulin has the odd effect of increasing blood flow to fat cells but its overall effects are still to inhibit lipolysis. The catecholamines work identically with beta-receptor activation increasing blood flow and alpha-receptor activation inhibiting it and the ratio of receptors determining the overall effect. ANP improves blood flow to fat cells. Finally, fat mobilization and blood flow tend to be related: fat cells that are resistant to mobilization have poor blood flow and vice versa (11). Once a fatty acid has been mobilized and transported away from a fat cell, it has a number of potential fates. One is that it can be stored in a different fat cell by the direct pathway I mentioned above. Fat may also be stored within skeletal muscle as IMTG for later use or, in the case of severe obesity, stored in the pancreas or liver. While that storage still technically reduces the size of the fat cell that the fatty acid came from, the fat is not truly lost from the body until the final step in the process has taken place. Burning/Oxidation The final step in fat loss is the actual "burning" or oxidation of the fatty acid. This can occur in many tissues of the body including the heart and liver although I will primarily focus on skeletal muscle. Ignoring complex details, the fatty acid is transported into the muscle cell, entering the mitochondria (the powerhouse of the cell) where it reacts with oxygen (hence oxidation) and is broken down to provide energy, water and carbon dioxide (which are then excreted from the body). At this point, the mobilized fatty

[illegible]

outset that human food intake is impacted by a staggering number of factors many of which are purely environmental (at most they interact with the underlying biology). Since people's environments don't tend to change enormously without conscious effort, I will only only focus on the biological factors that impact on EI and what changes occur there. Broadly speaking, there are two major systems that are relevant to food intake and researchers refer to these as the homeostatic and hedonic systems respectively (9). The homeostatic system is meant to regulate food intake based on the actual nutrient requirements of the body (i.e. if blood glucose falls, people get hungry for carbohydrates to raise it). This system is also affected by the body's overall energy stores. Body fat is a key player in this although LBM/muscle is turning out to have a role as well. These two factors work to control hunger which is purely a short-term response (someone will get hungry, get eat, get full and stop eating) and appetite (the overall desire for food). If the distinction is unclear consider older individuals who will get hungry (eating a meal and stopping) but eat little total food overall. The hedonic system is related to the fact that eating is pleasurable and biologically rewarding. Highly palatable, typically high-sugar/high-fat foods, tends to cause the largest response here but eating of any sort tends to have an effect here. This system is involved in any number of rewarding activities (such as sex) and it's interesting to note that the pathways seem to be related to those that control drug addiction (10). This is mediated by dopamine (DA) in the brain along with the opioid hormones both of which impact on how rewarding or enjoyed food (or other activities) are. For genetic reasons, many appear to start with a more responsive or sensitive hedonic system (i.e. they enjoy eating certain foods more than others). This is often found in obese individuals and is part of why they are more likely to gain weight in the modern environment (11). The naturally lean individual simply may not enjoy these foods as much. While the two systems above are somewhat distinct, they are also inter-related in terms of the factors that are regulating them. Here the system gets very complex with an absolutely staggering number of different things sending a signal to the brain. The levels of glucose, amino acids and fatty acids in the bloodstream play a role here and even the physical stretching of the stomach after a meal sends a signal to the brain to signal fullness. There are also an enormous number of hormonal signals, many sent from the stomach itself that play a role here. Cholecystokinin (CCK), peptide YY, glucagon-like peptide 1 (GLP-1) and others are released in response to food intake with the proportion and amounts of each hormone being related to the size of the meal and the amounts and type of nutrients consumed (12). Protein, fat and fiber have the biggest impact on CCK, for example with protein being the most filling nutrient of all. Of all of these, perhaps the most important hormone released from the stomach is ghrelin. When ghrelin goes up, it stimulates hunger (it is the only hormone to do so) and ghrelin levels actually increase before habitual meal times. Ghrelin has other roles related to the control energy expenditure as that I will discuss below. The hormone leptin, released primarily from fat cells plays an enormous role here although it tends to be somewhat more indirect, changing how the brain responds to the other hormones. But all of these hormones send an integrated signal to the brain (specifically the hypothalamus) that ends up influencing hunger and appetite. Many, if not most of these hormones, also impact on the hedonic system usually by altering the levels of dopamine (DA) in the brain to one degree or another. Adaptation to Diet and Fat Loss When someone reduces food intake or in response to dieting, there is a fairly stereotyped response in the above systems. Due to less total food being eaten, less of the fullness hormones such as CCK, PYY, etc. are released. Ghrelin levels are also increased overall and don't decrease as much after a meal. Leptin, released primarily from fat cells, decreases which means that the brain responds even less to those hormone signals. The increase in leptin and decrease in ghrelin, along with the other hormonal changes, has a potent overall effect which is to increase appetite and hunger along with decreasing how full people 80 get after a meal (at the extremes, dieters may be hungry shortly after finishing the previous meal). Those same hormonal changes also impact on the hedonic system with DA levels dropping (leptin plays a major role here) along with other changes. This makes tasty foods taste even better (think of how much better a cookie or piece of cake tastes when you're hungry) and dieters even notice and pay attention to palatable foods that much more easily. These changes can make dietary adherence more difficult. In addition to any short term changes in hunger, there are also longer term changes as fat is lost. In overweight individuals, for example, hunger increases by 5% and fullness decreases by 3% for every 2.2 lbs (1kg) of fat lost (13). The more fat is lost, the greater the effect. Although not well studied at the extremes of low BF%, hunger and appetite may be relentlessly high. In the Minnesota study the men became absolutely obsessed with food, talking about little else, near the end of the diet. This can be seen online in the behavior of lean athletes who are dieting who talk about looking at or posting pictures of tasty foods (which they call food porn) on social media. In that same Minnesota study, when the men were given free access to food, they ate with abandon, rapidly regaining weight and body fat. In fact, their hunger remained elevated long after their body fat was restored and didn't return to normal until their LBM was also regained (13). They ended up with a higher BF% than they started for this reason. It's now known that LBM sends an independent signal that drives hunger which makes preventing it's loss during a diet along with restoring it as rapidly as possible after a diet critical to avoiding fat regain (the combination of resistant training and sufficient protein, both of which were absent in that study, is the key to both goals). While the above primarily refers to food restriction per se, since it is possible to increase calorie expenditure to generate fat loss and I want to look at the impact of exercise in this regard. In the most general sense, at least in the short-term (1-2 days), exercise seems to have at most a small effect on increasing hunger. Even when people do eat more, it's usually less than the humber of calories that they burned during exercise. So someone might burn 400 calories with exercise and eat 150 more calories in a day than they otherwise would. The exercise still has an effect of creating a deficit, it's just smaller than would be expected (14). I'd mention that this short-term effect may not be representative of a long-term effect. Hunger often takes 3-4 days to even begin to increase on a diet and, over time, a deficit is a deficit and fat loss is fat loss. Eventually hunger will increase in response to exercise. I'd also note that the increase in hunger after exercise is hugely variable with some people showing a larger increase in the palatability of foods which may cause them to eat more than others following exercise (15). In leaner individuals the situation is somewhat more complex. While you would expect exercise to stimulate hunger more due to the lower BF%, in some cases, exercise may blunt hunger, causing athletes to unconsciously undereat. This may be related to the performance of higher intensity exercise and seems to interact with their diet (16). Finally I'd mention that there can be a psychological impact of exercise on eating whereby people may justify eating more (i.e. "I did an hour of aerobics, I deserve a cheeseburger.") There is a related phenomenon called disinhibition that I will discuss in a later chapter. Ultimately, it should be clear that food restriction, to some degree exercise, and fat loss tends to stimulate hunger and appetite along with making people desire/enjoy more palatable foods. However, these tendencies should only be seen as biological urges as they can clearly be overcome to one degree or another. People clearly do control their food intake both during and after a diet (I'll talk about other strategies to help deal with this) so there is no guarantee that food intake will increase. For this reason, it's relatively more important to look at the adaptations to energy expenditure. Energy Expenditure Regulation Moving to the topic of energy expenditure, recall from the last chapter that there are four distinct components of what is called Total Daily Energy Expenditure (TDEE): Resting Metabolic Rate (RMR) which are the number of calories burned at rest, Thermic Effect of Food (TEF) which are the calories burned in digesting and utilizing food, the Thermic Effect of Activity (TEA) which are the calories burned during formal exercise, and Non-Exercise Energy Expenditure (NEAT) which are the calories burned during activities which aren't exercise. As expected, all four of these both can and do change in response to dieting/fat loss. Logically, this would be expected if for no other reason that the fat loss alone. A smaller body will burn less calories at rest (RMR) and during activity (TEA, NEAT) and a reduced food intake will mean that TEF is reduced. And this certainly occurs. However, when the changes in each component are examined, there are often reductions in TDEE or each component that are greater than would be predicted on the change in weight alone. This additional decrease is called the adaptive component or adaptive thermogenesis (AT). 81 So if someone lost weight and their TDEE was predicted to drop by 100 calories but is measured to have dropped by 150 calories, that extra 50 calories is the adaptive component, another mechanism that works to slow fat loss and restore body fat levels after the diet is over. As with other components of this system, the adaptive component can be hugely variable between any two people causing one to lose fat or weight more quickly than another even on an identical diet and exercise program. This adaptive component can occur very rapidly during a diet, long before any measurable fat loss has occurred. In one study, TDEE dropped by 100 calories (ranging from 0-250 calories) within the first week. For someone ona 500 cal/day deficit, this could reduce it to 250-400 cal/day reducing fat loss immediately. After that initial rapid drop, there is a slower more gradual decrease as fat is lost. At the low extremes of BF%, the adaptive component increases enormously a although the major drop in TDEE is still from the total weight/fat loss. Finally, the adaptive component is much greater during active dieting but decreases when the diet is over. The Adaptive Decrease in TDEE Although I will look at the individual components below, I want to start by looking at the size of the adaptive decrease in TDEE first. As with the changes in hunger, much of this is determined by initial and ending BF% along with the total amount of weight and fat that is lost. In the case of the Category 3 dieter losing a moderate amount of weight, the total adaptive reduction in TDEE might be no more than 15% below the predicted level which might amount to 150-250 calories per day. This can range from zero to 500 calories per person with people showing the larger adaptive drop both losing fat more slowly and being more likely to regain it after the diet is over. At the extremes of fat loss, much larger drops have been measured. In the Minnesota study, the men showed a total drop in TDEE of 50-55% or 1800 calories per day (and large variance between the men) as they lost 25% of their total weight (and an enormous amount of LBM). This means that 25-30% of the total drop, or 450-600 calories, was due to the adaptive component while the other 1200-1350 calorie drop was due to the weight loss. More recently, and supporting the above numbers, a male bodybuilder was followed as he lost 28 lbs (14% of his initial body weight), losing 22 pounds of fat and 6 lbs of LBM (17). His TDEE dropped by 1300 calories or 37% from baseline. As 14% of that drop was due to the loss of weight, the other 23% is due to AT, broadly supporting the Minnesota study numbers. Anecdotally, both female and male physique competitors, who meticulously track their calorie and activity levels before and during a diet have reported a roughly similar drop in TDEE. These two values, 10-15% (150-250 calories) for moderate fat loss in Category 2/3 individuals and up to 25-30% (450-600 calories) for extreme fat loss in the Category 1 dieter will represent the low and high extremes for the adaptive component. The causes of the adaptive component of metabolic rate decrease are primarily hormonal. Among other changes when calories are reduced or fat loss occurs, hormones involved in energy expenditure such as the catecholamines (adrenaline/noradrenaline) and active thyroid hormones (T3) go down (the latter is due to impaired conversion of T4 to T3 in the liver). The catecholamines and thyroid hormones interact, each making the other work better and the drop in the two is a major part of the adaptive drop in metabolic rate. There are other hormones that change with dieting. Leptin, discussed below, is a major one but there are also changes in growth hormone (GH) and cortisol. The total change tends to be related to the loss of fat: greater fat loss to a lower BF% means a greater drop in hormones and larger adaptive component. In addition, estrogen, progesterone and testosterone all change with fat loss and the impact of this depends very heavily on diet Category. For the Category 3 female, the changes may actually improve her health and fertility. In Category 2, the changes are fairly inconsequential. But in Category 1, menstrual cycle dysfunction is common in women (males may reach castrate testosterone levels) as they reach the lower limits of BF%. Now let me look at each component of TDEE individually. Resting Metabolic Rate (RMR) The total decrease in RMR in response to a diet is due to two factors: the actual loss of body weight and the adaptive component described above. Here the adaptive component is due to the body become more efficient and burning less calories per pound of tissue. So not only is there less of that tissue overall but what is left is more efficient. As I mentioned in Chapter 8, RMR is predominantly related to the amount of LBM someone has. Here I am referring to all types of LBM, not just skeletal muscle. Recalling that organ, brain, etc. burns far more calories than skeletal muscle, the loss of organ mass would be expected to reduce RMR significantly. And this actually happens with a recent study (mimicking the Minnesota study for 3 weeks) found a loss of organ mass in the first week of dieting that explained most of the drop in RMR (18). There was still an adaptive component, mind you, it was simply smaller than had 82 been seen previously. Given the importance of LBM in determining RMR, it's usually been felt (and at least some studies find) that preventing LBM while dieting is the best way to limit the a reduction in metabolic rate (adaptive or otherwise). While there is at least some truth to this, the fact is that RMR tends to drop in response to large-scale fat and weight loss even if LBM is maintained (19). And this is due to the fact that body fat is sending the primary signal to the brain in terms of how it should adapt to dieting. Looking at the magnitude of the drop, a primary factor is still BF%. In the Category 3 individual losing a moderate amount of weight, the total adaptive drop may be no more than 150-250 calories. Of this decrease, perhaps 10-15% is due to the changes in RMR and this amounts to roughly 15-40 calories per day, an insignificantly small number. Even the dieter who experiences a 500 calorie total decrease will still only see the RMR drop making up 50-70 calories per day. At the extremes the picture changes with the Minnesota study being on of the very few data points available. Above I mentioned that the total adaptive drop was 1800 calories per day. Of that drop, RMR dropped a total of 40% (720 calories) of which 450 calories was due to the loss in weight an an additional 270 calories (roughly 4-5 times the drop in the Category 3 individual) was due to the adaptive decrease in RMR. This means that the majority of the total drop in TDEE, 60% or 1100 calories came from the other components of total energy expenditure. If the total adaptive adaptive drop in RMR only represents 10-15% of the total in the Category 3 individual and perhaps 25% for those who lose extreme amounts of fat, the majority of the drop must be explained by changes in the other components. I make this point as it's common to hear people talk online about how dieting has ruined their metabolism or put them into fat storing mode, at least broadly referring to the supposed changes in RMR. For the overweight individual, this is clearly untrue and, even at the extremes, it contributes less than half of the total drop (adaptive or otherwise) in energy expenditure. TEF, TEA and NEAT As the total drop in RMR can only explain a relatively small proportion of the total drop, that leaves TEF, TEA and NEAT as the possible contributors (these are sometimes grouped as Non-Resting Energy Expenditure or NREE). TEF is the simplest and ends up only being impacted by the overall decrease in food intake with no adaptive component. Since TEF is only 10-15% of total calorie intake to begin with, the change here is always fairly small. A 500 calorie food reduction only reduces TDEE by 50-75 calories while a 1000 calorie reduction in total food intake has the potential to reduce TEF by 100-150 calories. While this is certainly part of the overall adaptation to dieting and fat loss, it is clearly not a major part of the decrease in total energy expenditure. Moving to TEA, I'd first note that many studies examining weight loss and metabolic rate adaptation do not use exercise making changes here irrelevant. Only those dieters who are already on exercise programs or who are athletes will face adaptations here and there are three that might occur. The first is simply that people will be less motivated to exercise in general or be unable or unwilling to maintain the same exercise intensity. Assuming this is not the case and the exercise program is maintained, there are still two adaptations by which TEA will be reduced. The first is due simply to the reduction in body weight; a smaller body takes less energy to move. If muscle mass is lost, this effect could be even more pronounced as the dieter will find it more difficult to maintain their previous intensity in terms of running or cycling speed, weights lifted, etc. Fewer calories will be burned even if the same total amount of exercise is done. There is also an adaptive component where muscular efficiency may increase, causing 25% fewer total calories to be burned (i.e. someone burning 100 calories will now only burn 75), especially during low intensity activity (21). In premise, wearing a weight vest to restore the weight loss has some potential to reverse this but it can only partially do so (21a). In order to burn the same amount of calories, either more total exercise or exercising at a higher intensity will be required. While these adaptations occur, both for the non-exerciser (for whom the above is irrelevant) or the trainee, the changes still cannot explain the majority of the adaptive drop in TDEE. If RMR, TEF and TEA cannot explain the majority of drop in daily energy expenditure, only changes in NEAT are left. And, just as it represents the larger contributor to the variance in TDEE between people, changes here turn out to explain the majority of the drop that is seen with dieting. Ignoring the unconscious aspect of NEAT (fidgeting, changing posture), which may decrease, and focusing on the most conscious NEAT types of activities, reduction in calorie expenditure can occur here for a few reasons. The first two are identical to what happens with exercise: a smaller body burns fewer calories and there is the increased muscular efficiency that occurs. Since that efficiency primarily impacts on low-intensity activities, and many NEAT activities are low-intensity by definition, the change here could potentially have an even larger impact than it does on TEA. 83 Larger than either of those is the fact that NEAT levels typically go down with calorie restriction. People not only perform less of the activities which burn calories via NEAT but unconsciously perform less of higher-intensity activities. Clearly demonstrating this is a study where calorie restriction from 10-30% below maintenance resulted in daily reductions in NEAT from 100-500 calories per day (22). There was a 100-200 calorie per day variance between subjects with the people showing the larger drop being the ones who lose fat more slowly/regain it more rapidly. While not studied to my knowledge, given the fact that NEAT is often very low in overweight individuals, NEAT could potentially increase somewhat with weight loss as it becomes easier to move. As well, it's possible that those individuals with a lower initial level of NEAT see a smaller reduction simply because there is less to reduce. Regardless, the overall picture is that NEAT goes down when dieting. To put this into further perspective, I'd mention the Biosphere 2 study which accidentally turned into a 2-year long diet (23). While reaching a very low BF%, the subjects (both women and men) experienced an insignificant 2.7% decrease in RMR while their daily NEAT was reduced by half (23). Looking back at the Minnesota study, as I noted above, the drop in RMR can only explain 40% of the total drop (and most of that was from weight loss) leaving 60% or 1100 calories to be explained by TEF, TEA or NEAT. TEF can only explain perhaps 150 of those calories leaving TEA and NEAT. While we might debate if it counts as formal exercise, the men were forced to walk for 2 hours/day but performed no other exercise. Even the decreased calorie burn here still can't explain the largest part of the decrease. While not directly measured (since the concept and technology didn't exist), this means that NEAT the remainder of the day must have been reduced enormously due to fatigue and exhaustion. At the extremes of low BF%, exhaustion sets in causing people to unconsciously move around as little as possible if they are not forced (or are forcing themselves) to exercise. Maintaining normal exercise intensity is difficult enough under these conditions and keeping NEAT from falling excessively can be nearly impossible. All of this points to the simple fact that while changes in RMR, TEF and (if exercise is present), TEA are part of the overall adaptation they cannot explain the majority of the change. Just as differences in NEAT explain most of the variation in daily energy expenditure between any two people, changes in NEAT explain most of the drop in energy expenditure that occurs while dieting. And while a large part of this reduction in NEAT is assuredly in the unconscious part of it, this at least gives dieters the possibility of seeing less of a drop in energy expenditure by working to maintain NEAT. In this vein, people who sit more while dieting (meaning a lower NEAT) have been found to lose less weight on a diet than those who sit less (24). By deliberately choosing to engage in activities that increase NEAT such as parking further away from the store, using a standing or treadmill desk, etc. NEAT levels will either be better maintained or even potentially increased. New technology holds promise here with apps and activity trackers which give daily feedback on NEAT (indicating when it needs to be increased). Even a pedometer can be used and targeting a specific number of steps per day will increase NEAT. There is also formal exercise to consider. On the one hand, increases here could possibly offset the decreases in NEAT and work to maintain daily energy expenditure. On the other, exercise itself has the potential to decrease NEAT in some situations and it's been argued that human energy expenditure may be genetically set with increases in one component being offset by reductions in another (25). In one study, subjects performed 400 calories per day of exercise but only increased daily energy expenditure by 250 calories due to a reduction in NEAT at other times of the day (26). The impact of exercise on NEAT seems to be most pronounced in beginners and older individuals although trained athletes often reduce NEAT after particularly exhausting workouts. This effect tends to go away as fitness improves and the exercise isn't as fatiguing but this actually makes a case for lower intensity activity being a better choice in many circumstances. So consider the situation where a dieter performs a hard 400 calorie workout which causes them to reduce NEAT later in the day by 150 calories for a net 250 calorie increase. Compare that to a dieter who performs a more moderate 250 calorie workout who sees no reduction in NEAT. Both have burned the same 250 extra calories per day but the second achieved it with a much more enjoyable and sustainable approach. In this regard, the activities that increase NEAT are generally low intensity yet have the potential to greatly impact on TDEE. As I showed in the last chapter, increasing calorie expenditure from even 1 to 3 cal/min during an 8 hour work shift can burn nearly 1000 extra calories per day without causing undue fatigue. It would require 90 minutes of exhausting exercise to do the same and some of the calorie burn might very well be offset by reductions in NEAT later in the day. 84 Other Metabolic Adaptations to Dieting In addition to the changes described above, there are a host of other adaptations that occur in response to dieting/fat loss. One that is often unconsidered is sleep which can become disrupted with dieters either having trouble falling asleep or staying asleep (I'd remind readers that women's sleep can become disrupted during the late luteal phase of the menstrual cycle to begin with). A large amount of research has recently linked shortened sleep, below 6 hours/night, with increased hunger and appetite along with reduced energy expenditure due to fatigue (27). This is regulated through a chemical in the brain called orexin which regulates sleep and energy balance along with being involved in how much NEAT someone performs (28). Moving away from the energy balance equation per se, other changes occur that serve to both limit further fat loss and encourage fat gain if food intake increases. In Chapter 7 I mentioned that it become more difficult to mobilize fat from the fat cells as they shrink and they become less sensitive to fat mobilizing hormones. Along with this is an increase in fat cell insulin sensitivity meaning that insulin has a greater impact on inhibiting fat mobilization. Enzymes involved in fat storage also increase. As it becomes more difficult to mobilize and use fat for fuel, the proportion of LBM being lost increases (29). This can become significant as dieters reach the lower levels of BF% and I described previously how loss of LBM on a diet can increase hunger both during and afterwards (recall that women lose less LBM than men which is one of their fewer advantages in this regard). While all of the adaptations I have described are popularly referred as the body going into fat storage or fat hoarding mode (or in some cases metabolic damage), they really represent a completely normal and logical adaptation to dieting and fat loss. During the diet they work to slow fat loss and may even cause it to stop if energy expenditure decreases until it matches energy intake. However, claims that people start regaining fat while still in a calorie deficit are nonsensical. Rather, the body is simply primed to store fat at an accelerated and more efficient rate if and when calorie intake increases. How Long Do the Adaptations Last? A question that is often asked regarding the above adaptations is how long they last after someone has lost some amount of fat and kept it off. And the answer, depressingly, is effectively forever. Research has examined this over time periods ranging from 7 months to 7 years of post-diet weight maintenance and at least some amount of adaptation to TDEE is still present (30). So long as bodyweight and body fat are maintained below the pre-diet level, energy expenditure will never return to where it was prior to the diet. As with the initial adaptations themselves, much of this is due to maintaining a reduced bodyweight and food intake with RMR, TEF, TEA and NEAT all being reduced for that reason alone. But at least some proportion of the adaptive component also remains. It is reduced compared to during the phase of active dieting but it is still present. The question then becomes how much of an impact it has. And the answer is, at least for Category 3 individuals, not very much. In one study, the total drop in TDEE was 500 calories per day there was only a 3-5% adaptive decrease in RMR present (31). This amounts to perhaps 40-70 calories per day and again changes in RMR really aren't responsible for the large scale changes that are occurring. Adaptive changes in the other components have not been studied to a great degree but it is likely that they are maintained to one degree or another. Certainly the motivation and ability to exercise can increase when food intake is raised and it's possible that NEAT will recover or increase once the person is no longer active dieting. But the fact remains that TDEE will always be reduced with some degree of the adaptive component still being present. At least some increase in hunger and appetite is also usually seen although, once again, it will be less than during the period of active dieting since more food is being eaten and at least some hormonal recovery will have occurred. Most dieters report some fairly permanent degree of residual hunger and loss of fullness even if they are no longer dieting. Since they aren't often included in diet studies, very little work has been done on lean individuals in terms of how much change in TDEE or the adaptive component is occurring. When they are done they either examine eating disorders such as anorexia where extremely low body weights or are comparing female athletes with menstrual cycle dysfunction to those without (I will discuss this in detail in Chapter 12). Regardless, someone who is attempting to maintain a very low bodyweight/BF% is likely to experience all of the adaptations to an even greater degree in terms of a large scale reduction in TDEE, a relatively large adaptive component (that never disappears) along with increased hunger and appetite. Ignoring the fact that it is not generally healthy for people, women moreso than men, to maintain extremely low BF% for extended periods, it's worth addressing whether the above can be addressed or dealt with. Here exercise, especially for the general dieter has the potential to play a huge role. There are several reasons for this. One is that increased exercise can help to offset the metabolic adaptations which 85 are still present. It will also allow for more calories to be eaten, offsetting some of the issues with hunger and appetite, while still maintaining energy balance. Regular exercise is also often associated with better adherence to the diet which provides another benefit when maintenance is the goal. In this vein, while exercise do not generally improve weight or fat loss per se, it has been shown to improve long-term weight maintenance, assuredly for the reasons listed above (32). It's worth mentioning that, in direct contrast to the above, many who maintain a lowered bodyweight/BF% for extended periods often report that it becomes easier with time. Since it's clear that some amount of metabolic adaptation will always be present (and that the "Setpoint" never goes down), the reason is assuredly behavioral with people's new activity and eating patterns becoming more and more ingrained and automatic. Adaptations to Overfeeding and Weight/Fat Gain Having looked at the adaptations that occur to dieting, I want to look at what can or does happen in reverse, when people overeat and gain weight and fat. There are two situations here, one when someone gains weight from their current weight and the other when someone regains weight after a diet. Overeating from a Baseline Weight and Body Fat Looking first at what happens when people gain weight or fat from their current weight, I'll first say that if bodyweight/body fat are regulated in any meaningful way, it would be expected that the adaptations that occur with dieting would occur in reverse with weight gain. Appetite, hunger and the rewarding nature of food should decrease while TDEE should increase. In response to a 10% weight gain, RMR has been shown to go up about 20-25% (~150-250 calories with large variation) with 10% being due to the weight gain itself and the rest the adaptive component.

Surprisingly this was seen in both women and men and lean and overweight subjects. Other studies, usually providing 1000 calories per day of overfeeding, have found broadly similar results with at most an increase in RMR of perhaps 100 calories (33,34). TEF also increases in all situations due simply to eating more calories. This increase can't offset the total calorie surplus and an 8-9 fat gain is common. At best it helps a little and makes it slightly easier to return to the pre-overfeeding body weight. Assuming formal exercise is being done, TEA would be expected to increase. A larger body burns more calories and, if muscle is gained, higher power outputs, running speeds, weights being lifted, etc. should allow more calories to be burned. Muscular efficiency also decreases with weight gain so that more calories are burned. If weight gain is excessive, this can make exercise more difficult but there is a limit to this effect. As with dieting, that leaves NEAT as the place where potentially the largest impact might be seen. The same effect on bodyweight and muscular efficiency will be present here leaving the question of whether or not NEAT will increase with overfeeding. And the answer is that it does for some individuals. In the study that first identified NEAT, women and men were placed in a metabolic chamber before being overfed by 1000 calories per day (36). Based on the calorie surplus, fat gain should have been enormous but ranged from 3 to 15 pounds between subjects. And while there were small increases in RMR and TEF (exercise was not allowed), the major difference was in NEAT. Subjects who increased NEAT the most gained the least fat and vice versa. In fact, some subjects increased NEAT by an incredible 700 calories per day (leaving 300 to be stored) while one female subject actually decreased her NEAT. The impact was not only potentially enormous but enormously variable. The cause of the adaptive response to overfeeding is, once again, hormonal with hormones moving in opposite directions as seen with dieting. Levels of active thyroid increase as do the catecholamines with reproductive hormone levels improving and cortisol and GH levels going down. At some point, this does go awry, with obesity, both women's and men's hormones being negatively disrupted. But with moderate weight gain, the impact on hormones is generally positive in terms of increasing TDEE via an adaptive component with NEAT still playing the potentially major role. Hormones, Appetite and Weight Regain After a Diet Let me now address what happens when people begin overeating and regaining weight and fat after having previously dieted down and this occurs for two primary reasons. The most common is that people succumb to increased hunger, appetite and attention/enjoyment of palatable foods and start overeating again. The second is the athlete who has dieted to a low BF% for temporary (usually competition) reasons who needs to deliberately regain fat afterwards. In both cases, the starting situation is relatively identical in that hunger and appetite will be increased while daily energy expenditure will be decreased. As discussed in the sections above, what primarily differs is the degree of changes seen with greater adaptations in 86 both being seen as BF% goes lower. And in both situations, at least in the broadest sense, hormones, hunger, appetite and the various components of energy expenditure will recover and finally reach baseline when the dieter reaches their pre-diet BF% and body composition levels. Whether this happens quickly or slowly depends on how rapidly or slowly that pre-diet body composition is reattained. I'd mention that, despite various claims of "reversing metabolic damage" there is no practical way to restore TDEE (except perhaps by drastically increasing activity levels) without regaining the lost body fat to pre-diet levels. Since fat sends the primary signal here, it has to be regained to restore physiological normalcy. Recall from above that LBM is involved here and the system will not reset to true normalization until both fat and LBM have been restored. Demonstrating this, in the case study of the male bodybuilder, since less LBM was lost and he was training and eating sufficient protein as he regained weight and fat, no excess body fat was gained. His metabolism and presumably his hunger levels returned to 100% of it's starting value at the same BF% (~15%) he had started at. I will discuss several case studies of female physique athletes in Chapter 34 where the same phenomenon was seen. Just as with the recovery in energy expenditure with overeating, hormone levels will recover back towards normal. Levels of active thyroid hormone will increase back towards baseline as will the catecholamines and reproductive hormones. Cortisol will decrease as well. Just as energy expenditure won't recover until the level of body fat is reattained, neither will hormone levels (as well they will go above normal if body weight/body fat overshoots it's starting place). Individuality and Interrelationships In several of the sections above, I mentioned that there is a fairly large variation between any two individuals in the adaptive response of metabolic rate to either dieting/fat loss or overeating/fat gain. As it turns out, these responses are inter-related, partially explaining why some people seem to remain naturally lean in the modern world while others do not. For example, the people who increase energy expenditure in response to cold exposure also raise it the most in response to overfeeding (37). There is also huge variance here with the increases ranging from 38 to 380 calories per day. Similarly, there is a direct relationship between the increase in energy expenditure with overfeeding and the drop with dieting (38). People with a spendthrift metabolism raise TDEE the most with overfeeding and have it decrease the least during dieting; they gain weight with difficulty but lose it easily. In contrast, those with a thrifty metabolism show the least increase in TDEE with overfeeding and the largest drop while dieting; they gain weight easily and lose it with more difficulty. Does Yo-Yo Dieting Permanently Impair Metabolism? A commonly heard claim is that the process of losing weight/fat and then regaining it, often called Yo-Yo dieting (or weight cycling) can cause a permanent impairment of metabolic rate or make fat loss more difficult in the long run. I want to address both how this might occur and whether or not it has been found to happen. One way this might occur would be due to body composition worsening with a given cycle of diet and regain. If someone lost a significant amount of LBM due to poor dieting practices and failed to regain it after the diet, they would end up at a higher BF% (even at the same weight) which could potentially impact their energy expenditure. However, considering the small difference in calorie use between muscle and fat (replacing 5 lbs of LBM with fat only changes RMR by 30 calories), the only real way this might occur would be if repeat diet cycles caused this to happen repeatedly. Alternately, it's possible that where fat is lost from and regained could have an effect. If fat were lost from a relatively easy to lose area but regained in a more difficult to lose area, this might have a long-term negative effect. This might actually occur to a small degree and I will discuss this in the next chapter. There could also be some sort of permanent change in the brain or the hormones that respond to changes in body fat. In general, research has not found this to occur. It's far more likely that people with thrifty metabolisms are starting with a biology that makes fat loss more difficult rather than it getting worse with repeated cycles of dieting. Direct studies show that repeated cycles of weight loss and regain seem to have no long lasting impact on most components of energy expenditure (40). The changes when weight and fat are lost are reversed when the weight and fat are regained. While not the target audience of this book, this may not be the case in younger individuals. Wrestlers who weight cycled end up with a 15% lower RMR (250 calories per day) than those who didn't and this is significant (41). Given the common tendency for women 87 to start dieting at fairly young ages, it's possible that repeated cycles of loss and regain could have negative long-term effects. Even here, women recovering from anorexia (perhaps the most extreme example) completely normalize their metabolic rate when their body weights are normalized (42). A very small amount of data has documented a permanent reduction in NEAT with weight cycling. Two of those are case studies in arctic explorers and the same occurred in the Biosphere experiment I mentioned above. Nobody knows why but this shows that NEAT is still the major place where changes occur. Anecdotally some dieters do report greater difficulty in subsequent diets but this is likely due to issues with adherence. This isn't to say that there couldn't be a long-term metabolic effect but it simply hasn't been seen in the research to date. At the same time, physique athletes often report finding that getting very lean becomes easier with repeated diet cycles, likely due to the combination of good dieting practices and determining how best they should implement their own diet. Hormonal Responses I want to wrap up this chapter by examining some of the hormonal changes that occur in response to both fat loss and fat gain in slightly more detail than above, primarily focusing on the hormones that were not discussed in previous sections. I mentioned the concept of a setpoint early in the chapter, that a body weight or body fat that is regulated or defended by the body. For this type of system to work, the body would need to know how much someone was eating or how much fat/weight they were carrying so it would know how to adapt. The relevant structure here is called the hypothalamus and you can think of it as integrating signals that are coming into the brain from all over the body. Depending on what it senses, it tells a gland called the pituitary gland, a gland that controls other glands, what to do. So the hypothalamus might signal the pituitary to tell the thyroid gland to produce more or less thyroid. The pituitary also regulates reproductive function via LH and FSH. But it's the hypothalamus controlling the entire system. A huge number of signals "tell" the hypothalamus what is going on in the body. I already mentioned some of them above, CCK, PYY, GLP along with changes in blood glucose, amino acid and fatty acid levels will signal the brain about nutrient intake. Insulin is released primarily in response to eating with insulin levels signaling the brain about both nutrient intake and body fat levels. Ghrelin is the only hunger hormone known and levels go up with dieting and fat loss. Released primarily from fat cells, leptin's discovery in 1994 changed the face of obesity research as it showed that fat cells were an active tissue involved in controlling metabolism. In terms of fat cell signaling, leptin provided a signal to the brain about not only how much body fat was present but how much was being eaten. Leptin levels are directly related to subcutaneous fat levels (leptin is related to visceral fat levels) and women produce about 3-4 times the leptin as men. Leptin changes in response to both short-term food intake (dropping by as much as 50% in only 7 days of dieting) and fat loss (dropping much more slowly). Leptin can increase just as rapidly, increasing with as little as 5 hours of overfeeding. Overall, leptin is a major part of the signal to the hypothalamus that someone is either losing or gaining fat. And leptin ends up coordinating the entire system that is involved with appetite, hunger, etc. It impacts on how well hormones such as CCK and PYY work, directly controls the signals sent by the hypothalamus to the pituitary and directly inhibits cortisol release. Leptin plays a role in controlling brain levels of DA and serotonin along with directly impacting on fat mobilization and fat burning in muscle. When leptin levels are normal, the system works more or less correctly. I say more or less as the obese may be resistant to leptin's effects. Initial studies attempted to generate weight loss with leptin injections but this proved ineffective. In the same sense, the leptin injections could reverse all of the adaptations described above occur with the degree of adaptations being related to the drop seen (43). Quite a lot of research has been done that showed that, when leptin is replaced during dieting, leptin injections did mostly reverse the adaptations seen (44). Basically, falling leptin induces metabolic adaptation but increasing levels above normal do not. This raises the question of why the Leibel study showed the energy expenditure adaptations in the women who became lean regardless of their body fat level. Even though leptin's decrease coordinates the adaptations to dieting. Finally, while women and men have similar metabolic set points, there are no good reasons that I'm aware of why men and women will differ in their responses, but more data is needed. There are some other signals that women should be aware of. Even that doesn't explain why the system appears asymmetrical with weight gain being resisted less strongly than weight loss in some people (46). Most likely the effect there is due to the environment where the hedonic system can easily overwhelm any metabolic effects. Even animals who normally regulate body fat well become obese if fed the modern diet and humans are no different. Regardless, let me now finally turn my attention to gender differences in both physiology and metabolism. Chapter 10: Women, Fat Gain and Fat Loss: Part 1 Having examined a great deal of background information in the previous chapters, I want to now examine in some detail issues in terms of fat gain and fat loss. For this topic it will discuss there is generally at least one difference between women and men and while research continues to unravel the specifics and details, the major distinctions have been established. Since there is a great deal of information to cover, I have decided to split it into two chapters. This chapter will focus primarily on the underlying physiological differences while the next will look at how women differ from men in the broader sense which is more practical. In this chapter I look at body composition first before looking briefly at some of the effects of obesity, disease, fat loss and starvation. The different components of energy balance will be discussed next including differences in hunger, appetite and food preferences along with gender differences in energy expenditure. Next I'll examine how the different nutrients are used both after and between meals as well as during and after different types of exercise. Finally I'll look at the topic of what is being lost (i.e. LBM vs. fat) during a diet and address the issue of weight loss and how it can impact on body fat distribution. As needed, I will look at the impact of hormonal modifiers on all of these issues. Due to the large amount of information I want to examine, this chapter will jump around a bit between topics and a large amount of the information will come from a number of recent review papers. While it will not all effectively the same topic in broadly the same way, each takes a slightly different approach to the issue which is why I'm including them all (1-9). Individual references will be included as needed. Gender Differences in Body Composition While I discussed body composition, the relative proportions of fat, LBM, etc. in general terms in Chapter 5, I want to first look at specific gender differences in this regard. Women and men differ in nearly every aspect here and I'd note that the grand majority of changes do not show up until puberty. At this point, the increase in reproductive hormones interact with genetics and early physiological programming to generate the differences that are seen. On average, women are both shorter and lighter than men. But while the BMI, which is nearly the same, women have more body fat than men. Body Mass Index (BMI), discussed in Chapter 5, women carry approximately 10-12% more total body fat than men, some of which is sex-specific fat such as breast tissue (9a). Women also have more total subcutaneous fat and less visceral fat than men. In addition to carrying more total body fat, there are significant differences in body fat distribution between women and men with women carrying more fat in their lower bodies. Despite this, women often have more total fat in pounds of kilograms in their upper bodies than men. Women also have more total fat cells than men in both the lower and upper body. Finally, women also store more intraMuscular Triglyceride (IMTG, fat within the muscle) than men. Given that women have 10-12% more body fat than men, by extension they carry 10-12% less lean body mass (LBM) and here I am talking about all LBM, not just muscle). Of that total LBM, women carry about 5% less total muscle mass (47% vs 51%) than men due to having slightly more organ mass (9b). Proportionally, most of women's muscle is carried in her lower body compared to her upper body as well. For comparison, I've shown a woman and man at the same BMI in terms of their average body composition along with a woman "scaled up" to the same weight as a man. Woman Man Scaled-Up Woman Weight 150 165 165 BF% 22% 12% 22% Total LBM/Muscle* 117 lbs/55 lbs 145 lbs/74 lbs 129 lbs/60.5 lbs Total fat 33 lbs 20 lbs 36 lbs *Assumes 47% of total LBM is muscle for women and 51% of total LBM is muscle for men Let me reiterate a point I made in the first chapter which that the above represents only averages and only when comparing women and men who are the same circumstances. Any individual woman may be fuller or heavier than any individual man; on average they are not. A highly trained female athlete might have more LBM and less BF% than a smaller untrained male but this is not a fair comparison. When comparing an average woman and an average man, certain gender differences in body composition are fairly significant. Of more relevance to this book, there may be differences in how they see changes in body composition. At the same time, some of the supposed gender-based differences in physiology. Early studies made a common mistake of comparing women to men and just measuring some outcome in absolute terms. So consider a piece of research that determined that women burned 7 cal/min and men burned 10 cal/min doing the same task. This looks like a clear gender difference and the simplest conclusion would be that some physiological or hormonal difference was the cause. Except that you'd fundamentally expect women to burn less calories performing a given task simply due to being smaller on average. And when you correct for those differences in weight or body composition (and there is a great deal of technical argument about what the best way to do this), most of the differences disappear completely. It's not always all of them but most of them. Put differently, say you took the above values of 7 and 10 cal/min and then divided it by the women and men's body weights. So you might take the 7 and 10 cal/min values and divide by total LBM, finding that both women and men burned 0.25 cal/min/LB of LBM or something (the numbers are just for illustration) causing the gender difference to disappear. This is due to the fact that, with minor exceptions, a woman's body tissues are the same as a man's. Under a microscope, her muscle tissue looks more or less identical to a man's. She simply has less of it. Her liver would look more or less identical to a man's. Hers is just smaller. And this holds for most tissues in the body. Perhaps the biggest difference here is in the fat cells due to differences in women and men's fat distribution and the differences in how fat is stored. Women carry more of their fat in different parts of the body either more or less fat. Her larger amount of lower body fat will be more difficult to lose than his greater amount of upper body fat. Phrased differently, if a woman and a man had an identical body composition in terms of weight, total LBM and muscle mass and BF%, their physiologies would be almost indistinguishable in many ways. It's simply that this is almost never the case unless someone is comparing a highly trained muscular/lean female athlete and an untrained male. In practice, there will always be some apparent gender differences in physiology. Which isn't to say that there aren't still other differences in physiology outside of just body composition. There are with most of these being driven by the differences in reproductive hormone levels between women and men along with any changes that are occurring throughout the month. I'd add that even if these differences are relatively small, they still have enormous practical implications for diet and fat loss. I mentioned one of these in Chapter 1, how carbohydrate loading becomes nearly impossible for female endurance athletes because the amount of carbohydrate needed may exceed their daily calorie intake. Even if they have a greater capacity to store it, they still might be limited in other ways. The same holds true in body composition. Women's lower total calorie requirements, women often can not alter their diet to the same degree as a larger man would be able to. I'll discuss in the next chapter that, to burn the same number of calories with exercise, women have to exercise more or at a higher intensity due to being lighter. Effect of Hormonal Modifiers on Body Composition While a woman's average body composition develops at puberty, it can be impacted to one degree or another by the hormonal modifiers. In general, PCOS women are at higher risk of being overweight to begin with. As well, those PCOS women with elevated testosterone levels (and to a lesser degree women with subclinical hyperandrogenism) tend to have a more male-type fat patterning with more fat around the midsection and an increase in visceral fat. They frequently carry more skeletal muscle/LBM than other women although their BF% will still tend to be higher as well. As mentioned in Chapter 3, by and large hormonal birth control (BC) doesn't seem to impact body composition although some types may cause a small increase in fat with a small loss of LBM. Depo-provera is an exception and is associated with the greatest weight and fat gain. As obesity is defined by BF% to begin with, obviously overall BF% is higher approaching 50% or more of total weight at the extremes. With increasing weight there is some increase in LBM although this eventually hits a maximum with all further weight gain being from body fat. With aging, there is often a worsening of body composition some of which is due to age and most of which is due to reduced activity levels. As women approach or enter menopause, there is often a significant worsening and change in body composition if hormone replacement therapy (HRT) is not undertaken. Both total weight and body fat increases with fat patterning moving to a more male pattern (increasing relative disease risk) and LBM is lost. HRT appears to reverse the majority of this (9b). 90 Women, Obesity, Disease, Fat Loss and Starvation While a great deal of the perceived gender differences in physiology more or less disappear when the differences in body composition are taken into account, it's also clear that there are differences that cannot be explained solely by this. I'll address the specific physiology below but first want to look at a number of observations that have been made that show quite clearly that women and men differ in both their response to diet and exercise and with regards to the amount of fat they can lose. On top of carrying a larger percentage of body fat (and often more total fat) than men, women are far more likely to become obese than men with the biggest difference seen after women go through menopause. As well, women are three times as likely to be found in what is called the super obese category, marked by the highest extremes of BF%, in some cases as high as 50-60%. Clearly, within the context of the modern environment, women's bodies do respond differently in terms of their propensity to gain fat. Despite this, women are relatively more protected against developing certain diseases such as diabetes/insulin resistance (PCOS women excepted) and heart disease which are often associated with excessive body fat levels, especially when they are younger. This is due to having more subcutaneous fat in general and more of it stored in their lower bodies in specific (this pattern being more metabolically healthy overall). In contrast, men tend to carry more visceral fat which is where the health risks lie. When women start to gain visceral fat (as occurs with elevated testosterone levels, at menopause or in obesity), their risk of these diseases increases. Looking at weight and fat loss, it's generally felt or argued that women have more trouble losing weight and fat than men. Either they lose less total weight/fat or lose it more slowly (1). This has been found in the research when it comes to weight loss as well and the discussion from a recent paper that looked at the results of both women and men that were tested over one year. The same women lost a lot more. In the context of the past century, with many of the relevant data coming from situations in the early 20th century such as war and famine but the pattern was seen when showing a higher survival rate. And this is due to physiological reasons that make women better able to handle a lack of food, better conserving energy for survival in the long-term. I'll look at the mechanisms and reasons (in an evolutionary sense) in the next chapter. For now let me look at potential gender differences that could contribute to the overall differences that are seen. Differences in Appetite and Hunger (1) As I have with previous chapters, let me start by looking at gender differences in the various components of energy balance. In terms of overall calorie intake, women generally eat less than men although this is primarily due to their smaller body size. Women, in general, tend to eat more carbohydrates than men. At the same time, there are a number of factors that show women are a bit more hormonal influences at work. As testosterone stimulates hunger and appetite, women's much lower levels are often in response to the hormonal signals involved in fullness. For example, women show higher overall levels of ghrelin (which recall stimulates hunger) and experience a smaller drop when they eat. As described in the last chapter, both leptin and insulin play a role in regulating hunger. Here there is another difference with women's brains responding more potently to leptin and men's brains responding more potently to insulin. As leptin is related more to subcutaneous fat (found in greater amounts in women) and insulin is related more to visceral fat (found in greater amounts in men), this makes some degree of sense. But while insulin changes rapidly in response to a meal, leptin does not and this might be another part of why women's hunger doesn't decrease as much after a meal. Finally, women do not see the same reduction in hunger when they eat more meals while men do (16a). Women's taste buds differ from men's and they experience foods as sweeter and creamier than men do. Women also often prefer the taste of carbohydrates and fats while men prefer protein and fats (16b). Somewhat related to this, while obese women show a relationship between their total carbohydrate and fat intake and their body fat levels (lower carb/higher fat intakes meaning a higher body fat level), men show no such relationship (12). Women frequently lament that the same high-fat, high-calorie foods that cause them to readily gain fat don't cause the same in men and I'll explain why below. It's important to remember that most, if not all, of the above changes to one degree or another during the menstrual cycle with a follicular phase (especially in the days before ovulation) while increasing in the luteal phase (along with this comes an increased cravings for carbohydrates and fat). Meals might digest more quickly during the luteal phase, meaning that women will not stay full as long and may be more likely to overeat (12a). As already discussed, this is due to the changes in estrogen and progesterone which are occurring. 91 A final issue to consider is more psychological than physiological and has to do with what researchers called restrained eating, which is similar but not identical to dieting, and disinhibition (which I mentioned briefly previously). Restrained eaters are those who are concerned with their bodyweight and overall food intake while disinhibition refers to a situation where environmental triggers such as stress, being around others who are eating or being around highly palatable foods causes them to break their diet and overeat (13). Restraint can actually be rigid or flexible which actually have very different effects in this regard but I will discuss this in a later chapter. I bring up the topic here as women are not only more likely to be concerned about their weight and/or be actively dieting but tend to have higher dietary restraint than men (14). If they are rigidly restrained, they may be at risk for disinhibition and overeating. Effect of Hormonal Modifiers on Food Intake Looking briefly at the potential effects of the hormonal modifiers, I mentioned in Chapter 3 that, overall birth control doesn't seem to increase hunger or appetite. This depends on the generation of progestin with first and second generation progestins potentially increasing hunger and appetite while the third and fourth generation lack this effect. Due to the hunger stimulating effect of androgens, women with subclinical hyperandrogenism or PCOS may experience greater hunger and appetite overall. PCOS women have also been found to eat more highly palatable carbohydrates and saturated fats and that their bodies do not regulate hunger as well as they should (15). Finally, women with PCOS are also more prone to binge or other types of disordered eating (15b, 15c). As I mentioned the obese often show a hunger, appetite and reward system that isn't working well in the first place although this may be as much a cause of them becoming obese as a consequence. Regardless there is often a dysregulation of the systems involved in hunger and appetite that contributes to the problem. Given the role of estrogen in decreasing hunger, any situation where estrogen levels decrease would be expected to increase hunger. This includes the loss of menstrual cycle (amenorrhea) and it's known that hunger and food intake increase at menopause (moreso if HRT is not begun). Along with other changes in physiology, this all contributes to age-related gains in weight and fat. Differences in Energy Expenditure Moving to the energy expenditure side of the equation, I want to look at each of the four components which make up total Daily Energy Expenditure and will do so separately. Differences in Resting Metabolic Rate (RMR) Since women are generally shorter and lighter than men, their RMR will be lower. For many years this was felt to represent an inherent gender difference related to some aspect of physiology. But as I described above, almost all of the difference ends up being related to the differences in body composition. As RMR is related predominantly to LBM, women's lower levels will mean a lower RMR. When RMR is normalized for LBM, the difference disappears (16). As noted above, women's tissues burn the same number of calories per pound as a man's, they simply have less of them. That said, research has found that women may have slightly lower or higher energy expenditures relative to their LBM than men. In the follicular phase, it may be slightly lower while in the luteal phase it may be slightly higher. In both cases, the difference is only a few percentage points either way which is insignificant. Overall women do show a lower Thermic Effect of Food (TEF) but this is primarily due to eating less total food. Limited research shows that, as a percentage of total calories, women have a slightly lower TEF than men although the difference is quite small (16a). TEF does change throughout the menstrual cycle, being slightly lower during the luteal phase but the effect is only about 3 calories per meal (16b). One unstudied, but potential gender difference, could be related to the choices of foods. As dietary protein has the highest thermic effect, women's tendency to underat protein relative to carbohydrates and fats might lead to a somewhat lowered TEF as well. Any effect here will only be for the most extreme dietary comparisons and the overall small effect of TEF overall make gender differences fairly insignificant. Moving to activity, women have been found to be less physically active than men at all ages although it's difficult to tell if this is an actual biological difference or driven more by environmental and social factors (17). What is clear is that women even exercise for different reasons than men with women being driven by more external factors such as body weight and appearance while men tend to emphasize aerobic training while men emphasize resistance training. In terms of the energy expenditure of formal exercise (TEA), women generally burn fewer calories during exercise than men for two primary reasons. The first is due to simply weighing less as it takes more calories to move a heavier body. The second is related to body composition with women's lower LBM meaning they generate less force. As there are also the issue of power outputs, weights lifted, etc. 92 Finally there is Non-Exercise Activity Thermogenesis (NEAT) which has not been well studied in terms of gender differences. As with formal exercise, women will burn less calories during NEAT due to their lower body weight. Women would be expected to have lower NEAT than men. What little data exists suggests that it depends on societal factors in terms of what tasks women do or do not perform. On the Ivory Coast, where women perform 95% of domestic and 30% agricultural duties, their NEAT is higher than men's. In Australia, Canada and the UK, women's levels of NEAT may be 1/3rds that of men's. In the United States, women and men's NEAT appear to be roughly equal (18). This doesn't indicate if there are any true gender differences outside of environment for NEAT. At most there is the indirect observation that the four worst responders in the overfeeding/NEAT study I described in the last chapter were women. But more research is necessary. Effect of Hormonal Modifiers on Energy Expenditure There tends to be limited data on the hormonal modifiers and their effects on the different components of energy expenditure. Birth control does not appear to have a significant impact on RMR. In PCOS women, the combination of insulin resistance and elevated testosterone can actually increase a woman's RMR by about 5%, similar to what is seen in the luteal phase, due to the progestin component. Amenorrhea, discussed in detail in Chapter 12, is associated with a significant decrease in RMR, similar to what is seen at the lower limits of BF% in men. While women with PCOS appear to have a normal RMR compared to non PCOS women with a similar body composition although they do show a slightly reduced TEF (perhaps 30 calories per meal) in response to a meal (18a). Overall, the effects of these differences are small. Obese women show a greater age related decrease in energy expenditure than men, roughly 143 calories/decade versus 34 calories, probably related to changes in body composition (19). At menopause energy expenditure goes down due to both the loss of LBM and an apparent decrease in energy expenditure in the organs; this is reversed with hormone replacement therapy (20). Two Implications of the Above Before delving deeper into the physiological gender differences, I want to address two practical implications of the above sections relative to fat loss (and I will mention these again). The first is that, for the most part, due to their lower body weight and total calorie intake, women are frequently unable to achieve the same degree of calorie restriction as men. For a woman with a maintenance calorie level of 1700 calories to reduce her food intake by 500 calories per day is 30% decreased (a drastically different than a man with a 3000 calorie to do the same (only a 16% decrease). Not only will this impact her fat loss it also means that she will have to face less food to face for hunger Category 3 women as their lighter starting food intake allows a greater reduction with fewer problems. The second is related to exercise with the same issue arising. To be doing smaller and having less LBM, women burn less calories during any given workout. She will have to exercise more or at a higher intensity to burn the same number of calories. Differences in Nutrient Metabolism In addition to the global differences in hunger/appetite and energy expenditure, women's metabolism of the different macronutrients (protein, carbohydrates, fats and alcohol) differs from men's to one degree or another and this is true both after a meal is eaten, between meals and both during and after exercise. Differences in the use of these different nutrients are related to body composition but in other cases it's clear that they represent a true physiological difference. Given the differences in body composition and fat patterning, it would be surprising if they were not the case. At the same time, many early (or logical) assumptions about nutrient metabolism are turning out to be incorrect with some surprising things occurring. As I will be focusing predominantly on carbohydrate and fat metabolism in the next sections, I want to address both protein and alcohol here first. Both at rest and during aerobic exercise, women use less protein for fuel and this is clearly due to the effects of estrogen (21). Quite in fact, if men are injected with estrogen, their metabolism changes to mimic that of a woman's. Practically this means that a woman's protein requirements is relative to her LBM will be lower than a man's. This does change slightly from the follicular to the luteal phase when increased progesterone increases protein breakdown but this effect is extremely small and can be safely ignored (21a). There is limited research on the impact of hormonal modifiers here although any loss of estrogen signaling (i.e. amenorrhea, menopause without HRT) has the potential to negatively impact on protein metabolism. Aging also plays a role here and protein requirements will go up with age, possibly increased significantly in older individuals. 93 Women's bodies also metabolize alcohol differently than men's. While some of this is related to differences in total body water to dilute the alcohol but women also have lower levels of the liver enzymes that metabolize alcohol (22). While I won't discuss alcohol much in the context of overall diet in this book, there is an interesting gender difference worth mentioning in general, alcohol intake doesn't seem to generate the weight or fat gain that would be expected based on the calorie values and some calories seem to be "missing". There is also a gender difference here where women show a lower Body Mass Index (BMI) with increasing alcohol intake while men show an increasing BMI (23). While the mechanism isn't clear, alcohol intake leads to lower body weights in women but higher body weights in men. This appears to be mostly a social phenomenon since women frequently drink instead of eating while men commonly drink in addition to eating high-calorie, high fat foods. Type of alcohol also plays a role with wine being associated with a lower BMI and hard liquor associated with a higher BMI. This may be interacting with gender differences in alcohol preference that may be present. I mention this only for completeness and am in no way recommending women attempt to drink themselves thin (BMI is also not a perfect indicator of BF%). Nutrient Metabolism After a Meal First let me look at differences in how nutrients, focusing on carbohydrate and fat, are metabolized following a meal. I'll be assuming a mixed meal containing some combination of protein, carbohydrates and fats. These enter the stomach where they are digested and absorbed, eventually reaching the bloodstream. This puts the body into a storage mode where the nutrients are either burned for energy or stored for later use. Carbohydrate is stored in muscle or liver as glycogen while dietary fat can be stored within muscle as Intra-Muscular Triglyceride (IMTG) or within fat cells. After a meal, women burn roughly 14% less fat than men for energy, their bodies using more carbohydrate. I'd note that as will generally be eating less total fat per meal than a man, the total amount of fat stored may be less in absolute terms even if the percentage is higher. A large part of this is due to the greater ability of women to store fat. Women have roughly 12% more total body fat than men, they store just over 12% more of the total fat eaten (with no apparent difference between the follicular and luteal phase). An additional factor is that women's subcutaneous fat is more sensitive to the anti-fat mobilizing effects of insulin; the hormonal response to eating shifts off fat mobilization. In contrast, men's visceral fat is more resistant to insulin and continues releasing fatty acids into the bloodstream after a meal, causing more to be used for energy. Any fat not burned for energy is stored primarily in the subcutaneous fat with women tending to store it in the lower body fat (at least relatively) with men tending to store the fat in their upper body. Given the difference in body fat distribution, readers might assume (and researchers did the same) that women's bodies would preferentially store fat in the lower body while men did so in the upper body. But that turns out to be wrong. Rather, after an average sized meal, both women store roughly twice as much fat from the meal in the abdominal/upper body area as in the lower body. Women also store double the dietary fat in both areas as men (who store relatively more dietary fat in visceral fat). This does depend somewhat on a woman's body fat patterning (as occurs in obesity or with elevated testosterone levels) tend to store relatively more fat in the upper body while women with a lower body fat pattern store relatively more in their legs. There is one exception to the above. When women eat a very high-calorie and high-fat meal (in this case 100 grams of fat), this excess calories will be stored directly in lower body fat (24). An old saying, at least in the US is "A moment on the lips, a lifetime on the hips." and the combination of this type of direct lower body fat storage along with a general resistance to lose lower body fat, this is basically true. Irrespective of that one exception, the post-meal fat storage is in the upper body fat areas in both women and men. Given the difference in lower body fat patterning, this came as a bit of a shock to the researchers. Stranger still, research found that the post-meal fat storage patterns did not predict where fat was gained in the long term (25). Despite storing fat in their abdominal area immediately after a meal, women still tend to gain fat in the lower body with time. This fact, among others, was confusing the researchers. The answer, as detailed below, has to do with what happens between meals. Nutrient Metabolism Between Meals Several hours after eating a meal, the nutrients that were eaten will all have been burned for energy or stored and the body will begin to rely more on stored calories such as muscle and liver glycogen, IMTG or fat. The same occurs after an overnight fast to a more pronounced degree. This effect is hormonally driven. As storage hormones such as insulin drop and nutrient mobilizing hormones increase, fatty acids will be released from fat cells to be used for energy. The liver will also break down stored glycogen to glucose (or produce it from other substances in the body) which is released into the bloodstream to maintain blood sugar. Of the fat being released into the bloodstream, 80% comes from upper body fat cells and the remaining 20% from lower body fat cells and this occurs in both women and men. This provides the reason that both women and men store fat in the upper body after meal: these fatty acids can be used for energy more quickly between meals. If women did not store fat in the upper body right after a meal, there might be not enough fatty acids available to provide energy to their bodies. And while women do store fat in the lower body, their lower body fat patterning. If that is why the lower body fat patterning is stored then... Of the fat that is in the upper body and not mobilized and burned, what happens is that over the next several hours, some of the fat stored in the upper body is redistributed from the upper body to the lower body fat in women or how does it eventually "end up" in the lower body? Surprisingly, women show up to 40% higher rates of fat mobilization than men although this is primarily coming from the upper body. They also show 15% higher levels of fatty acids in the bloodstream. Given the general pattern of having higher body fat overall and a greater difficulty losing fat, this makes very little sense as women would be expected to mobilize less fat, not more. And this has been more confusing when two more contradictions are considered. The first is that high blood fatty acids tend to cause insulin resistance which can lead to diabetes or the Metabolic Syndrome. Yet, as I mentioned above, pre-menopausal women are generally protected from this disease. Women have higher levels of fatty acids in the bloodstream without the negative effects that would normally occur. The second is that, in general, the more that a given nutrient or fuel is available, the more it tends to be used for fuel. When carbohydrates are available, the body burns more carbohydrates and when fat is available, it burns more fat. Women have 15% higher levels of fatty acids yet they burn less fat compared to men. Given that women have higher levels of fatty acids in their bloodstream between meals, we would expect them to burn more fat overall. One reason for this is that women in the fed state obtained a greater percentage of their fat burn from IMTG than men. Upon closer analysis, women were found to burn more fat than men. Women also have... Upon more examination, it seems that the fatty acids mobilized by women are being preferentially stored in the lower body fat which is where IMTG... However this is primarily related to differences in body composition. Since women have a lower total energy expenditure, women have higher levels of fatty acids in the bloodstream, in contrast, as a percentage of total energy burned, women burn more fat than men as a percentage of total energy burned. So while women do burn more fat, they burn less fat in absolute terms. Given the higher levels of blood fatty acids which isn't matched by either the negative health effects of insulin resistance or the use of fat for fuel that would be expected. If fatty acids are being released but not burned for energy, this means that they must have some other fate which they do. This is called non-oxidative disposal which just means that the fatty acids are disposed of somewhere in the body without being burned (oxidized). And this happens to a greater degree in women (27). Recall from earlier that women store more IMTG than men and much of the fatty acids being released into the bloodstream are just being stored back within a fat cell. While being stored in this type of a futile cycle (which is costly), women are still getting the benefit of being able to shift to using fat for fuel more readily (as during exercise, discussed below). But there is no law that says that fatty acids released from one fat cell have to be re-esterified into the same fat cell. If blood flow is sufficient, those mobilized fatty acids can be transported elsewhere in the body. While they may be used for energy, they can just as easily be stored in a different tissue. This could include the situation where they are stored within muscle as IMTG. As I've mentioned, in cases of extreme obesity, fat is stored in inappropriate tissues such as the liver or pancreas. Of relevance to the topic of fat patterning, these fatty acids can also be stored in lower body fat. This may be due to a direct (fatty acid storage pathway) I mentioned in a previous chapter, a pathway by which fatty acids released from one fat cell can be stored/re-esterified in a different fat cell. Which is exactly what is going on here. Fatty acids which have been released from the upper body fat cells, but which are not burned for energy, can eventually be stored in lower body fat cells. This occurs more in lean women than obese women, and to the greatest degree in obese women. The direct fatty acid uptake pathway is the reason that women carry more fat in their lower body and it explains the observations above. In this way, fatty acids being mobilized from upper body fat which aren't burned for energy can be redistributed to the lower body between meals and effectively "moved" there. While this is a small effect, it adds up over time. That said, it is nowhere near as drastic in this... this final piece of the puzzle explains all of the above observations. After a meal, women not only store more total dietary fat but that fat is predominantly stored in upper body fat cells. After a meal, women show higher rates of fatty acid release (especially from the upper body) but burn less of it for fuel due to a lower energy expenditure and having less LBM. Those unburned fatty acids are either stored back in the fat cells they came from or are stored elsewhere in the body, including lower body fat cells. In this long-term redistribution of fat from the upper body to the lower body that explains both the development and maintenance of women's lower body fat patterns. The fact that primarily occurs in the case where body fat is being gained as, during weight maintenance, any fat stored would be burned off for energy before being redistributed to other areas (9). Nutrient Metabolism During Exercise Continuing from the above, I want to look at gender differences in nutrient metabolism during exercise and to also explain some of the differences seen in fat loss and fat partitioning. While I will only be focusing on two types of exercise in Chapter 4, I will only be focusing on three types in the following sections. The first is aerobic exercise, any continuous whole-body movement done for 20 minutes or more. The second is High Intensity Interval Training (HIIT) which alternates short bouts (15-90 seconds) of very high-intensity activity with similar durations of low-intensity activity. Finally is weight or resistance training. As the differences in nutrient use and metabolism during exercise is in the following sections focused on aerobic exercise although I will address the limited work on other types of exercise (especially as HIIT and weight training have some potentially enormous benefits or women's fat loss). Fuel Use During and After Exercise Perhaps surprisingly, women actually use a larger percentage of fat for fuel during low- and moderate intensity aerobic exercise than men. This effect is even more pronounced during the luteal phase when insulin sensitivity is elevated due to elevated progesterone limits carbohydrate use further. When exercise starts, women also show higher rates of fat mobilization from upper body fat although any fat mobilized is burned for energy. At higher exercise intensities, women and men use roughly the same amount of fat and carbohydrate although there are still some small differences present. I say surprisingly as this would seem at first glance to contradict women's generally lower rates of fat loss. But there are several factors at work here to explain this. The first is that, except for highly trained athletes, women tend to burn fewer calories during exercise. Even if a higher percentage of fat is used, the total amount may not be greater. For example, consider a woman and man doing an aerobic workout. The woman burns 300 calories while the man burns 400 calories, with both using fat for fuel. Although women show a higher percentage of fat use, the total amount of fat burned is similar or lower in women. A woman with a lower overall level of fat use during exercise typically makes up a very small part of the overall day's energy expenditure. Unless extremely large amounts of activity, hours per day, are being done, the total number of calories and fat burned during exercise will be small. Consider an example: an intensive study, women and men performed 90 minutes of exercise at a fairly hard intensity (75% of maximum heart rate). The women burned 660 calories while the men burned 985 calories (33% more). Of those 660 calories, women burned 36 grams of fat (less than one tenth of a pound) while the men burned 45 grams of fat (28). Since one pound of body fat contains 454 grams of fat, it would take women nearly 13 workouts to lose one pound while men would require only 10. This assumes that 100% of the fat being burned was coming from body fat in the first place which turns out not to be the case. I mentioned earlier in the book that women's muscles store more fat within their muscle (called IMTG) and it turns out that women's bodies use more IMTG for fuel than men during aerobic exercise (29). This reduces the total amount of body fat burned during exercise and, when all is said and done, of the total fat burned during exercise, only 12% of it comes from fatty acids mobilized from fat cells. Of the 36 grams of fat used for fuel during exercise, only 4 of that would be coming directly from fat cells and most of that would be coming from upper body fat cells to begin with. I'd note that the depleted IMTG will eventually be replaced and this can technically come from either body fat or dietary fat. In a dietary deficit, where there is a fat burning and this effect is seen in the hours after exercise, the body should take fat from fat cells and transfer it to the muscle as IMTG. Even this will be coming primarily from upper body fat meaning that a woman's lower body fat is being hit. I want to finish by mentioning an absolute inverse (post) effect where women's bodies take up stored fat into muscle. Regardless of the IMTG use during exercise it may be that women actually store a bit more fat when they exercise. This is through a pathway that allows IMTG to be replaced from fat cells that are actively storing fat. However, the effect is insignificant when compared to the fat use. As noted, women tend to burn more carbohydrates after exercise than during exercise (30). This is an important thing to note: the additional fat burned after exercise is relatively small, women burn more fat after exercise since men burn it after. This is also important in that, while the fat lost during exercise is similar between women and men, the subsequent fat loss during the day after exercise will differ. Finally, there has been limited research on this. But there is some evidence that men will burn more fat than women after exercise and that it may be related to higher intensities. When exercise intensity is very high, men have been found to increase their fat use following exercise compared to women (31). The last aspect that I want to address is the impact of the different types of exercise on fat loss and specifically their effect on stubborn fat. Following exercise, and especially in the morning or afternoon, the lipolysis rate at rest during the day can be increased for hours. In obese women, a large amount of aerobic exercise actually causes an increase in lower body fat loss. Where women with small BF% tend to see the greatest differences in lower body fat loss with exercise. Even if this effect is relatively small, it can add up over time. So with any exercise, you may want to include at least some exercise at a higher intensity as well, it seems to have a small benefit in lower body fat loss. Effects of Hormonal Modifiers Looking at hormonal modifiers, in women with PCOS, fat mobilization is decreased significantly at rest which may be part of their predisposition to weight gain. Weight loss in general along with aerobic exercise can overcome this, possibly through increased sensitivity to the hormone ANP (discussed previously (36,37). It is unclear whether women with subclinical hyperandrogenism experience the same. Through a variety of mechanisms, all of which contribute to a decreased ability to mobilize and burn fat, obese women have a much more difficult time losing fat. In menopausal women without HRT, fat mobilization is decreased relative to fat use and there is a shift toward storing more fat in the visceral area. After menopause, some aspects of fat mobilization and use become more male-like, suggesting that higher estrogen levels are needed for the benefits in terms of fat mobilization, although this is reversed with HRT. Exercise reverses this (39). At menopause, there is a similar decrease in fat mobilization (reversible with caffeine) and fat burning during exercise although hormone replacement therapy (HRT) as well as exercise can reverse this (40). Composition of Weight Being Lost In Chapter 7, I talked about altering body composition and how the proportion of fat, muscle, water and glycogen being lost or gained can change. Focusing only on the proportion of fat and LBM (some of which is muscle) being lost, women tend to lose a lower proportion of LBM than men (this can be due to higher initial fat levels). Even so, this difference in proportion tends to be relatively small. Where women with the same BF% lose less LBM, this difference in LBM will make a difference in fat loss in the long run. This means that a woman will lose more fat for a given deficit than a man in terms of percentage. But as they also have less LBM to lose in the first place, and they also lose less total weight, women lose fat more slowly in absolute terms. Finally is the topic of weight loss impact on fat distribution. What is clear is that women have an easier time losing upper body fat and, if they do lose weight, they will eventually lose fat from the lower body although with greater difficulty. Fat Loss and Fat Regain Interestingly, there are some studies suggesting that when women lose weight and regain fat, the fat may be regained differently. It's possible that a woman who loses fat from the easier to mobilize upper body fat but regains it in the more difficult to lose lower body fat. Even if she ends up at her pre-diet BF%, she might have more trouble losing fat during later diets. I described a study earlier in the book where Category 2 women and men were overfed, gaining 6.5 lbs of fat over 8 weeks. Of that, 4 lbs of fat was gained in the upper body with the fat cells increasing in size, with the other 2.5 lbs being gained in the lower body and fat cell number increasing (over time, the gained upper body fat would be redistributed to the lower body). During a second 8 week time span, they were put on a diet and lost 6 lbs of fat with 5 lbs coming from the upper body and 1 coming from the lower body (43). Presumably if they had kept dieting they would have lost the remainder of lower body fat but this points out that, in both women and men, upper body fat is both easier to gain and lose overall. In a related vein, Category 2 women performed 6 months of combined aerobic exercise and weight training; they lose 3% body fat with no fat loss from the legs (44). This is problematic as several studies suggest that when and if fat is regained, fat distribution can and does change. In one, women who regained weight after a diet were found to increase their thigh body fat levels by 102% (2% over where it started) despite only regaining 83% of the lost weight (45). Had they regained all of the lost weight, presumably this would have gone even higher. Another found that despite a 93% total fat regain after dieting, women increased their hip fat by 6% over where it had started (46). While not measured, it's conceivable that lower body fat cell number also increased in both cases. Summing Up Putting the above data together leads to the following overall picture. When women gain fat, it tends to be in both the upper and lower body with the gained upper body fat being redistributed to the lower body over longer time frames. When they lose fat, proportionally more of that fat will come from the upper body as it is easier to mobilize (as discussed in Chapter 7). When fat is regained, there may be a proportionally greater increase in her lower body fat with levels increasing above their pre-diet levels. Over several cycles of Yo-Yo dieting that alone has the potential to lead to proportionally greater lower body fat levels. If a woman diets poorly (insufficient protein, no resistance training, rigid, she may lose LBM along with the fat (coming from the upper body with the LBM representing muscle). Her diet become unsustainable and she begins overeating in the face of lowered energy expenditure. As she regains body fat, she will gain proportionally more in the lower body (potentially increasing fat cell number) and this will be more true if she is eating a large amount of high-calorie, high fat foods which are preferentially stored there. Since she will not be regaining the lost LBM, her hunger will remain higher than it would otherwise and she may end up with not only a higher BF% but proportionally more hip and thigh fat. This situation potentially worsens with each cycle of fat loss and regain. This all points not only to the importance of using proper dieting practices but is critically important for those women attempting to diet to a very low BF% and who typically do lose their lower body fat in doing so. When they regain fat (as they eventually must) they must ensure that there is no body fat overshoot or proportional increase in the amount of lower body fat present. Because while the above is mostly in Cat 2/3 women, there are at least anecdotal reports of increased lower body fat in Cat 1 women after a diet, especially if the post-diet phase includes bingeing on high-calorie, high-fat foods. 98 Chapter 11: Women, Fat Gain and Fat Loss: Part 2 Continuing from the discussion of physiological differences between women and men in the last chapter, I now want to look at the direct research that has been done in terms of fat gain and fat loss. It's generally accepted that women lose less fat (or lose it more slowly) than men in response to diet or exercise and there is certainly some truth to the idea. At the same time, there are a number of pitfalls to this type of research that need to be addressed and that I want to discuss. Following those comments, I want to first look at research that has compared women and men in terms of their response to specific weight/fat loss interventions. As most research is done on overweight individuals, I will address potential differences between lean and obese women. I'll finish by looking at gender differences in energy homeostasis (how the body regulates energy balance) along with a brief look at the evolutionary reasons those differences exist. Direct Research on Weight/Fat Loss: Introduction When I discussed some of the difficulties in doing comparative gender research in Chapter 1, I touched briefly on the issue of how women and men should be matched in the first place. That is, within the context of this chapter, how should diet studies be set up or how should the results be compared. Should the women and men weigh the same (in which case a woman's BF% will be much higher), should they have the same BF% (in which case the men's weight will be much higher), or should they be at the same BMI (in which case weight and BF% will differ)? If you do match them in some way, the question remains as to whether or not that will reflect the real world (where weight, BMI and BF% may all differ). Once that has been addressed the next question is how should the results be compared. There are two primary ways of doing this: you could compare the total amount of weight or fat lost in absolute terms (i.e. women and men lost 4lbs and 5 lbs respectively) or you could compare the percentage of weight or fat lost relative to their starting point (i.e. both women and men lost 5% of their starting weight). The second approach is probably the more valid one as a relatively lighter woman would be expected to lose less total weight than a man regardless of any other differences. So consider a woman who weighs 250 lbs and loses 12.5 lbs and a man who weighs 300 lbs and loses 15 lbs, both losing 5%. Her absolute loss was smaller but, once initial body composition was taken into account, her relative loss was identical. Even if most studies use it, weight loss per se isn't the ideal goal or even the best result to look at in the first place. Changes in body composition are far more relevant and, as I've discussed, the relative changes there impact on both total weight loss and the rate at which it occurs. As discussed, women lose less LBM and more fat than men under most conditions which means less total weight loss (but often more total fat loss) along with that weight loss being slower. In many exercise studies, women often gain some LBM (due to starting out with less than men) even if they lose similar amounts of fat. They may lose less total weight but their fat loss is identical and their body composition has improved. Finally is the issue of individual variability. Most studies only report the average result but, just as it did with the issue of birth control and weight gain, this can mask the results that any given individual might experience. There is actually some evidence that women are more variable than men in their response to diet or exercise interventions and this acts to cancel out some of the changes that would otherwise be seen. Finally there is the fact that most research on weight and fat loss is done in Category 3 individuals for what should be logical reasons. While they certainly experience metabolic adaptation, it becomes more pronounced into Category 2 and Category 1 and the results between women and men may differ. With those potential pitfalls addressed, I want to look at the direct research that has compared women and men in terms of their response to various weight loss interventions. I'll separate these into two sections, the first will include diet only and diet plus exercise, while the second will focus on exercise only studies. I'd note only for completeness that there is some indication that women may do better than men when they include weight loss medications in the program but this is beyond the scope of this book. Diet Alone or Diet Plus Exercise (1) I want to start by addressing studies looking at diet alone or diet plus exercise. These tend to vary enormously in how they are set up and might compare low- to high-protein, low- to high-carbohydrate, low- to high-fat or look at the Mediterranean diet or any other number of possible combinations (I will provide my recommendations in Chapter 19). If exercise is included, it may be low-intensity aerobic exercise, high-intensity aerobic exercise, resistance training or a combination of one or more (there might also be diet plus aerobic exercise compared to diet plus resistance training). This can make comparing the results of different studies very difficult. 99 Variations in the calorie intake or the side of the deficit relative to maintenance may also differ. This is important as it ties into the baseline differences in energy expenditure that are present. If both women and men are placed on some fixed calorie level such as 1200 or 1500 calories per day, the woman will have a smaller daily deficit since her total energy expenditure is lower to start with. If calories are reduced by some percentage below maintenance, the same will occur. If a woman and man with maintenance levels of 1800 and 2700 calories respectively reduce that by 25%, her deficit will be 450 cal/day while his will be 675 cal/day. Of course she will lose less weight (and lose it slower) than him. The only time that you'd expect a woman to lose the same amount as a man would be if both created an identical daily deficit. If both women and men reduced their calorie intake by 500 calories/day or increased their exercise by 500 calories/day this would be expected to generate the same results. This raises the practical issue where it may be impossible, or at least very difficult, for women to be put on the same intervention as men. With all of that said, in the aggregate, the research shows that women do in fact lose less total weight than men in response to various types of interventions. The difference actually isn't enormous with women losing roughly 12.5 lbs (5.5 kg) and men 18 lbs (8.5 kg), a mere 4.5 pound (2kg) difference. But this would be expected for all of the reasons discussed above. Looking at the results in terms of the percentage loss, the difference is much smaller with women losing 7% of their starting weight and men 8%, a mere 1% difference. I would mention in this regard that some studies find that women show a slower initial loss of weight than men which could be related to men having more of the easy to lose visceral fat. Since they are smaller, women might also be losing less body water and glycogen. Finally, since women are losing less LBM than men, their total and rate of weight loss will be slower (it's hard to see men's greater loss of LBM as a benefit). Perhaps surprisingly, women appear to show slightly better long-term weight maintenance than men and this may actually be due to a slower initial rate of weight loss (2). Most studies find that maximum weight loss occurs at about the 6 month mark (supporting all of the research from the last chapter on how the body starts fighting back) but women may continue to lose past that point. Their slower initial losses may be offset by more continued losses over the long-term. While exercise alone has not been shown to be terribly effective for weight loss (this is discussed below), its inclusion in a weight loss program has been shown to provide benefits. Of perhaps the greatest importance is that diet alone can cause a loss of bone alone studies but this is where only looking at changes in body weight becomes misleading. If LBM loss is reduced or LBM is gained due to exercise, total weight loss will be reduced even if fat loss is not. When the above results are expressed as a percentage, women seem to show slightly worse results with women losing 3.6% of their starting weight and men 5.2% (a 1.6% difference vs. 1% for diet alone). This is assuredly a consequence of the impact of exercise on LBM. Since women start out with less LBM than men, they gain it more easily with exercise. They may lose less weight but lose as much fat while improving body composition. Exercise and Weight and Fat Loss While it is slightly out of order to address exercise in the absence of any dietary control or modification, I have done so for two reasons. The first is that exercise alone is often recommended as being nearly magical for both weight and fat loss, even in the absence of dietary control. The reality here is far different with exercise being generally ineffective in terms of its effect on weight or fat loss for both women and men. There are a number of reasons for this but a main one is that the amount of exercise that all but highly trained athletes can realistically do is usually fairly small. This is double true for women due to their being lighter and smaller. The calorie burn from exercise is simply too small to matter. More important to the topic of this chapter is that the impact of exercise on weight/fat loss is the place where the largest apparent gender differences in terms of total losses or the adaptations and compensations that occur to limit losses show up. 100 Looking at the topic observationally, early research showed no relationship between a woman's activity levels and her BF% while men who were more active had a lower BF%. Similarly, when women increase NEAT, they show no loss of fat, presumably due to increasing their appetite while men's BF% does decrease (4). Perhaps shockingly, in response to 5 months of half-marathon training, women showed no overall increase in their Total Daily Energy Expenditure probably due to decreasing NEAT while men's TDEE did increase (5). In contrast to the above, and supporting the importance of weight training, a recent study found a moderate relationship between the weight training frequency and intensity and women's BF% (5a). Specifically, for every day of added exercise, BF% was reduced by about 1.5% and LBM increased by 1.5 lbs. The overall difference was fairly small, however with non-exercisers having a BF% of 32.3% vs. 30% in the weight training group. Moving on to more direct research, a number of early studies that I will not detail generally observed that women did not lose significant amounts of weight with exercise alone (almost always aerobic exercise) while men did. This led to the general conclusion that exercise without diet ineffective for women (6). Indirectly supporting this was research showing that women's fat cells did not increase their response to fat mobilizing hormones in response to 20 weeks of training while men did (7). This is probably an artifact of women already mobilizing more fat than men which means that they have less improvement to make. Similar results have been seen for the impact of exercise on insulin sensitivity where women show no improvement from exercise while men do. But this is simply due to women being more insulin sensitive to begin with and having less room for improvement. Women also show a large amount of variability in weight loss or gain both in general and compared to men in terms of their response to exercise (8,8a). Some women have been found to be weight-loss "responders", losing as much weight as predicted (and showing less of a reduction in NEAT) while others are "non-responders", losing less weight than predicted due to greater reductions in NEAT (9). There is likely to be variability in the changes in hunger, appetite or the enjoyment of palatable food between any two women. It's also likely that women who show the greatest reduction in NEAT show the greatest increase in hunger, appetite, etc. As I noted earlier in the chapter, this variation can make reporting of average results a problem. In one study on exercise and weight loss, women showed about an equal distribution of weight loss and weight gain while all the men lost at least some weight during the study. This made the average weight loss appear much smaller for the women. As I mentioned above, exercise has generally been found to be ineffective for weight and fat loss due, among other reasons, to the relatively low numbers of calories that can be burned. Even studies using larger amounts of exercise,generating significant amounts of weight loss in men, usually fail to have the same effect in women. At best women avoid weight gain or gain a small amount of weight (10). Coupled with a woman's smaller body size and muscle mass along with a greater tendency to decrease NEAT or increase food intake, exercise alone is simply that much less effective for woman than men (11). While the above conclusion, that exercise alone is ineffective for causing weight loss in women, seems well supported, there are some problems with it, some of which I already mentioned. One is the reporting of average results, mentioned above. Another is that most exercise studies generate very little weight loss in in women or men. Finally is the fact that changes in body weight can mask changes in body composition. One commonly cited study on this topic demonstrates this well. In it, women and men were trained over 40 weeks to complete a half-marathon and the study concluded that men but not women lost weight but this is misleading (12). On average, women lost just under 2 lbs while men lost just over 2 lbs. The women's loss wasn't statistically significant while the men's was but neither group lost very much in the first place. The body composition changes also paint a very different picture. Here the women lost 5.5 lbs (2.5kg) of fat while gaining 2.6 lbs (1.2 kg) of LBM while the men lost 8 lbs (3.5 kg) of fat and gained 3.5 lbs (1.5 kg) of LBM. The fat loss represented 3% of the women's starting body fat and 4% of the men's. Certainly the women lost less total fat (as would be expected) and a smaller percentage (similar to the diet and diet plus exercise studies) but the increase in LBM in both groups made the actual change look far smaller than it was. Focusing on the small change in weight loss per se leads not only to a poor conclusion but misses the point about the much more important changes in body composition. Although NEAT was not measured, the women's food intake did tend to increase about halfway through the training program while the men's didn't, once again suggesting a difference in how women and men regulate energy balance. Even more importantly than the above is the fact that not all studies find that women lose less weight (or fat) than men. A recent analysis of short-, medium- and long-term studies finds in some studies women lose less weight/fat than men but in others they do not (13). It also pointed out a number of potential factors that might cause the results to be so mixed. A major one has to do with the generally small amount 101 of total weight loss that is seen in the first place, often no more than a few pounds. In many cases, the measured changes are well within the normal day-to-day variation in body weight and that alone could obscure the true results. Many earlier studies also failed to control for the women's menstrual cycle and that alone could easily explain the differences. As mentioned, in several studies women gained LBM while men did not meaning that their total weight (but not necessarily fat) loss will be lower. But there are two much larger issues at work here. The first has to do with adherence and whether or not the exercise program is being followed in the first place. In studies where the exercise program is supervised by the researchers, weight loss is always greater than when it isn't. Quite simply, many people, regardless of gender, show poor adherence to the exercise program. The second, and far more important issue has to do with an issue I've mentioned several times which is that women will always burn fewer calories than men for any fixed amount of exercise. Studies that give exercise based on frequency, time and intensity (i.e. 5 days/week for 45 minutes at 70% of maximum heart rate) will always generate a lower calorie expenditure in women than in men and that alone would predict lower amounts of weight and fat loss. In fact, this may be the primary factor in the differences that have been seen to date (14). This raises the question of whether or not women and men will lose the same amount of weight and fat if they burn the same number of calories during exercise. In one study of Category 3 women and men, this appears to be the case (15). In it, the subjects performed supervised exercise five days per week working up from 150 calories burned per workout to either 400 or 600 calories burned per workout over the first five months of training. They maintained that level for an additional five months. In the 400 calorie/day exercise group, there was no difference between women and men in either weight or fat loss: the women lost 8.4 lbs (3.8 kg) and the men 9 lbs (4kg) of weight and almost 100% of it was body fat. In the 600 calorie per day exercise group, the results were slightly different. Here the women only lost 8.9 lbs (4 kg) or 0.5 lbs (0.2 kg) more than in the 400 cal condition while the men lost 13.5 lbs (6kg) or 4.5 lbs (2.2 kg) more. For statistical reasons the results were considered identical but suggests a trend where performing more exercise was less effective for women. It's impossible to tell if this is just a statistical blip or if the women's bodies started to compensate at the higher energy expenditures. Regardless, this study points out that if women and men burn the same number of calories in exercise, their fat loss is effectively identical. It also suggests that the results of previous studies had less to do with actual gender differences and more to do with women simply being smaller and burning fewer calories overall. I'd note that this study did use a fairly large amount of fairly intense exercise although it was built up to gradually over many months (this is important for reasons I'll discuss in later chapters). But there is another point to consider which is that, in burning the same number of calories during exercise, the women had to exercise longer than the men (50 vs. 32 minutes in the 400 calorie/day group and over an hour vs. 42 minutes in the 600 calorie group). Other research supports this observation. In one, women and men who burned the same number of calories with supervised exercise lost identical amounts of fat, but the women had to exercise for 54 minutes to the men's 43 minutes (16). Indirectly supporting this, when women burn the same 400 calories as men during low- or high-intensity exercise, they burn more fat both during and after exercise (relative to their LBM) than men (17). This effect is especially pronounced after highintensity exercise but means that the apparent gender differences in fuel use after exercise may simply be due to burning fewer calories. As with the other studies, the women had to exercise significantly longer to achieve this (120 vs. 89 minutes for low intensity exercise and 86 vs. 50 minutes for high-intensity). These studies make two primary points. The first is that, at least in overweight women, a major cause of the apparent gender difference in weight loss is that women simply burn fewer calories for any given amount of exercise. When they burn the same number of calories as men, their fat losses become essentially identical with the drawback being that they have to exercise for anywhere from 20-35% longer than men. Presumably this could be partially offset by women exercising at a higher intensity (and I will discuss other potential benefits of including high-intensity training for women in Chapter 14) but that brings up other potential issues having to do with excessive amounts of intensive exercise overstressing women's bodies. This will be discussed in detail in the next two chapters. The same general conclusion will hold for diet in that women will more or less lose the same amount of fat as men if the same deficit is achieved. But, practically it is more difficult for them to do so. Lean Versus Obese Women The above issues, whereby it is more difficult for women to achieve the same calorie deficit with diet or calorie burn with exercise becomes even more pronounced as women are lighter and leaner. Not only is it more difficult for the woman to even achieve the same degree of calorie reduction or exercise energy 102 expenditure as men as they become lighter, but many of the adaptations and compensations start to become more pronounced in leaner women. While studies done in obese individuals may not find significant differences in terms of weight and fat loss (assuming a few conditions are met), this stops being the case as women enter Category 1 or try to reach lower limits of female BF%. It not only becomes effectively impossible for a woman to achieve the same daily calorie reduction or exercise energy expenditure as men but, even when they can, their results still come more slowly. This difference between categories makes perfect sense within both the context of the general adaptations to dieting along with the specific issues that pertain to women (discussed at the end of this chapter). Fundamentally a Category 3 woman is not at risk of starving to death as she has plenty of fat to lose in the first place. Even here, there may be differences based on body fat patterning. Women with a male-like upper body fat pattern lose fat from exercise alone while women with a lower-body fat pattern do not (18). Perhaps bizarrely, while upper body obese women and men lost fat in response to exercise, lower body fat women actually gained fat (19). This suggests that the effect is as much due to total body fat/Category as it is to fat distribution and patterning (recall from the previous chapter that 6 months of exercise training caused fat loss in the arms but not legs). The Category 3 woman with the upper body fat pattern has easily lost visceral fat (like men) along with relatively easier to lose upper body fat; the woman with a lower body fat pattern does not. This is important to this discussion due to the fact that women with a typical body fat patterning tend to lean out effectively from the top-down. Their upper bodies, face, breasts, shoulders and even abdominal area (in the mid to high teens of body fat) will become lean while their lower body fat may not have changed significantly. The greater the relative proportion of the difficult to mobilize lower body fat, the less of an effect any intervention will tend to have. Studies find that while exercise generates at least some fat loss in both obese women and men, it has no effect in lean women (20). In one study, three months of exercise caused fat loss in obese women and men but didn't generate fat loss in women who had been matched for body fat to the men (21). An interesting observation in this study was that men with smaller but more fat cells lost no fat while men with larger but fewer fat cells did (recall that fat cell size is a determine of how easily fat can be mobilized and lost). As women's lower body fat is typically made up of smaller but more fat cells, this probably explains the lack of exercise alone. Other factors could be involved here. One is the potential for food intake to increase after exercise, which has been shown to occur to a greater degree in lean than obese women. When exercise alone is used, lean women will exactly increase their food intake to cancel out the calories burned from exercise while obese women will not (22-23a). Lean women also report an increase in the sensory attractiveness of food (i.e. food becomes more appealing) after exercise that men do not (24). This seems to contradict what I wrote in an earlier chapter about exercise not increasing appetite or hunger. While true in the short-term, it's clear that women do in fact increase their calorie intake in response to exercise over the long-term (25). This occurred in the half-marathon training study I mentioned above but ultimately means that, in lean women, exercise alone is unlikely to be effective for generating fat loss if the diet is not controlled. The lean woman's body will simply adapt and cancel any effect of the exercise. I would note again that, in highly trained female athletes, the above may reverse itself and hunger may be blunted in response to exercise (26,27). This can cause a different problem where female athletes are chronically undereating. Regardless of that, the fact is that all of the adaptations that are occurring in the leaner female dieter will act to slow fat loss, frequently to rates lower than that of men. While little research has been done on this (researchers don't often do diet studies in lean individuals), anecdotally, Category 1 women always lose fat more slowly as they approach the lower limits of BF% than men, even if they are on broadly similar exercise programs and calorie deficits. This could be due to greater reductions in NEAT or some other component of energy expenditure but occurs nonetheless. For reasons I will discuss next, the adaptations are always more pronounced in leaner women (and often moreso than in leaner men). Gender Differences in Energy Homeostasis (28-30) As readers may remember from science class, the concept of homeostasis is that the body will, to one degree or another, attempt to maintain some relatively fixed or stable level. Body temperature is one where the body will shiver if someone becomes to cold or sweat if they become too hot. The bodyweight or body fat set point is another with the adaptations in the different components of energy balance at least attempting to maintain homeostasis. And while much of the difference in weight or fat loss can be explained by differences in initial body composition, it's still clear that women's bodies, to one degree or another, regulate energy balance and homeostasis differently than men. Their adaptations are, in many 103 cases larger along with being more variable. They return to baseline faster after high-intensity exercise and there are other examples of this. One has to do with how well or poorly women handle heat or cold stress compared to men. Under some conditions, women handle heat better during exercise (although they cool down more slowly afterwards) and start shivering later (saving energy expenditure) in response to cold. These do point to physiological differences between the and all ultimately point to women's bodies being more efficient at storing calories when they are available while limiting their use for fuel when they are not. While many of those differences appear to be less important in overweight women, larger effects start to become far more prevalent as women become leaner. Given what the system is "trying" (in a biological sense) to accomplish, this all makes sense. Before addressing that, I first want to look at what end up being the physiological controllers of the differences in energy homeostasis. As I've focused on some of these already, I'll be focusing here on insulin, leptin and estrogen. Both leptin and insulin play numerous roles in the body but, specific to energy homeostasis, they are both related to levels of body fat and food intake. Leptin is related mostly to subcutaneous fat and changes very slowly over time (i.e. hours or days) while insulin is related to levels of visceral fat and changes relatively quickly (i.e. minutes). Given women's greater amount of subcutaneous fat you would expect it to be a more important signal compared to men for whom insulin might more important given their larger amount of visceral fat. In animal models this is true and one study has shown that men reduce their appetite and lose weight in response to inhalable insulin while women do not (31). Since leptin appears to be the more important signal for women, I want to look more at some of the gender differences here. First and foremost women have four times the leptin levels of men. Some of this is related to having more total fat but women's fat cells produce more leptin per pound of fat or kg as well. This is a direct effect of estrogen with testosterone reducing leptin production. At the same time, due to having more leptin to begin with, women are relatively more leptin resistant than men overall (32). Recall that leptin sensitivity also changes during the menstrual cycle, being relatively higher in the follicular phase and relatively lower in the luteal phase. And while not studied in humans, the hypothalamus (the structure that senses the changes in leptin and other hormones) of female animals is more sensitive to the changes in leptin compared to when it has been masculinized during fetal development. All of this means that the same drops in leptin levels between women and men are likely to send a stronger signal and generate greater adaptations in women. Adding to this difference is the act that women's levels drop both faster and to a greater degree than men's in response to both diet and exercise. For example, in obese women, leptin levels drop nearly as much in response to three days of fasting as in men who lose 21% of their starting weight (33). In older individuals, a similar degree of weight loss in both women and men reduce leptin by 45% in women but only 21% in men (34). Finally, in response to exercise alone, a week of regular exercise will reduce a women's leptin levels by 61% compared to a 38% drop in men (35). While not my focus, the hormone ghrelin increases to a greater degree in women in response to exercise and this occurs even if calories are raised to prevent a deficit from being created (36). This combination of changes would tend to stimulate hunger and food intake, even in the absence of a calorie deficit. Moving on to estrogen, I mentioned many of the effects that it has back in Chapter 2 and it's clear that it plays a major role in positively regulating women's energy balance (37). Recapping only briefly, estrogen is involved in all aspects of fat storage and oxidation while also blunting appetite. The loss of estrogen signaling at menopause has a number of significantly negative effects including increases in bodyweight, body fat and a shift in fat patterning all of which are reversed by HRT. While estrogen clearly has effects on fat and muscle cells themselves, much of this effect is due to estrogen's effects in the brain. As I discussed in Chapter 2, estrogen only sensitizes the brain to the effects of leptin but sends it's own leptin-like signal. This explains why the estrogen peak before ovulation (and more generally estrogen being the primary hormone during the follicular phase) has such a profound effect on reducing hunger and appetite. Relative to energy homeostasis, estrogen levels drop (along with progesterone) when calories are reduced and fat loss occurs in premenopausal women (38). All of these changes, along with others I'll discuss in the next chapter on menstrual cycle dysfunction, explain why the system works differently in women (especially lean women) compared to men. Essentially, in response to any sort of energetic stress, calorie reduction, exercise, or the combination there will be a drop in leptin levels. This drop will be occurring on a background of already higher leptin resistance causing the drops in leptin to have a more potent effect. This combines with the decrease in estrogen levels which will have an impact due to both estrogen's direct effects along with its effects on leptin signaling. These changes will all signal the hypothalamus to begin the adaptations to dieting and fat 104 loss that I described in such detail in Chapter 8. The adaptive component to RMR, TEA and NEAT will all occur along with an overall increase in hunger. The latter is primarily due to the changes in levels of serotonin and dopamine with dieting, similar to what occurs already during the luteal phase. And since the overall hormonal signals are more potent, you'd expect the adaptations to be both larger and faster. Why is it Like This? To wrap up this chapter and topic, I want to briefly look at some of the explanations that have been offered to explain why all of the gender differences in fat storage, loss, and and energy homeostasis exist. By explanation, I don't mean the physiological or biological reasons that I've already explained but rather the speculative evolutionary reasons that caused these differences to develop in the first place. Usually this means what benefits these changes might have had during evolution for survival in one way or another. The basic idea here comes down to the difference in reproductive roles between women and men and the idea that women were far more important for the survival of the human race, necessitating the differences that are seen (39). Women's bodies produce a singular highly energetically costly egg on a monthly basis while men's bodies produce large numbers of relatively low energy value sperm all the time. If and when pregnancy occurs, women bear an enormous energetic cost to support the development of the fetus in utero to begin with coupled with the energetic cost of breastfeeding after birth. While the cost is not enormous (perhaps 300-400 calories/day), it does add to her calorie requirements. Finally, women were tasked with raising the child after birth to the point that it was most likely to survive (about age 5). This necessitated being able to survive along with provide sufficient resources for the child. All of this added up to a woman's body needing to be relatively better at storing calories when they were available along with storing them in the relatively more difficult to mobilize hip and thigh fat (which recall are used preferentially at the end of pregnancy and to support breastfeeding). If food was not available in sufficient amounts, she had to be able to resist nutritional stress to a greater degree. I mentioned that women are more likely to survive a famine in the last chapter and it's interesting to note that women can generally maintain normal breast milk production in the face of insufficient food. In addition to storing calories more effectively, they are better at resisting nutrition stress and caloric insufficiency. Since dieting is just controlled starvation on a longer time scale, these same effects occur. Men in contrast faced none of these demands. Having provided their relatively low cost sperm to the reproductive process, they are not required further. Certainly it can be helpful if they are around in terms of providing resources or protection. But in the case where food is not available, I might argue that it is better for men to die earlier as this will leave more resources available for the pregnant mother and unborn child. But ultimately these differences in reproductive requirements between women and men drove the development of the differences by increasing reproductive success. Women who were more able to store calories or survive food shortages passed on their genes to the surviving children while those women who weren't did not. In keeping with the above ideas, it's worth considering the entire structure of the menstrual cycle as a whole as it is effectively geared towards a woman's both becoming pregnant and sustaining that pregnancy (the long-term adaptations to metabolic rate are more for long-term survival). The early follicular phase is, in one sense, the tail end of the previous cycle as the system starts to develop the follicle and prepare for the remainder of the cycle. But going into the late follicular phase, prior to ovulation, several changes occur as the follicle is about to be released and implanted. First is the small spike in testosterone levels which likely exists to increase sex drive. The large spike in estrogen is when a woman's appetite and hunger are at its lowest and it's been suggested that, by decreasing her desire for food and drink, a woman's desires for pleasures of the flesh will increase (40). A recent study supports this hypothesis, showing an inverse relationship between hunger and sexual interest: when hunger is highest, sex drive is lowest and the lowest hunger/highest sexual interest occurred when she was most fertile (41a). Adding to this, a number menstrual cycle of women's behaviors change around or at ovulation, more or less subconsciously that are geared towards reproduction. She becomes more physically attracted to masculine looking men (presumably with better genetics), becomes more receptive to sexual advances and her hips even swing in a way that is meant to be more sexually attractive (41-43). This is clearly being driven by the hormonal changes during the menstrual cycle as the use of birth control has been shown to modify or alter these. Specifically, women's preferences in terms of physical attraction change when they are on birth control as her body effectively "thinks" she is pregnant (44). 105 Entering the luteal phase after implantation of the egg, more changes occur to ensure that, if pregnancy occurs, there will be sufficient calories available. Hunger and appetite both increase with total calorie intake and cravings for carbohydrate and fat occurring. The increase in consumption of those foods, along with progesterone's direct effect on fat storage in the legs, all acts to ensure calorie and fat storage in the hips and thighs. If pregnancy occurs during a given cycle, her physiology will change completely as her storage of calories in her lower body will increase further, helping to ensure sufficient calories are available to support the later stages of pregnancy and breastfeeding. If pregnancy does not occur, her system will wind everything down to expel the un-implanted egg and endometrial lining during menstruation as the cycle starts all over again. A tangential question in this regard is why women's bodies to through the seemingly wasteful process of expelling the endometrium monthly rather than just allowing it to remain. This seems to be yet another adaptation to reduce energy expenditure. At least part, if not most, of the increase in energy expenditure during the luteal phase is due to the calorie needs of the endometrial lining and shedding it monthly will save her body 100-300 calories per day during the duration of the follicular phase compared to maintaining it for the entire time (45). I want to mention, simply for completeness, that many of the gender differences are not only driven by the reproductive issues mentioned above but by sexual selection. That is, many aspects of the body were selected due to being sexually or physically attractive. In most cases, these external manifestations were also indicative of her underlying physiology in one way or another (46). This includes such factors as her waist to hip ratio (indicative of her estrogen to testosterone ratio and overall reproductive potential), hair and skin (indicative of overall health), and permanently enlarged breasts (indicating her hormone levels, genetics as well as acting for sexual attraction), along with others. Some of these such as her hair/skin or breast size and shape even change throughout the menstrual cycle in concert with the changes in estrogen and progesterone. While the reasons appear to be manyfold, it's fairly clear that a woman's physiology is truly geared around her overall reproductive role (I'd note that so are men's, they are just set up differently). This is seen in her general tendency to store fat more effectively while losing it with greater difficulty and in a different general pattern than men. Her body shows overall energy conservation in both directions as she is more likely to become super obese as well as to survive famines. Even the monthly shedding of the endometrial lining is an adaptation to conserve energy, saving several thousand calories per month. If the menstrual cycle is lost completely, the normal increase in metabolic rate during the luteal phase will be lost and this can conserve 30,000 calories per year. Quite in fact, the dysfunction that can occur in menstrual cycle function may be one of the clearest indicators of differences in energy homeostasis regulate between women and men. This is discussed in detail in the next chapter. 106 Chapter 12: Menstrual Cycle Dysfunction Continuing from the last chapter, I want to look at the issue of menstrual cycle dysfunction. Not only does this represent one of the myriad ways that a woman's body can adapt to conserve energy, but it also represents a potentially very damaging physiological change that often occurs in response to diet, fat loss, exercise and stress. I want to re-emphasize that there is no such thing as a truly "normal" menstrual cycle to begin with. There is a general structure and pattern that occurs but this can vary enormously between any two women or even in the same women from month to month or throughout her age. I mention this as some of the dysfunctions (i.e. anovulatory cycles) that I'm going to describe may happen from time to time under otherwise normal conditions. This chapter is only addressing chronically occurring dysfunction. There can be quite a number of medical reasons that menstrual cycle dysfunction can occur but those are far beyond the scope of this book. The only one that potentially falls under a medical heading is frequent oligomenorrhea that is often seen with PCOS or elevated testosterone levels. Rather I will be focusing on four types of menstrual cycle dysfunction that are seen to occur in a dieting, exercising or athletic population. In that context, these dysfunctions would represent a change from the otherwise normal cycle. While each of these can absolutely occur in any woman, they are found at a many times greater frequency in the athletic/dieting population which points to a clear link between the two. In addition to looking at the different types of menstrual cycle disorders that can occur, what they represent and their physiological consequences, I will look in some detail at their causes. As there are many older ideas still floating around, many of which are incorrect, I'll look both at those older ideas along with what is felt now to be the primary cause of menstrual cycle dysfunction. One of those causes, stress, is so important that I will discuss it in detail in the next chapter. The majority of what I will be discussing in this chapter applies only to Category 1 women who are normally cycling to begin with. Certainly there can be menstrual cycle dysfunction for women in Category 3 but these are related to hormonal changes that occur with obesity (or underlying PCOS) or other medical disorders. For the most part, the presence of hormonal modifiers removes the possibility of menstrual cycle dysfunction although there are exceptions. The oligomenorrheic woman with PCOS/hyperandrogenism can develop amenorrhea and even women on birth control (BC) report increased breakthrough bleeding or other changes. There are also other hormonal changes of importance that occur in all Category 1 women that I will describe. Types of Menstrual Cycle Dysfunction While early work on menstrual cycle dysfunction identified only oligomenorrhea and amenorrhea, which were often grouped together as oligo/amenorrhea (this was prior to the realization that many athletic women were oligomenorrhea as a consequence of elevated testosterone), more recent work identifies four distinct dysfunctions that may occur. These are luteal phase defect/deficiency, anovulation, oligomenorrhea and amenorrhea which will all be described shortly. Luteal phase defect and anovulation are referred to as subclinical menstrual cycle disorders due to the fact that they aren't directly apparent as the menstrual cycle appears normal in duration and menstruation still occurs. Except for the inability to become pregnant (if that is the goal), there is no real reason for a woman to suspect she has either and the determination can only be made through medical workups. In contrast, oligomenorrhea and amenorrhea are termed clinical disorders as they do have external manifestations. In oligomenorrhea, the cycle is longer than 35 days and in amenorrhea it is completely absent. Luteal phase defect or deficiency refers to a situation where either the luteal phase itself is shortened to less than 10 days (the follicular phase lengthens here) or remains the same length with insufficient progesterone being produced. Estrogen levels may be low and this used to be called follicular phase deficiency (modern descriptions seem to group luteal and follicular phase defect together). In the case where estrogen is low, the egg may or may not be viable in the first place but some amount of endometrial tissue still develops (causing menstruation). Even if the egg is viable, there is insufficient progesterone to support implantation of the egg and infertility results. As noted, the cycle looks normal but is not. Anovulatory cycles, which are far more common than realized under normal conditions, refers to the situation where an egg itself is not released from the follicle for implantation. Estrogen remains lower than expected during the follicular phase and the normal increase in FSH (causing the follicle to burst) and LH (stimulating hormonal production) do not occur. There is still some development of the uterine lining which must eventually be shed, causing menstruation. Again, the cycle looks normal but is not. Oligomenorrhea is defined as infrequent menstruation with a cycle only occurring every 35 to 90 days. Here, estrogen and progesterone levels show an erratic pattern. On some days an oligomenorrheic 107 woman's hormonal profile may be identical to a normally cycling woman's but these days will not fall on the expected days of the month. On other days, the hormonal profile will show a completely different pattern from any day in a normal menstrual cycle. As in the subclinical disorders, infertility is common in oligomenorrhea but it is also outwardly clear that the cycle is abnormal due to it's length. While both represent oligomenorrhea, I want to mention that two types of oligomenorrhea can be distinguished here based on what is causing them The first is in women with overt PCOS or subclinical hyperandrogenism who's elevated testosterone levels are the cause of the lengthened cycle. In this case, the oligomenorrhea is likely to have been present from a very early age. The second type of oligomenorrhea is seen in the woman who were previously normally cycling and who developed oligomenorrhea in response to the causes I will discuss below. It is this second type that I will primarily focus on in this chapter Finally there is amenorrhea which is most generally defined as a lack of menstrual cycle but which is typically divided into two types. Primary amenorrhea has occurred if a woman does not begin menstruating (termed menarche) before age 15. As it's not likely to be that relevant to most readers of this book, I will only mention it briefly again below. Secondary amenorrhea occurs when a woman who has begun menstruating does not menstruate for more than 90 days and/or there are fewer then three cycles per year. Among other changes that are present below, amenorrhea is marked by both low estrogen and progesterone, neither of which show the normal cyclical changes. In this case, no egg is released, no uterine lining develops and menstruation does not occur. As I mentioned above, secondary amenorrhea can occur for a number of medical reasons but these are beyond the scope of this book. Rather I will be focusing solely on a specific type of amenorrhea that is marked by changes in both Follicle Stimulating Hormone (FSH) and below which problems start, a leaner women will cross it much sooner than a woman carrying more fat. When the individual responses to low EA are examined, leaner women always have a larger decrease in LH pulsatility (27). As I mentioned before, women in Category 2 and 3 almost never experience menstrual cycle dysfunction due to low EA. The lone exception being women who undergo bariatric surgery who have EA drop to effectively zero almost immediately. 114 Psychological Factors in FHA Having examined some of the physiological factors that can contribute or outright cause FHA, I want to look at other causes/contributors starting with psychological stress (discussed in great detail in the next chapter). Psychological stress is just as real to the body as physiological stress, often generating the same hormonal responses. Cortisol is a key player here; not only is it released in response to psychological stress (also going up with low EA) but chronically elevated levels can independently inhibit menstrual cycle function. Observationally, women with FHA often report increased interpersonal stress with friends and family (28). More directly, researchers have identified a subgroup of women with FHA who are not dieting or exercising excessively but who still show signs of menstrual irregularities or FHA (29). When psychologically tested, these women show a common cluster of behaviors including preoccupation with their weight and a high degree of perfectionism (traits that are often found in female athletes or dieters to begin with). Along with this is high dietary restraint, discussed previously, describing a pre-occupation with food intake and body weight. Not only is dietary restraint more likely to be found in women than in men, women with menstrual cycle dysfunction show higher degrees of dietary restraint than women without (30). Other research has found that women who have an overall high stress responsiveness, marked by dysfunctional attitudes, difficulty coping with daily hassles, subclinical depression or anxiety and others (30a). While these women often report some slight differences in their food intake (decreased fat intake, higher fiber intake), it does appear to be a case where nothing more than mental stress, and the chronically elevated cortisol responses that occur, is causing FHA (31). Further supporting that this is a psychologically driven issue is the fact that Cognitive Behavioral Therapy (CBT), which aims at teaching coping skills and alternate modes of thinking, has been shown to reverse FHA in these women (32). In one study, 87.5% of the women who underwent CBT regained menstrual cycle function versus only 25% of women who did not (33). To little saturated fat has a similar effect on hormones. While the effect isn't enormous with estrogen being reduced by 7% or more depending on how low fat intake is taken but it does exist. This is certainly beneficial from a health standpoint, in terms of reducing the risk of breast cancer (34), but it could contribute to the risk of menstrual cycle dysfunction as estrogen will be dropping from a lower starting point under such conditions. The studies on this topic are problematic in that they generally compare changes in both dietary fat and fiber, making it impossible to know if it's the fat, fiber or the combination that is problematic. Overall, though, low fat, high fiber diets do tend to lower estrogen levels. Two studies are worth mentioning here; in one women went from 35-40% of calories from fat to 25% of calories from fat while increasing fiber intake from 15 to 40 grams per day (35); these were low saturated fat diets. In the other, it's impossible to know what might have happened since the diet was low in fat but also high in fiber. This is relevant as, independent of calories, changes in fat and fiber intake have been found to alter a woman's hormonal profile. Specifically a low-fat/high-fiber diet lowers levels of both estrogen and progesterone compared to a high-fat/low-fiber diet (36). In one study, women had lower estrogen and progesterone and this is likely due to the lowering of both estrogen and progesterone in the first place (35). The same low-fat diet, over a 2-year span, has also been shown to reduce breast density (36). In that they are typically low in fat and high in fiber, vegetarian and vegan diets have the potential to cause menstrual cycle dysfunction (37,38). In one of the studies I cited above, while dieting itself increased the risk of menstrual cycle disorders, women on vegetarian diets were more likely to experience them. Perhaps surprisingly, the generally considered to be healthy Mediterranean diet (based around moderate protein, high-vegetable and relatively low-fat intakes) can have the same negative impacts on menstrual cycle function (39). While a low-fat/high-fiber intake may be a partial cause, there are other reasons that vegetarian/vegan diets could be contributing to menstrual cycle dysfunction. One is that the 115 nature of the diets often lower caloric intake, which could take women below the critical EA threshold (especially if they are active). Limited intake of specific foods such as red meat might also cause nutrient deficiencies (i.e. iron and zinc). In one study, almost 25% of amenorrheic women were vegetarian and 100% of them ate no red meat (40). They also consumed far less calories so a lowered/low EA was also present. The above is not meant to necessarily argue against a low-fat/high fiber intake. Clearly it is beneficial for improving some health markers, reducing symptoms of PMS and reducing breast cancer risk. At the same time, it may not be optimal for athletes or leaner/dieting women in terms of maintaining normal menstrual cycle function. Optimal fertility might also be harmed with certain dietary patterns and women seeking to become pregnant should look at their overall diet in this regard. This is also a potential area where a typical male approach to dieting (especially for the Category 1 individual) with a very low-fat and high-fiber intake might not be ideal for women. Men's hormones can certainly be impacted but they have no menstrual cycle to lose and clearly don't suffer the same overall consequences. Having looked at the potential impact of low-fat/high-fiber diets, I want to look at the other extreme and talk about high-fat/ketogenic diets. There are diets typically containing moderate or high protein intakes, low levels of carbohydrate (50-100 grams or less) with relatively higher fat intakes. Anecdotally at least, there are reports of leaner women who had lost their menstrual cycle regaining normal function when they follow such diets. I tend to think that this is related to these types of diets being higher in fat and often lower in fiber by definition but what little research exists is mixed. In obese women with PCOS, ketogenic diets have been shown to cause weight and fat loss and improve menstrual cycle function but almost any weight loss in this population has similar effects (41,42). In leaner, women I'm only aware of a single study. In it, women were placed on a ketogenic diet for the treatment of adult epilepsy (this is one of the uses of the diet) and, despite being at maintenance calories, 100% of them showed some degree of menstrual cycle dysfunction (43). This is not an ideal model for dieting women, mind you. The ketogenic epilepsy diet is set up differently than the fat loss version and the presence of epilepsy (or use of medications) could have interacted somehow. However, ketogenic diets effectively mimic starvation while eating food but the same overall hormonal responses to starvation are typically seen. Adding to this, the female brain requires roughly 80 grams of carbohydrate per day (in contrast to a male's 100-120 grams) and ketogenic diets automatically provide less than that. If the brain's carbohydrate availability, rather than EA per se, is a controller of LH pulsatility, the lack of dietary carbohydrates (along with the brain's shift to using ketones for fuel) might contribute to menstrual cycle dysfunction. Ketogenic diets can be modified to address this issues. Overall, the above research points to a situation where extremes of diet are potentially problematic for lean female athletes at risk for suffering menstrual cycle disruption. It would appear that for lean women sufficient dietary fat and carbohydrate are required for a woman's optimal hormonal and physiological function to at least one degree or another (sufficient carbohydrate is also necessary to support high-intensity training). And this brings up an issue I have mentioned throughout the book: during a diet, due to women's smaller sizes, there is often not enough room to cut calories in the first place. Indeed, there are solutions to this addressed later in the book but this represents another situation that men often don't have to face. Due to being larger and having higher energy expenditures, their diet often allows more than enough room for all the nutrients. Causes of a Low EA While there are other potential contributors to menstrual cycle dysfunction, in a vast majority of cases, it is low EA that is the primary cause. This has in turn led to looking at the causes of low EA. I want to look briefly at some of the reasons that a woman's food intake might be insufficient to prevent her EA from falling below the critical threshold (44). The first are conscious restrictions of food intake, usually aimed at decreasing BF%. As I've mentioned previously, performance athletes often benefit from a reduced BF% although this can harm performance if taken to the extreme. In the physique sports, a low BF % is part of the competition itself and reductions to very low levels represents a necessary evil. So while it might be debatable if a female endurance athlete needs to reach 10-12% for optimal performance, a female bodybuilder or physique competitor will have to do this to be competitive. Beyond even competition reasons are the simple fact that women are currently and have always been under social pressure to be thin which, as often as not, pressures them to diet. This is true in the athletic realm as well as in the general public with relatively "normal weight" women often wanting to lose weight or fat. Tied in with the issue of the diet itself is the impact of exercise, especially high-intensity exercise on appetite that I discussed in a previous chapter. I mentioned that this impact can be variable often increasing 116 hunger but, in trained athletes, intense exercise may actually blunt hunger. This effect is increased on a high-carbohydrate, high-fiber, low fat diet and this combination can cause female athletes to unconsciously undereat relative to their daily requirements (another reason to maintain adequate fat intake). Women with menstrual cycle dysfunction have also been shown to choose foods low in calories for their volume (low energy density), this keeps them full with insufficient calories to support their training (44a). Finally is the often high-prevalence of overt eating disorders (EDs) in both women in general and female athletes in specific that cause women to deliberately reduce their food intake, often to extremely low levels. The term anorexia athletica is often used and, even when full-blown EDs aren't present, subclinical forms may be. This is especially true in those sports requiring low body weight/body fat levels and which are focused on thinness (45). I'd remind readers that the presence of ED's may be part of the reason that these women are successful in sport to begin with, rather than being a consequence of involvement. I've even argue that, especially in the physique sports, the presence of a subclinical ED can be seen as a competitive "advantage" in that it helps the person sustain the extreme diet that is required to be successful. These comments are in no way meant to diminish the huge damage that EDs, whether overt or subclinical, do or that they should be ignored without treatment. Who Keeps Their Cycle? While amenorrhea is obviously fairly common among lean female athletes, it is also clearly not universal with female athletes reaching very low levels of BF% and body weight without losing their cycle (though most probably show subclinical menstrual cycle dysfunction). Some of this could be due to better overall dieting or eating practices such that the critical EA threshold is not crossed (or is crossed later before being raised once the fat loss goal has been reached) but females are also found below the critical threshold who are not amenorrheic. The question is why and the answer is very simple and fairly unknown. Researchers often refer to the concept of robustness in terms of menstrual cycle function when discussing this topic. The basic idea is that, in the same way all biological systems can vary between people, so can women's reproductive systems and/or hypothalamus are more robust than others. The same degree of low EA, weight/fat loss or exercise simply does not affect them as much as it does other women. Going back to the issue of women and famine is the fact that, even during the most extreme situations (i.e. starvation/concentration camp victims), some women are still able to conceive and bear children. Presumably their systems are more robust. Practically this becomes an issue where only individual women (who may have a more robust system) is able to diet or reach a certain BF% without problems and assumes this applies to all women (who are not as robust). Sadly, in almost all of the discussions of the topic I've found, nobody has offered any actual reason for these differences. As is usually the case there may be genetic contributors to the relative risk of FHA occurring (45a). At least some studies have found that women with pre-existing low luteal dysfunction are at risk of losing their cycle completely. Given the progressive continuum of menstrual cycle disturbances that can occur, this most likely represents them already starting out with some degree of psychological stress or low EA. Women who start dieting with a pre-existing dysfunction will simply get worse. Outside of genetics, only one factor has been directly identified that can limit menstrual cycle dysruption and that is a concept referred to as reproductive or gynecological age. In contrast to biological age, which is how long a woman has been alive, reproductive age represents the number of years since she began menstruating. If a woman starts menstruating at age 15 and is now 25 years old, her reproductive age is 10 years. If she is 30, her reproductive age will be 15 years. Women reproductive systems are often very unstable when they first reach menarche but becomes more stable with age. Full reproductive maturity appears to coincide with the time that her pelvis stops developing and this occurs at roughly a 14/16 year reproductive age. And it has been shown, again by Ann Loucks, that the impact of a low EA on LH pulsatility disappears in women after they reach a reproductive age of 14 (46). The same changes still occur in thyroid, insulin, IGF-1, leptin and cortisol to this in just won't impact LH pulsatility. The reproductively mature Category 1 woman attempting to diet down to the extremes of leanness is still at risk for menstrual cycle dysfunction (47). The women in Loucks's study were at 26% body fat, just above my Category 1 cutoff and this may have contributed to the lack of change in LH pulsatility. The reproductively mature Category 1 woman attempting to diet down to the extremes of leanness is still at risk for menstrual cycle dysfunction, not while a topic of this book, the reality is that many females athletes are at least a 14 year reproductive age, putting them at the greatest consequence for stress/low EA on their reproductive systems. 117 Putting It All Together I've discussed a lot of information in the previous sections and I want to try to put them all together into a cohesive model of menstrual cycle dysfunction. There are, of course, still a number of gaps in the research and/or conflicting research. In a general sense, it's fairly clear now that the primary factor that causes menstrual cycle dysfunction is a chronically low energy availability (EA) with a proposed critical EA threshold existing below which problems begin (and above which they don't). Adding to this are the individual differences in robustness. Women with menstrual cycle dysfunction often show a higher degree of dietary restraint and this in and of itself, in turn, causes weight and/or fat loss. If exercise is not performed and it has been shown that greater dietary deficits produce a higher frequency of menstrual cycle dysfunction although they do not predict the severity (48). Exercise may have its own independent effect if an excessive amount is done without a build-up and dieting/weight loss further increase the odds of the problem. I want to explain the earlier studies with reproductive age explaining why younger women are more likely than older to have problems. Weight/fat loss leading to any specific weight or body fat percentage are also likely to be relevant to the degree of dysfunction the person may experience. The lean woman whose athletic drive is causing them to have low EA (e.g. below a certain BF%) might, of course, still stay at least some risk. Which brings things full circle to the role of BF% in all this. At least until such a time as the body adapts by lowering energy expenditure, a low EA, by definition will be creating a caloric deficit, causing both weight and fat loss. It's most likely that the low EA/calorie deficit is causing the low BF% seen rather than low BF% causing the dysfunction. This would explain the fact that menstrual cycle dysfunction doesn't occur at any specific or critical body fat percentage (BF%). In addition to genetics or reproductive age, a woman at a higher BF% could be experiencing a chronically low EA while a leaner woman might not be. Even here, while related to BF%, leptin is far more sensitive to changing calorie (and carbohydrate) intake meaning that it will be impacted most by a chronically low EA. And, regardless of the other contributory factors, it is that chronically low EA that eventually causes a woman's normally cycling to luteal phase defect to anovulation to oligomenorrhea and, potentially at least, to full-blown amenorrhea. Psychological factors such as personality profile or other stress may also play a role. It can be difficult to separate out energetic from psychological stress and I will discuss this more in the next chapter. Both factors can contribute independently, interact or act as a driver for the other (ie. many people will exercise to relieve psychological stress but exercise can cause energetic stress). In a practical sense all of these factors are often clustered together. Women with a certain psychological profile who are exercising excessively while chronically undereating/eating to specific diets may be creating a perfect storm to generate menstrual cycle dysfunction. If certain genetic factors or a low reproductive age are also present, the problem worsens. The Effects of Amenorrhea As I mentioned earlier in the chapter, it's now known that the various menstrual cycle dysfunctions represent a progressive movement from a normal menstrual cycle to FHA and that the changes that occur in a woman's hormonal and physiological state tend to go hand in hand with the changes in the menstrual cycle, and they also go in the same direction in the "endpoint" of menstrual cycle dysfunction, and you have had multiple impacts on all aspects of a woman's physiology. I will discuss FHA first with the understanding that the lesser degree of menstrual cycle dysfunction represent lesser degrees of the same effect. As a low EA is often a consequence of dieting and fat loss, there is at least some overlap between FHA and the normal adaptations to dieting. As I mentioned in Chapter 3, a primary change in amenorrhea is an overall decrease in a woman's reproductive hormones. The normal cyclical changes in LH and FSH are also lost which means that no egg develops or is released, no corpus luteum develops and there is no menstruation. Estrogen may be reduced to 33% of normal with progesterone at 10% of normal and no cyclical changes occurring. Given the profound effect of these hormones on a woman's body, this drop has an enormous number of effects. Some of these effects could be looked upon as somewhat of a positive. The loss of progesterone signaling means that its effect on stimulating fat storage via ASP are lost. As well, the lack of cyclical changes in a woman's hormones means that the typical shifts in water weight and retention disappear. This can make tracking a fat loss diet easier and may reduce the mental stress in female dieters that comes from bodyweight jumping up and down. Women who suffer from PMS often see a reduction in those symptoms along with the loss of menstruation. Even that has a small benefit in that it reduces a woman's monthly iron loss, reducing her risk of developing anemia. While the above do represent potential positives of amenorrhea (and some women speak of "banishing the estrogen", actively embracing the loss of the cycle), I cannot overstate that the negative effects are both more numerous along with being far more potentially damaging to a woman's physiology and long-term health. 118 Looking first at the drop in estrogen, a number of metabolic effects will be seen. The lack of estrogen signaling means that hunger will no longer be well controlled as both its direct effects and leptin sensitizing effects will be lost. Along with the loss of estrogen's anti-inflammatory effects, anti-oxidant effects and beneficial effects on muscular remodeling (Metabolically, the positive effects of estrogen on fat mobilization and oxidation will be lost with the consequence that the use of protein for fuel during aerobic exercise may be increased and a typical estrogen patch can reverse this (49). Contrary to what might be expected, insulin sensitivity increases, shifting fuel use towards carbohydrates and away from fat. Again, the loss of progesterone signaling means that the normal increase in metabolic rate that would occur during the luteal phase is lost significantly reducing monthly calorie expenditure. This is in addition to the normal dieting adaptations. Other hormonal changes occur and I have mentioned that there is a common hormonal pattern which is seen with low EA including low insulin, elevated cortisol (a stress hormone discussed in the next chapter), low leptin, low levels of thyroid hormone (T3) and a decrease in the levels of a hormone called Insulin-Like Growth Factor -1(IGF-1). This impacts on all aspects of a woman's physiology but here I want to focus on its metabolic rate. In amenorrhea (and to a lesser degree the other stages of menstrual cycle dysfunction), T3 levels can be a significant reduced, with up to 17-20% decrease in basal metabolic rate (BMR) compared to normally cycling women. I'd note that this drop is also seen in lean women without amenorrhea along with dieting and many of these changes can be 25-51. For comparison, BMR can be reduced up to 30% or more in anorexia, a state which shares many commonalities with amenorrhea (52). Other components of daily energy expenditure have not been measured as much in such detail but all of the adaptations seen with dieting in general will be in place. NEAT is likely to decrease to overall fatigue and lethargy. In terms of training, injury risk may increase (see below) while muscular protein synthesis, muscular strength, endurance, recovery both during and between workouts and training response and competitive ability may all be decreased. In one study of junior elite swimmers, amenorrheic athletes had a 10% drop in performance compared to a 8% improvement in normally cycling women, an 18% difference (52a). Judgement and coordination may be impaired along with an increased risk of irritability and depression Amenorrheic athletes are at risk for nutrient deficiency, although this is primarily due to the low total food intake and food choices that are often seen. There may also be decreased immune system function along with cardiovascular, gastrointestinal and renal complications. A commonly reported effect is an increase in mental stress, and I'd note both depression and lethargy are being seen. Normally melatonin goes up at night and decreases in the morning but in many kinds of depression, melatonin stays elevated throughout the day, causing lethargy while being lowered at night, causing sleeplessness (53,54). Called a phase shift, this is common in people suffering from Seasonal Affective Disorder (SAD) and occurs to some degree during the late luteal phase. It also occurs in amenorrhea due to the changes in reproductive hormones that occur (55,56). The above negative effects, while significant, pale in comparison to what may be the single largest negative effect of amenorrhea which is the potential for bone mineral density (BMD) loss. Women already have lower BMD than men and only have a limited time to develop peak levels. Roughly 90% of the increase in a woman's BMD occurs by age 18 with the remainder finishing by roughly age 30. From that point on, a slow loss of BMD typically occurs, accelerating at menopause (especially if HRT is not begun). Proper training and nutrition may be able to slow or eliminate this normal age-related loss. If the loss of BMD is excessive, osteoporosis (defined as a BMD 2 standard deviations below normal for age) occurs, predisposing women to falls and fractures. Practically this means that the peak BMD a woman reaches in her youth (between adolescence and age 30) is a huge determinant of her risk for osteoporosis later in life. With the development of amenorrhea, not only will BMD not increase during critical times of a woman's life, it will actively be lost. Women with amenorrhea may lose 2-5% BMD per year when she would have been gaining 2-3% BMD during the year if she were not amenorrheic meaning she will end up 4-8% below where she should be. So long as the amenorrheic state is maintained, not only is a female potentially losing bone mass permanently, she is limiting the peak BMD she may ever reach with some studies finding that amenorrheic athletes have BMD lower than even sedentary women. This predisposes women involved in sports that involve high impact forces such as running or gymnastics to be at a significantly higher risk for stress fractures in the lower leg (57). Cyclists are at risk for hip stress fractures as well. There is also some indication that the bone lost when amenorrhea develops is irreversible (58). This isn't universal and some degree of "catch-up" may occur if a woman re-establishes normal menstruation and optimizes her training and nutrition. This reversal, if it occurs, can take years (59,60). 119 While the optimal approach to limit or eliminate BMD loss is to restore normal menstruation (discussed later in the chapter) and optimize nutrition, a number of pharmacological approaches have been used to attempt to improve BMD loss in amenorrheic women. Of these, the use of estrogen replacement (in the form of hormonal birth control (BC) should have helped the problem. While some studies have suggested a benefit, the evidence is considered at best weak that providing synthetic estrogen to amenorrheic women can improve BMD or reduce stress fracture risk. This is due to the importance of other hormones such as leptin, thyroid and IGF-1 that all play a critical role in bone metabolism. Just replacing estrogen isn't enough and the optimal solution to the problem of bone loss is to avoid developing amenorrhea in the first place or reverse it as soon as possible if it does occur. (Progressive) Increasing Changes While amenorrhea is the most extreme endpoint of menstrual cycle dysfunction I mentioned above that the progression from normal to luteal phase defect to anovulation to oligomenorrhea to full blown amenorrhea is progressive in many ways with increasing levels of menstrual cycle dysfunction. From luteal phase defect to anovulation to oligomenorrhea to full blown amenorrhea the effects increase with a progressive loss of bone density and worsening of hormonal status and metabolic rate (62). Levels of thyroid hormone progressively decrease as does insulin, leptin and IGF-1 with progressive increases in cortisol and this increases the overall metabolic response that occurs. Overall, all of these adaptations point to an energy conserving/starvation state. I would mention that even if the bone density is not always reversible, there is no evidence that any other aspect of reproductive function is permanently damaged (many female athletes report pregnancies soon after regaining normal menstrual function). I've summarized changes that occur with progressively increasing menstrual cycle dysfunction below (63). Some values are compared to sedentary women which is why even eumenorrhea in active women shows changes. The arrows represent the degree of change and should be interpreted as a proportionally larger decrease or increase in a given aspect of function with a clear indication of the progressively worsening effects. Eumenorrhea Luteal Phase Deficient Anovulatory Oligomenorrhea Luteal Phase Defect Anovulatory Oligomenorrhea* Menstrual Cycle Normal Appears Normal Appears Normal Lengthened (35-45 days) Absent (<3/year) Thyroid (T3) —Metabolic Rate —Leptin —Insulin No Change —GH No Change —IGF-1 No Change No Change —Cortisol + + + +++ Blood Glucose No Change No Change —Follicular LH Pulsatility —Estrogen —Progesterone — Can be due to pre-existing elevated testosterone levels as well. Source: Adapted from Reference 58 The Female Athlete Triad/Relative Energy Deficiency Syndrome In the earliest days of research on the topic, a cluster of behaviors was identified that was being seen in female athletes including amenorrhea, eating disorders and a severe loss of bone density. This was rapidly given the name the Female Athlete Triad (somewhat unfortunately abbreviated FAT) or simply "The Triad". The concept of the Triad has been modified over the years in many ways primarily being expanded to include the subclinical versions of the original three factors rather than just the extreme endpoints (64). Essentially, the Triad is now conceptualized as a continuum of each of three primary components, each of which can fall along a spectrum of dysfunction. In this conceptualization, menstrual cycle function can range from a normal menstrual cycle through the subclinical dysfunctions to oligomenorrhea (again, not PCOS/hyperandrogenism related) with amenorrhea as the final stage. Energy availability ranges from an optimal or sufficient EA through a reduced EA (with or without an eating disorder) finally to a low EA (below the critical threshold of 1.35 cal/lb or 30 cal/kg LBM). Bone density ranges from optimal bone density (for a woman's age) to low bone mineral density (a BMD 1 standard deviation below normal) to full-blown osteoporosis (2 standard deviations below normal). I've shown this schematically below from optimal function to the most extreme end-points of the triad in the chart below. 120 Returning to the issue of bone density loss, it's important to realize that this is an effectively "silent" component of the triad (65). By this I mean that low EA can generally be identified by comparing activity to food intake and at least some form of menstrual cycle dysfunction are readily apparent. Without specific testing (i.e. a DEXA scan), a woman may be losing bone without ever being aware of it. The Female Athlete Triad Energy Availability Optimal/Sufficient Menstrual Normal Cycle Function Reduced with or without an eating disorder Luteal phase defect Low Bone density Anovulatory (1SD below normal) Oligomenorrhea (non PCOS/hyperandrogenism related) Low (below critical threshold) with or without eating disorder Amenorrhea Bone Density Optimal (or age Osteoporosis (2SD below normal) In the early days of the triad, it was thought that a woman had to exhibit all three components to be diagnosed with the Triad. Now, as noted, a woman can fall under the umbrella without showing all three components of the Triad. Even if nothing more than a low EA is present, a risk exists as the components tend to happen sequentially. Chronically low EA can eventually lead to menstrual cycle dysfunction, potentially developing to full-blown amenorrhea and that can occur before there is any meaningful impact on BMD which will occur last and over the longest time frame. The recognition and reversal of a low EA when it is first identified, may prevent the other two components from developing. Given the primary role of low EA in driving the negative effects which occur, it's been recently suggested that FAT be renamed Relative Energy Deficiency Syndrome or RED-S (66). This is meant to account for almost a decade of research showing that many of the other factors caused by a low EA beyond menstrual cycle dysfunction and BMD loss (note that these are already included in the most current version of the FAT). RED-S is also meant to be more inclusive, including both recreationally exercising women (who may not identify as "athletes") along with men. This has not been without controversy and the original authors of the FAT position stand have pointed out numerous errors in the original RED-S position stand (67). Their greatest concern is that changing the name might reduce awareness of the profoundly negative effects that are seen specifically in female athletes due to the consequences to reproductive function or bone health). The original authors of the RED-S have addressed these issues in an update to their original position stand (68). As this is more of an academic debate, I only mention it here for completeness. Regardless of the above, the primary message of this chapter is that a chronically low EA, below a critical threshold, is the primary issue to be addressed for women. While it's not impossible that some FHA occurs even with sufficient EA due to psychological stress, for a vast majority of cases, the goal should be to avoid or reverse a low EA as early as possible to prevent the more long-term changes from occurring. Reversing Menstrual Cycle Dysfunction In an ideal world, women would avoid menstrual cycle dysfunction, or at least full-blown amenorrhea completely. In the real world, for various reasons ranging from pathological dieting practices to the requirements of some sports, menstrual cycle dysfunction will occur to some degree or another. Given this fact, the rapid reversal of the dysfunction is of primary importance and I want to finish the chapter by looking at how that is best accomplished along with some of the issues that might surround what needs to be done. In many situations, various pharmacological approaches are used (or required) but those are beyond the scope of this book and I want to focus only on the non-pharmacological approaches. Inasmuch as the fundamental cause of hypothalamically based menstrual cycle dysfunction is a chronically low EA, the cure is as simple as raising EA and maintaining that increase until normal menstrual cycle function resumes (69). In practice this means increasing calorie intake, decreasing activity levels or some combination of the two. This will invariably be accompanied by an increase in body fat and weight but this is a necessary aspect of this approach. It is also one of the primary reasons that female athletes often resist doing it, discussed further below. 121 Research, either involving a single or small groups of athletes, has typically approached this by increasing daily calorie intake by 250-350 calories per day (many use 300 cal/day due to the use of a commercial protein/carbohydrate supplement) with a reduction of one day of training per day. The success rates of these interventions in terms of restoring menstrual cycle function has ranged from as little as 18% up to 100%. Typically it is the smaller studies showing the best success rates and this likely represents the amount of control that can be exerted over the subjects. In one study of four women who had lost their cycle for 6-9 months, three began menstruating within 2-6 months (one dropped out) although two did not have ovulatory cycles for several months afterwards (70). In contrast, in another study only 18% of 51 amenorrheic athletes regained their cycle after 5 years. In all cases, the women who gained more total body weight were more likely to resume menstrual cycle function. This likely just represents the fact that the women did increase their food intake/decrease their activity and maintain those changes at an appropriate level for long enough to recover their cycle (71). In cases where menstrual cycle does not occur within the length of the study, benefits are still found. In one, a runner who had been amenorrheic for 14 months, reporting chronic fatigue and injury was compared to three normally cycling women over 15 weeks as she increased her calories and reduced training (72). Despite not menstruating during the study (she would start 3 months after it ended), she showed a huge 50% drop in cortisol levels with an increase in LH levels a drastic/performance increase after increasing EA. In another, 5 amenorrheic and 26 oligomenorrheic females did not begin menstruating during a 3 month intervention although all showed signs of hormonal and metabolic recovery (73). The study was most likely simply too short to see full menstrual cycle recovery. In addition to being the most important factor in restoring menstrual cycle function, increasing BMD is the single most important factor. I mentioned earlier in the chapter that there is often a lag time between the resumption of menstruation and the reversal of all the other negative changes which occur. It has been shown that BMD starts to increase within 1-2 months to restore normal function (74). I'll describe two case studies on physique athletes in Chapter that seem to support this. Looking at the studies as a whole, the amount of time it takes for a woman to regain normalcy can vary from a matter of weeks to up to 3 years and there is no real way to predict how long or not it may take for a woman's cycle function to resume (75). In the case where a woman has not regained her cycle over a multi-year span, it's possible that there are other causes that need to be addressed medically. It's important to realize that even when menstruation resumes, the cycle may not be completely normalized for up to one year. In one study, two female athletes who had lost their cycle for either 33 or 23 days were followed for one year (76). The first athlete had her first menstruation after 2.5 months but it was anovulatory. She also reached her cycle and started ovulating after the first year of intervention. It does make it clear that even with normal menstruation a woman's cycle could still be in the process of recovery and this is clear that the athlete is still in an anovulatory state. While some women took approximately six months although their EA was still evidenced luteal phase defect. One other studies mentioned above found that regaining a sufficient EA is the most important factor. I would note that the longer a woman has had amenorrhea, the longer it may take to recover (77). 122 Factors such as the rate/size of calorie increase (to raise EA), the period of time (level of weight/body fat regain) slowly over time to a point where the woman regains their menstrual cycle and individual factors such as stress, sleep or genetics impact on this. In most studies, the degree of weight gain is a critical factor with women gaining 4.5 lbs (2 kg) or more being the ones who begin menstruating. Given the generally weak relationship between weight, BF% and menstrual cycle function, the weight gain is likely a consequence of the increased EA rather than being the actual cause of recovery. While the above is certainly simple in principle, it is often less simple in practice. Many female athletes are often extremely resistant to increasing calorie intake, decreasing activity or gaining any amount of body fat even if it is required for their long-term health or performance. In many cases, the athlete will compensate for one change with another, increasing activity to offset calorie increases or training more or harder on other training days to compensate for reducing the number of days they train. Inasmuch as certain psychological profiles or attitudes may be present as causing or at least contributing to FHA, just recommending that calories be increased or activity be decreased may not be sufficient. CBT to alter modes of thinking or coping might very well be required for real success or as an adjunct to raising EA and decreasing activity (i.e. by reducing preoccupation on food intake or body weight). In the case where an overt or subclinical eating disorder is present, psychological counseling is likely to be a required part of the recovery process (77) 123 Chapter 9: Stress While I've mentioned stress and its effects on the body in earlier chapters, I want to look at it more in depth in this chapter in terms of its potential impact on a woman's physiology. Here, as would be expected there are significant gender differences in the stress response and, just as with the issues discussed in the last chapter, has implications for how women can or should approach the goal of dieting (whether in general or to the extremes). Many approaches that might be tolerable for men are, at best, not ideal for women and, at worst, physically damaging to them. The primary focus of this chapter will be the hormone cortisol, a hormone that I've deliberately saved detailed discussion of until now. First I want to define stress in some meaningful way before looking at what cortisol is and what it does. Unsurprisingly there are significant difference in how women and men respond to stress. This will provide the background for a discussion of how the types of chronic stresses that are often seen among female athletes, recreational exercisers and dieters can cause problems in the long-term, including one potentially permanent effect. What is Stress? Given its importance in human health and physiology, it may be surprising that the concept of stress is not well defined in a biological sense (the term originally came from engineering). Somewhat circularly, stress is sometimes defined as the body's response to a stressor where a stressor is defined as anything that causes stress. As I stated in the last chapter, a slightly more useful definition would be the body's response to anything that tries to push it out of homeostasis, the relatively fixed level of function that it tries to reach. In general, stresses can be broken down into a few broad categories: heat or cold), physiological stress (intense exercise), energetic stress (caloric restriction) or psychological stress (worrying about taxes). Each causes the body to respond in some form or fashion physiologically. Heat will induce sweating while cold will cause shivering, exercise causes a short-term increase in calorie burning and nutrient mobilization, etc. While many tend to think of stress as nothing but a negative, this is incorrect. Rather, the way the body reacts to stress can determine whether stress is beneficial or harmful to the body. When stresses are relatively short-lived, occurring and then ending relatively rapidly, the body not only has time to recover but is often stimulated to grow stronger from that stress. Exercise is perhaps the best example of this where a workout or series of workouts stresses some tissue the body (both physically and hormonally) which stimulates the body to adapt (i.e. muscles and bones grow stronger, the heart and enzymes adapt, etc.). While this adaptation can take the same workout less stressful in the future It is only when too much stress occurs for extended periods that problems occur. Here the body never has a chance to recover or rebuild itself and this causes it to eventually break down. I would point readers interested in more details of this topic to the eminently readable Why Zebras Don't get Ulcers by Robert Sapolsky. He explains the ins and outs of the stress system along with providing advice on decreasing stress. While each type of stress tends to cause a specific physiological response, they also stimulate a more generalized stress response. Surprisingly, extremely different stresses tend to generate a fairly similar stress response. There may be subtle differences in the stress "signature" but the overall effect is identical for the purposes of this book. This explains a point I made in the last chapter which is that every stressor applied to the body ends up adding to the overall stress (or allostatic) load. One stressor may be bad but two stressors is worse than one, three is worse than two, etc. and this is due to each stressor causing the same general physiological and hormonal response. And while other hormones such as adrenaline are involved, the primary hormone of relevance in the context of stress is cortisol. Cortisol The hormone cortisol is actually one of several hormones that are grouped under the heading of corticosteroids but I will refer to them all as cortisol for simplicity. Cortisol is released from the adrenal gland which releases many other hormones such as adrenaline, the adrenal androgens such as DHEA and the mineralocorticoids, hormones involved in water balance, such as aldosterone. As with many hormones, the signal to release cortisol comes from the hypothalamus which, via release of Corticotrophin Releasing Hormone (CRH), stimulates the pituitary gland to release adrenocorticotrophic hormone (ACTH) which signals the adrenal gland to release cortisol. The general trigger for this process to occur is, as the name of the title and the previous section suggest, stress. 123 Cortisol has broad reaching effects in the body and, due to many of its effect, is often thought of as being a bad hormone. This is prevalent in both the athletic community as well as the general public due to endless advertisements claiming that it increases belly fat. But just as the effects of stress can be positive or negative, the effects of cortisol (which really mediate the effects of stress) can also be positive or negative. Simply, acute pulses of cortisol (caused by short term stress) tend to be beneficial while chronic elevations of cortisol (caused by long term stress) is not. In the short-term for example, cortisol acts to generally mobilize energy. Fatty acids are released from fat cells, glucose is produced in the liver and even protein breakdown is stimulated. Even this latter effect is adaptive, helping muscle to rebuild and remodel itself in response to that stress. Acutely, cortisol does impair bone growth but this is primarily a way to conserve energy (and when the stress ends, the bone can rebuild). Cortisol pulses help to form memories which is why people tend to remember acutely stressful events and acts as an anti-inflammatory, explaining its use to treat injuries. Perhaps confusingly, cortisol acutely impairs immune system function but this too makes sense: the immune system uses a stunning amount of energy and inhibiting it in the short-term helps to provide energy for whatever stressor is being dealt with. Cortisol also impairs the function of the reproductive system; in the short-term this makes perfect sense for the same reasons menstrual cycle dysfunction makes perfect sense when insufficient energy is available. But in the aggregate, all of these effects are extremely beneficial for the body and are part of what helps it to adapt to stress over time (1). In contrast, when cortisol is elevated chronically, its effects become distinctly negative. Cortisol causes insulin resistance and leptin resistance in the brain, both of which can have negative effects on body weight and body fat regulation. If insulin levels are high when cortisol is chronically elevated, visceral fat storage can be stimulated (hence the claim that cortisol leads to belly fat). With chronic stress, cortisol increases protein breakdown but without the recovery period, tissue never get a chance to repair themselves. In the long-term this causes muscle loss and other bodily damage. Bone production is inhibited continuously and eventually this will cause the loss of bone mineral density (BMD). Memory is impaired and immune system, reproductive function and sex drive are all inhibited as well. As background for later in the chapter, there is a slightly more complex aspect of cortisol dynamics called feedback inhibition that I need to describe. In general, when levels of a hormone go up, this inhibits further release of the hormone and this is meant to maintain that hormone at a relatively constant level. Relative to cortisol and the HPA, the hypothalamus has cortisol receptors that, when cortisol binds to them, is meant to inhibit the hypothalamus from telling the pituitary gland to have the adrenal gland release more cortisol. This loop stops working correctly, a huge number of problems can occur. As an example, impaired feedback inhibition of cortisol release is seen in 95% of people with depression. Finally let me mention that cortisol is metabolized extensively in fat cells (especially visceral fat cells). Active cortisol can be converted into inactive cortisone and vice versa and local levels of cortisol may be as important as blood levels in determining many aspects of metabolism (2). The details of this system are unimportant and are only relevant for discussion of some of the hormonal modifiers later in the chapter. It is important to note that acute pulses of cortisol are generally beneficial and important to help the body adapt to stress over time while chronically elevated cortisol levels are bad. As well, while chronically elevated cortisol is extremely damaging to the body, having too little produced is equally bad. It should be noted that at the extremes this shows up in Addison's disease (where the body can't mount a stress response or cortisol release at all) or Cushing's disease (where cortisol is chronically elevated). People with Addison's may pass out simply standing up as their bodies can't raise blood pressure adequately and other problems can be life threatening. Cushing's patients are in a state of chronically elevated cortisol with all that implies. While disease states, there are situations that occur in response to stress that are somewhat similar but to a much lesser degree. For example, chronic stress (such as might be seen in an athlete) can lead to an overactivated HPA axis in which cortisol levels are elevated and also play a major role in the overall control of bodyweight (3). Confusingly, stress may increase or decrease hunger depending on a host of factors including the nature of the stress and the person's psychological profile (discussed below). In the short-term, primarily due to increases in CRH, stress tends to blunt appetite (it may also increase energy expenditure) and this would tend to reduce bodyweight. As expected, chronic stress has the opposite effect. Hunger is increased and leptin resistance seems to develop which is why people eat more in response to stress, especially comfort food (which is high-sugar/high-fat "comfort foods"). Sugar intake reduces cortisol levels which increases the feelings of stress and people may end up self-medicating their stress in this fashion (4). This can lead to a vicious cycle of stress-eating over time. 124 Another way that cortisol can impact on body weight is by causing water retention. Due to similarities in structure, cortisol can bind to the same receptor that would normally bind hormones involved in water regulation such as aldosterone. While cortisol doesn't bind that strongly, there is up to 10,000 times as much cortisol and the effect can be pronounced. In the extreme situation of Cushing's, the condition of chronically elevated cortisol I mentioned, water retention is absolutely enormous. And, to a lesser degree, chronic dieting, overtraining or stress can cause this (starvation edema has been known to occur since the 50's). Falling leptin on a diet allows cortisol to increase further and lowered thyroid levels also cause water retention. This can cause apparent weight loss plateaus and I will discuss this in detail in Chapter 26. Gender Differences in the Response to Stress While all stressors cause a relatively similar stress response (hormonally at least), there are gender differences in how the system works. As I mentioned in Chapter 2, there is a slight difference in the hormonal response between women and men (and here I'm talking about more than just cortisol) but the details of this are beyond the scope of this book. Here I'll only consider how those levels change in response to stress. While it depends on the specific stressor being examined, women show an overall reduced stress response compared to men and this is thought to exist to protect the developing fetus from excessively high cortisol levels (4). This difference manifests itself in that women and men differ in their propensity for specific stress-related diseases. Women are more likely to suffer auto-immune diseases (probably contributing to their higher likelihood of developing Hashimoto's, an autoimmune thyroid disease); contributing to this is the fact that women have a stronger immune system than men (6). Women are also more likely to suffer from depression (often related to cortisol) with this increasing with age (7). In contrast, men are more likely to have problems with heart disease or infectious disease due to a generally heightened stress response and poorer innate immune system function. Certainly there are other issues contributing to the above differences in disease risk (i.e. women vs. men's fat patterning) but it does appear that women and men's HPA operate differently (8). Of perhaps more interest is the fact that the feedback inhibition loop I described above works more poorly in women than in men (9); this may be part of why women are more likely to suffer depression. Classically the stress response has been thought of as the fight or flight response and certainly this is true when looking at men's hormonal and behavioral response to most stressors. In contrast, it's now thought that women's stress response triggers more of a tend and befriend response (9). Tending here refers caring for/protecting children while befriending refers to women's tendency to form social bonds with other women as this decreases their stress levels. Subjectively, women seem to experience stress more, that is they report things as being more stressful. Women also appear to report stress more frequently although this seems to be mostly due to men reporting stress less frequently (10). Technical reasons related to how cortisol is measured, it's unclear if these subjective reports show up in an increased cortisol response. Or more importance than the subjective reports are the actual changes in cortisol and it's clear that women and men show relatively greater or lesser stress responses compared to one another although this depends heavily on what type of stress is being examined (10). In response to heat and cold stress, for example, women show a larger stress response, releasing more cortisol. Depending on the scope of this book, women also show an increased cortisol release in response to certain drug challenges (i.e. Naloxone). In other cases, women may release similar amounts of cortisol in response to exercise and diet. It's when the topic of psychological stress is examined that the topic becomes more complicated. First and foremost, men have a stress response in anticipation of psychological stressors while women show no increase or even a decrease. This makes some logical sense within the context of what I described above regarding the purpose of the stress response. A man's stress system, oriented towards fight or flight, has to gear up prior to either, raising heart rate and mobilizing fuel for energy. A woman's, oriented towards tending and befriending, has no such need. In terms of the actual stress response to psychological stress, it has been classically believed that women always release less cortisol than men. But this turns out to depend on the type of stress being examined. Specifically, women show a larger stress response than men in response to stresses of interpersonal conflict and social rejection while men show a greater response when they have to solve math problems or speak in front of people. The reason for this seems to be related to the difference in purpose of the stress system. Women would be more likely to find social rejection (an inability to befriend) more stressful while men would find an inability to perform a competitive task more stressful. 125 There are other gender based differences in cortisol release that can play a role here. One is that, even when women release less cortisol than men, they release it more quickly, causing blood levels to reach a higher peak. In women this can cause more than 20% more cortisol to be released and the lower amount of cortisol not always stays elevated for a longer duration. This makes any absolute type of stress a relatively longer lived stress for a woman than a man. I'd emphasize that how relatively here, it's not as if a woman will maintain a day-long cortisol response to something that a man might only show an hour response to. But on average, her cortisol levels will remain elevated for a longer duration than his. Adding to this is the fact that the feedback inhibition loop I described above works more poorly in women than men and this might contribute to women's slower recovery after being exposed to stress. That is, when cortisol levels are elevated for some reason, women's bodies are generally poorer at preventing further cortisol release. In response to stress, a woman's cortisol levels may continue to rise and rise while a man's generally will not. There are individual differences here as some women are relatively better than others in terms of how well or poorly feedback inhibition works: when their cortisol is elevated, further production is inhibited. But overall women show decreased feedback inhibition compared to men. I'll discuss the implications of this later in the chapter. Finally, and while not represented by any research that I am aware of, it seems possible that women may face more total overall stress (a higher allostatic load) than men. Whether or not this was true historically, in the modern world women either expect (or are expected) to work or simply have to do so due to the requirements of a two-income household. To that they may have obligations or expectations to take care of the home and children while maintaining personal relationships. Within the context of this book, to that we might add general societal pressures on appearance and pressure on women in both the general public and among female athletes. The latter is even true for those athletes not involved in sports emphasizing thinness often wear revealing outfits with the reality that women of all walks of life are judged on their appearance in a way that men rarely are. Women are far more likely to show higher levels of dietary restraint than men, and this all adds up to a high allostatic load and effectively pre-stressed psychological profile before diet and/or exercise are added to the situation. The Impact of Estrogen and Progesterone With the above in mind, I want to look at the hormonal basis behind them. The gender differences in the stress response, there is also a reason behind them. Certainly some of it may be genetic and/or occur during development but, as always, the differences in reproductive hormones are likely to play a role. Let me say up front that this topic gets extremely complicated very quickly as hormones can be having an effect on the hypothalamus, pituitary gland or adrenal gland. In many cases, effects are seen at more than one place and determining how the system works is no simple matter. Rather than attempting to explain all the potential places where reproductive hormones might have any impact, I want to focus on what seems to be the primary consequence which is a woman's relatively poorer feedback inhibition and how it is impacted. As always, estrogen and progesterone are key players. In addition to estrogen, I want to note the importance of testosterone determining a man's response. The data on testosterone's effects are all over the place and, as they are not really relevant to this book, I won't even try to describe them. Focusing on women, estrogen appears to play a primary role in determining women's greater stress response, at least certain types of stressors. Supporting this is that giving estrogen to men increases their stress response to psychosocial stress to levels similar to that seen in women (11). There is some indication that it is the lack of estrogen, rather than the presence of testosterone that is responsible for men's greater anticipatory stress response. This doesn't explain the similar stress response to exercise for both women and men, however. Clearly there are differences in purely psychological and physiological stress so this may not be that surprising even if the mechanisms behind it are not known. The role of progesterone on the stress response is unclear in terms of whether it has an effect in the first place and what those effects might be. Observationally, women's stress response to exercise is increased during the luteal phase (12). As well, women's response to the types of psychological stress that they generally have a larger response to in general is elevated to be similar to that of men during the luteal phase (13). So progesterone is having some effect although whether it's a direct effect or by blocking the effects of estrogen is unclear. Women's already less responsive feedback inhibition loop is further impaired during the luteal phase compared to the follicular and a general conclusion would be that women's overall stress response will be elevated in during the luteal phase (and possibly in any progesterone-like hormonal state). 126 The Effects of Hormonal Modifiers Looking at the other hormonal modifiers, I mentioned in the previous chapter that cortisol levels are elevated in women with menstrual cycle dysfunction and that the increase becomes greater with increasing levels of menstrual cycle dysfunction. In amenorrhea, cortisol may be elevated 10-20% above normal levels but levels can be doubled in women with full blown anorexia (14); this appears to be due to an impaired feedback inhibition loop. Essentially, these women's bodies are no longer able to shut off cortisol release effectively. They also have a larger cortisol response to exercise than women who have not lost their menstrual cycle which only compounds the problem. Research on hormonal birth control (BC) is somewhat inconclusive. One study cited above found that women using combined birth control had a lower cortisol response to exercise than women in the follicular phase who had a lower response than women in the luteal phase. It appears that the ethinyl estradiol component increases levels of cortisol binding protein in the liver. This means that, even if the same amount of cortisol is released, less free cortisol will be present and the overall effect will be lessened. But not all studies find this, possibly due to differences in study design or the type of BC used. There simply isn't enough research to draw a conclusive picture. Women with PCOS/elevated testosterone show a hyperactive HPA response to stress and this appears to be due to negative changes in the metabolism of cortisol within fat cells that I mentioned above (15). This altered fat metabolism of cortisol may be part of what is maintaining the hyperandrogenic PCOS woman's increased output of adrenal androgens, further contributing to the problem. Women with more visceral fat (as seen in PCOS and obesity) tend to show an overreactive stress response and this is seen very clearly in the case of women who develop abdominal, but not lower body, obesity (16). The entire system becomes dysregulated due to the accumulation of body fat and the increase in adrenal output is part of what elevates obese women's testosterone levels. This can become a self-perpetuating cycle due to the way that cortisol can impact on bodyweight and food intake. With aging, the overall stress response in both genders increases but this increase is roughly three times higher in women then in men (17). As always this suggests that the decrease in estrogen, progesterone or both at menopause is responsible. In response to hormone replacement therapy (HRT) while estrogen-only therapy increases the cortisol response to stress, a combination of estrogen and progesterone decreases it (18). Regular exercise also reduces the stress response here (19). Individual Differences In addition to the gender differences that exist in response to stress, there are also large individual differences in how people will or will not respond to a stressful situation. Readers may be familiar with the terms Type-A or Type-B personalities and, even if they are not, everyone has known someone who will have an enormous (over)reaction to the smallest of situational stresses while others remain calm in the face of disaster. Even when faced with an identical stress, two people may perceive it differently, showing a completely distinct stress and cortisol response. Adding to this is are differences in how two people may recover from a stressful situation after it has ended. After a given stress, one person may move past it quickly while the other remains stressed out over it hours (or days) later. The latter individual will be chronically over-activating their HPA and generating what amounts to a chronically elevated stress response even if the stress itself was relatively short-term in nature. So consider two people who are in a slow moving line at a store. One may be seething with rage over the speed while another is completely calm about it. Hours later, the first may still be angry about the situation, maintaining an overactive stress and cortisol response long after it was necessary, while the second was barely affected at all. Effectively, some people show greater stress reactivity compared to others, both reacting more strongly to stress to begin with and possibly maintaining that stress response for longer after the stress has ended (recall that this, along with an inability to cope with daily hassle was found in a group of women suffering psychologically caused amenorrhea). While some of that behavior may be learned, it's clear that there is a also a biological hardwiring during fetal development with high levels of cortisol exposure causing an overactive HPA later in life. In response to any stressor, the stress response will be greater and potentially longer, also predisposing the person to certain stress-related diseases. Even here there is a gender difference with women being more sensitive to this effect (20). That is, when exposed to elevated cortisol levels in utero, women are more likely to develop an overactive HPA later in life (this can also predispose them to certain stress related diseases). While clearly part of a person's underlying biology when it is present, it's equally clear that coping skills to reduce stress can help enormously here (recall that CBT restored menstrual cycle function in psychologically stressed women). While not studied to my 127 knowledge, this could explain why some women show a poorer feedback inhibition loop to stress than others. That is, perhaps a generally overactive HPA is part of why their bodies continue to produce cortisol in response to additional stress (whether real or perceived) when output should be inhibited. Adding to the above is how a given person perceives a given stressor. So consider something like speaking in front of people (a commonly used stress test as mentioned above). Someone who is outgoing and loves attention might not see it as a stress at all while an introvert or someone who is terrified of embarrassing themselves might see it as an enormous stress, showing an elevated cortisol response. Similarly, while one person might find a massage calming and stress releasing, someone who is uncomfortable with being touched by a relative stranger might show a tremendous stress response. While this is most likely to apply to more social or psychological stresses, it could also impact on the stress response to diet or exercise. Physiologically speaking, any specific workout shouldn't have an enormous impact on the stress response seen but there could be a psychological aspect as well with some people perceiving it as more stressful in a psychological sense. This could be related to any number factors including beginners feeling embarrassed about being in a public gym (or one filled with more advanced people). Fear of not knowing how to do a given exercise correctly or that the workout will be beyond them could contribute to this. The same can hold for diet in that, while any similar diet should be broadly physiologically the same, people can perceive how restrictive or difficult it might be Playing into this is the concept I discussed previously and will discuss more below which is dietary restraint. This refers to a general preoccupation (or obsession) on bodyweight and food intake which can cause some women to show elevated cortisol levels even before they begin their diet. I'll discuss this more below. I have to think that these individual differences, whether biological or psychological, are contributing to the relative robustness (or fragility) of women's reproductive systems and/or hypothalamic function in some way. Women who may have an overactive HPA, or a psychological profile causing them to be more pre-stressed, who are exposed to more total stress via diet and exercise, who have an excessive cortisol response to stress and/or don't recover from that stress as rapidly will show an exaggerated and chronic stress/cortisol response compared to those women who do not. This combination of underlying physiology and psychological profile only adds to the stressful effects of diet and exercise, discussed next. The Stress of Dieting While stress can clearly occur at any time, regardless of the specific goal, I want to look at how stress relates specifically to the dieting and fat loss process. Or more accurately, how the fat loss process itself can generate stress and increase cortisol levels. Just as in the previous chapter, I will be focusing on exercise, diet and psychological stresses in terms of their individual and combined effects. However, while the focus in the last chapter was on menstrual cycle dysfunction per se, here I will focus primarily on cortisol levels (note that there is overlap between these two issues as an increase in cortisol is part of the hormonal profile of menstrual cycle dysfunction). Exercise and Diet Related Stress Looking first at exercise, the impact on cortisol is related both to the type and intensity of activity being done. In general, low intensity exercise does not cause a significant stress response unless it is performed for multiple hours at a time. This tends to be uncommon outside of female endurance athletes such as runners or cyclists (who typically consume carbohydrates and fluids during exercise which offsets much of the hormonal response) although there are female dieters who perform hours of aerobic activity per day. As I've mentioned, women do need to perform somewhat more (25-30% or so) aerobic activity to burn the same numbers of calories as a male but many take this to an unhealthy extreme. In contrast high-intensity exercise, whether aerobic or HIIT, causes a universally large cortisol response regardless of duration and this can cause enormous problems if excessive amounts are done (21). This has been a trend in recent years as dieters have attempted to replace the more traditional lower intensity aerobic activity with nothing but high-intensity activities. While some may get away with this, it's important to realize that even high-performance athletes rarely perform truly high-intensity training more than two to three times per week (and even then only moderate amounts are done). Attempting to do more than this in and of itself will cause a chronic over-activation of the HPA and increase in cortisol and this can burn people out even if they are eating enough. When it is done while dieting and restricting calories, it can be a disaster. I'd note that a single day of rest from training allows cortisol to return to normal levels and this is an importance consideration for those athletes or dieters who are attempting to exercise (especially at high-intensities) on a daily basis. 128 An additional concern here is that women, in contrast to men, may continue to produce high levels of cortisol during high-intensity exercise even if it is elevated (i.e. due to dieting or psychological stress) to begin with (22). This is due to the relatively less effective feedback inhibition loop I mentioned but is also a place where there is a great deal of individual variability. Specifically, some women will continue to produce cortisol when it is already elevated while others will not. The former type of women may be destroyed by the types of training and dieting approaches that the latter frequently "get away" with. As I described in the last chapter, the effect of large amounts of high-intensity exercise can be magnified if it is started immediately (23).

and athletes in terms of how they might add to the stress of exercise and/or dieting itself. I do want to reiterate that all life stressors, whether related to diet and training or not, are relevant in terms of the total stress seen. A diet or training situation that could readily be handled under one set of conditions can become excessive if other life stresses increase. Problems with interpersonal relationships, a lack of family or friend support, death in the family, work or school stress, etc. all play a role in whether or not a given amount of other stresses can be handled or not. In this vein, individuals experiencing high levels of stress make poorer strength gains and recover more slowly from resistance training (31,32). For the general dieting or exerciser, this could include self-imposed psychological stress worrying about actually adhering to their program. Dieting and the goal of weight and fat loss can also act as its own type of psychological stressor. Even before true hunger sets in, there is often a feeling of deprivation and the sense that the person is restricted in what they can eat. All of the above can be exacerbated in those women who are unfamiliar with the concept of body composition or issues relating to the scale and daily or weekly fluctuations in weight. Unrealistic expectations about weight loss rates adds to this as dieters feel as if the effort they are putting towards their goal is not generating meaningful results. All of this adds to the already existent psychological stress load. For athletes of any sort, there may be additional issues contributing. Adherence to training is rarely an issue although performance pressure, anxiety, coping with injuries or interpersonal problems with coaches or teammates (in team sports) can all add to the overall stress load. Physique athletes often have their own set of issues, somewhat unique to their sport. Many attempt to follow an extremely rigid schedule 129 of eating and being unable to follow it can add to their stress levels. In many cases, they will avoid social events, friends and family for no other reason than to adhere to their meal plan. With appearance as the end metric, a perceived lack of progress or not being on schedule to reach their goal in time for competition adds to their stress levels enormously. The final stages of contest dieting can be enormously psychologically stressful as the diet and training program must be maintained in the face of enormous physiological adaptations and fatigue or the feeling that their appearance is not what it needs to be. In both cases, there can be additional stressors if the dieter or athlete is not supported by their family or friends who may not understand the importance of the diet (or its requirements) or exercise program. They may encourage the individual to skip training sessions or break their diet "just a little bit", etc. At home and tying into my suggestion above that women may face more total stresses in the modern world, female dieters are often tasked with making two or even three meals in some cases: one for themselves, one for their partner and one for their children (if present). In many cases, this may occur after a full day of work and the day's training session and the amount of stress generated can be significant. In addition to all of the above are two other factors. The first is the cluster of personality traits such as high stress reactivity, poor coping skills, an inability to handle daily hassles, perfectionism and others that contribute to stress and raised cortisol levels and which may cause FHA in and of themselves. The second is the issue of dietary restraint which I have mentioned several times already in the book. More common in women than men, this refers to a situation where someone is chronically concerned with their body weight and overall food intake. This should not be considered synonymous with dieting as it is possible to be concerned with what is being eaten without trying to lose weight. I'd mention again that restraint can be rigid or flexible and here I am focusing only on rigid restraint. Women with high dietary restraint show an overactive HPA/stress response and elevated cortisol levels to begin with due to their extreme concern over what they eat (33,34). The same high dietary restraint is also related to menstrual cycle dysfunction, lowered bone density, and stress fractures in runners and at least part of this is mediated through elevated cortisol levels (35-37). Somewhat surprisingly, people with high levels of rigid dietary restraint tend to weigh more than those who show lower levels of restraint. This is explained by the fact that restrained eaters are the ones who often overeat in response to various types of stress (38). This is called disinhibition and the degree of disinhibition seen in any given person is highly related to weight gain or weight regain (39). This is due to the fact that the foods most commonly eaten when people become disinhibited are highcalorie, high-sugar, high-fat comfort foods. This may cause weight gain and this can put the person into a vicious cycle where weight gain due to disinhibition may cause the person to become that much more restrained and focused on their eating. The next stress can cause disinhibition and overeating to occur and you can see how this cycle can become self-perpetuating. Repeated cycles of this cause a gain in body fat and in those women who start gaining abdominal/visceral fat, the stress system becomes that much more over-activated, generating a chronic cortisol response with everything that implies. All of the above points to the fact that a variety of psychological stresses, ranging from inherent psychological profile to life stress to various potential concerns about eating and training can add to the biological stress and cortisol response to dieting and exercise. What specific stresses are present may depend somewhat on the population being discussed but some stresses are likely to be present . In all cases, the combination of dieting, intense training and psychological stresses will contribute and add up to determine the total amount of stress present and whether or not it overwhelms any given individual. Stress and Amenorrhea In the previous chapter, I examined the various causes of menstrual cycle dysfunction with the general conclusion that a low Energy Availability (EA) is the primary cause with other factors such as psychological stress, genetics, reproductive age and possibly BF% modulating that. Focusing only on the first two it's been traditional to separate energetic and psychological stresses in terms of their effect on the reproductive system but this is a mistake (40). The two factors contribute to the overall stress on the system and may interact in complex ways. For example, someone might choose to exercise for stress reduction and end up imposing an energetic stress on their body. Alternately someone might restrict calories, causing them some degree of mental stress over feeling deprived in terms of what they can eat. At the extreme, Ann Loucks, who developed the EA concept, has suggested that all stress, including psychological stress, is energetic to begin with (41). She contends that even psychological stress is generating a lowered EA state by reducing food intake or increasing energy expenditure. At the other extreme are those that question whether athletic amenorrhea (FHA found specifically in athletes) should be 130 considered an energetic or psychogenic (i.e. psychological) challenge (42). That is, they suggest that the typical cluster of personality traits that is seen in women, coupled with the additional stresses seen in athletes involving training, travel, team dynamics, etc. may be the cause of FHA in these women rather than low EA itself. Given that behavioral therapy can be incredibly effective in restoring menstruation in these women, there seems to be good evidence that this is, or at least can be the case. There are also anecdotal reports of women who restored their EA to sufficient levels while gaining weight and fat who did not resume normal menstruation until they addressed their psychological stresses. In a practical sense, there will always be some degree of energetic and psychological stress present when someone is seeking fat loss and it will only be a matter of degrees as to which is potentially more or less responsible. This is especially true for a concept that I want to introduce, an extension of the paper on psychogenic stress above, that I call the psychogenically stressed dieter. This refers to the female who is starting with some degree of psychological pre-stress before adding to that with calorie restriction or exercise. This could take many forms. At lesser levels it might simply be generally high-stress reactivity or a poor ability to cope with daily hassles. In the physique sports, and potentially any sport with a component of appearance, narcissism, perfectionism and dependence on external validation is likely to be present. All athletes have to deal with the overall stress of their training, dieting, travel, etc. At the very extreme, overt eating disorders may be present and, even when they are not, subclinical disorders may be. Even if only extremely rigid dieting attitudes are in place, not only does this raise cortisol levels by 10% above normal to begin with, it can actually lead to eating disorders over time (43). Highly related to this is a relatively "new" eating disorder called orthorexia which would seem to represent the far extreme of rigid eating attitudes. Orthorexia describes an obsessive preoccupation with the relative goodness/healthiness of foods which, contradictorily, may lead them to become less healthy due to the elimination of nutrient dense foods that are deemed unhealthy for some reason. The orthorexic attitude may be best represented by the idea of "clean" eating. This approach to diet is found in the physique community although it has leaked out into the general public and the idea is that some foods (typically unprocessed foods) are clean while others (refined foods) are not. Tying this back into the psychologically stressed dieter, orthorexia has even been linked to both narcissism and perfectionism and I think this explains the high degree of it found in certain subcultures (44). Like the orthorexic, clean eaters become obsessed about what is or is not a clean food with the words themselves connoting a moralistic or hygiene aspect of eating. And while they may be meticulous about their food intake under certain conditions, they can show an enormous amount of disinhibition: the slightest deviation from clean eating lead to enormous binges which are then followed by an even more intense focus on restriction going forwards (binges may also be dealt with by performing hours of exercise which takes it into the realm of exercise bulimia). These dieters often engage in many other somewhat pathological behaviors such as avoiding social events or interactions for fear that they might break their diet in the most minor way. The Psychogenically Stressed Dieter Regardless of the specific cluster of factors present, the psychogenically stressed dieter is one that is starting with high levels of chronic psychological (or life) stress prior to starting their fat loss diet or exercise program. This means that their cortisol levels are elevated to begin with and that, regardless of the diet or exercise approach that they take, they are likely to show a greater stress response to it overall. I have typically referred to this group of women (and make no mistake, it is also seen in male dieters) as being tightly wound and they are often found in online forums using all capitals with exclamation points about their lack of daily or weekly weight loss (or any loss at all). You can almost "hear" the tension and stress in their typing. In my experience, they are often the ones that are drawn to more extreme dietary or exercise approaches to begin with and this is, more often than not, absolutely the worst approach that they could possibly undertake. This may include very aggressive diets in terms of the degree of calorie restriction (which may or may not be coupled with a very rigid approach to dieting) along with excessive amounts and/or intensities of exercise, often done with no build up or progression (i.e. they will reduce calories to very low levels immediately while starting a multi-hour per day exercise program). Not only does this often take them below the critical EA threshold immediately (with the hormonal and metabolic effects that implies), it compounds their overall stress levels, elevating cortisol enormously and creating a chronic stress situation on both an energetic and psychological level. This creates a perfect storm of problems. 131 The Cycle of Chronic Stress and Dieting Even in the case where a female is not psychogenically stressed prior to beginning the diet, any combination of diet and exercise that causes a chronic stress response to occur is a problem due to the constant elevation of cortisol. Here I am not focusing so much on the metabolic or physiological effects but the fact that chronically elevated cortisol, in addition to many other factors, can cause water retention. In addition to causing the person to look puffy or watery, this can serve to mask true weight or fat loss, especially in the short-term (days or weeks). The diet may be working perfectly well (in the sense that fat loss is occurring) but it is not becoming apparent on the scale or in terms of appearance. This may be coupled with not understanding the difference between weight loss and body composition or unrealistic expectations about the amount or rate of losses. Those who expect the losses to occur linearly may be doubly stressed as fat and weight loss tend to occur in stops and starts to begin with (I'll talk about plateaus and other potential causes in Chapter 26). And there can be two potentially detrimental cycles that occur. The first is that the dieter assumes that they should intensify their efforts. They reduce calories that much further or increase their activity that much more. Rather than helping the situation, it makes it worse. EA drops even further, more metabolic and hormonal adaptations occur, exhaustion and fatigue reduces NEAT or the dieter may become injured (often working through it). Cortisol rises that much more which causes further water retention. Which causes the dieter to work and stress over the lack of results to that much of a greater degree. More and longer stalls means more restriction which prolongs the stall. Eventually a limit is reached and calories can only be reduced so far and activity increased so far. Either the dieter attempts to maintain this for extended periods, usually in the absence of good results, or they become injured or crack, abandoning the diet and exercise program completely. Their calorie intake will go up, their activity down (eliminating the low EA) while their mental stress over the lack of results may drop. And as frequently as not, this causes body weight to drop and appearance to improve within a few days as the diet and exercise related stress dissipates, cortisol drops and water is lost. At this point the diet is often resumed but, rather than realize that giving their body a break from the diet was good, they return to the same extreme approach, causing the same cluster of problems to occur again. The second cycle that can occur is almost the opposite of the first but can cause the same type of issue. This cycle is mainly seen in the extremely rigid or orthorexic eater, the type that can become easily disinhibited. They will submit themselves to extreme restriction and high levels of activity to the greatest degree possible but at any sense of a slip-up they will abandon the diet and go on enormous high-calorie, high-fat food binges. In smaller women, this can be sufficient to offset the dietary restriction during the week, slowing or eliminating fat loss. Even when it doesn't, the frequent guilt and shame about having broken the diet leads to even more extreme degrees of restriction which propagates the same cycle of stress and cortisol elevation with repeated cycles of restriction and disinhibition based binge-eating. Shockingly, it is often impossible to convince women engaging in either of the types of pathological dieting practices to try a different approach, even in the complete absence of results. Better dieting practices and strategies, that I will present throughout this book fall on deaf ears since they go against how these women think fat loss is best achieved. Some of this ties in with the psychological factors that may be present but at least some of it is due to the fact that successful individuals often do get away with these types of approaches. This may be a situation where these women have a less over-reactive HPA or show better feedback inhibition. While not often talked about, there is also the reality of drug use at the elite level with one effect of anabolic steroids being to reduce cortisol signaling. Regardless of the reason, even if extreme diet and training approaches work for some women, they do not seem to work for the majority. Of more importance, if they are not working for any individual woman, they should be changed. What the above hopefully points to is not only the need for many, if not most women, to take a different approach to dieting, training and fat loss overall but to find ways to decrease their overall stress. In some cases, full-blown therapy may be required but other stress reduction strategies are also useful. Meditation, massage and yoga are all possibilities here with asana yoga having been found to lower cortisol levels (44a). For some years I have "jokingly" suggested that the chronically stressed woman get drunk, stoned or laid. This might depend on whether or not the sex is satisfying but a hot bath, candles, a glass of wine and some personal time might be the best solution here. Ideally avoiding excessive work, life or relationship stress would be a good thing but this isn't always realistic. In the modern world, it's nearly impossible to avoid all forms of stress but the reality is that chronic stress of either a diet, exercise of psychological nature will eventually takes its toll on the body. If dieters or athletes stress the system too hard for too long, very real problems can occur. 132 When Chronic Stress Goes Wrong When stress was first conceptualized, three phases were described which were alarm, resistance and exhaustion. Alarm was the initial stress response, resistance the positive adaptation that occurred if recovery were allowed and exhaustion the state if the stress was unrelenting and the body was not able to adapt. Here I am focusing on the exhaustion stage and want to look at two primary hormonal changes that occur. The first I'll mention only briefly which is an elevation of catecholamine (adrenaline/noradrenaline) levels even in the resting state which causes people to feel generally anxious or overstimulated. The second and far more important change has to do with the levels of cortisol itself which switch from being chronically elevated to being too low, called hypocortisolism. Normally cortisol shows a peak in the morning (this helps people to wake up) with a decrease over the course of the day. When hypocortisolism develops, this pattern is lost with no morning cortisol increase and lower overall levels the rest of the day. In addition the body loses its ability to increase cortisol levels in response to stress, reducing or eliminating their ability to cope with that stress. Conceptually similar to Addison's disease, sometimes called primary adrenal insufficiency, where no stress response can be mounted, this only reduces someone's ability to respond to stress. This is referred to as secondary adrenal insufficiency. The Consequences of Hypocortisolism Hypocortisolism has been found to occur in a number of different situations, all of which share the commonality of being related to or caused by chronically high levels of stress. One of these is burnout, due to work stress, which is characterized by physical exhaustion, depression and chronic inflammation. The risk of autoimmune disease may also increase. Burnout has also been related to an increased risk of heart disease, depression and immune system dysfunction (45). Possibly related to burnout are diseases such as Post-Traumatic Stress Disorder (PTSD), fibromyalgia and chronic fatigue syndrome (CFS), all of which can be related to chronic stress and all of which show markedly similar symptoms of fatigue and exhaustion during the day (46). This fatigue is often coupled with sleep impairment, most likely due to the elevations in catecholamine levels (people are exhausted but overstimulated). This sets up a vicious cycle, difficult to break, where stress leads to poor sleep which leads to an inability to cope with stress. While not studied in the context of dieting itself, athletes may experience similar types of changes which are referred to as overtraining, the consequence of a long-term imbalance between the training being done and overall recovery (i.e. sufficient food intake, sleep, days off). A chronic stress situation, the earliest stages of this imbalance can cause performance to begin to decrease and here another vicious cycle occurs as athletes begin training harder, worsening the problem. If this continues for extended periods, eventually overtraining will occur, marked by lethargy, a depressed immune system, a lack of motivation to train and mood changes such as depression. Muscles may feel chronically heavy or inflamed and athletes in some sports find it impossible to raise their heart rate or perform and this is a clear indication that the body is no longer able to mount a stress response. The specific cause of overtraining is currently unknown but it is clearly a response to chronic stress and represents a type of adrenal insufficiency (47,48). At the extremes of true overtraining, athletes may take months to fully recover completely and there are anecdotal reports that some athletes never do. Physique competitors or those who have dieted down to low levels of body fat over report symptoms of joint pain, exhaustion and muscular fatigue not only during their diet but for weeks or months after the competition and diet are over. This is especially true if they use many of the common extreme approaches to dieting. In the aftermath, fatigue, lethargy, depression, chronic inflammation, impaired immune system function and a host of others all occur and this comes along with low morning cortisol, low levels of overall cortisol and a general inability to increase cortisol in response to stress. Adrenal Fatigue or Adrenal Adaptation? I expect many readers will recall a diagnosis that was popular years ago called adrenal fatigue. Sometimes called adrenal depletion, the idea was that the adrenal glands become fatigue or exhausted from chronic stress. A variety of mechanisms behind it were proposed, the list of side effects was so vague as to apply to everyone and endless supplements were sold with not a single person ever reporting being cured to my knowledge. This is likely due to the fact that adrenal fatigue, as it was popularly conceptualized, does not exist (49). In no way does the adrenal gland or any other part of the HPA become fatigued in the common sense of the word. The concept of adrenal depletion, that the gland was depleted of compounds needed to produce cortisol was equally incorrect as chemical stimulation of the gland cause cortisol to be produced without issue. This meant that any problems were occurring in other parts of the system. 133 Specifically changes were found to be occurring in the hormonal receptors in the hypothalamus, pituitary and adrenal gland. At the level of the hypothalamus, the feedback inhibition loop becomes hypersensitive such that even small amounts of cortisol inhibit the release of any more. The pituitary gland may also become resistant to the stimulating effects of the hypothalamus. Both of these lead to reduced cortisol levels both at rest and in response to stress but this does not indicate that any part of the system is either fatigued or depleted. Rather the system has adapted generating what is better conceptualized as adaptive hypocortisolism (50). While these changes clearly have many negative effects, it is still positive in the sense that it is meant to protect the body from the ravages of chronic stress and elevated cortisol (51). Treating and Preventing Hypocortisolism Whether or not the reduction in cortisol is seen as a positive adaptation or negative consequence, the fact is that the effects can be profoundly negative for both health and function. This is worsened by the fact that there are at least some indication that the problems are permanent. Perhaps more accurately, I am not aware of any research showing that the effects reverse themselves. As I mentioned above, I'm unaware of anyone ever actually being cured of "adrenal fatigue". Certainly much of what was recommended in this regard such as reducing stress, reducing the amount and intensity of exercise along with ensuring adequate nutrient and a healthy diet was good advice but the situation never seemed to reverse in any meaningful time frame. In the extreme versions of hypocortisolism such as PTSD, fibromyalgia and chronic fatigue, only treatment is available and, so far as I can tell, there's no reason to expect the adaptive hypocortisolism in response to chronic stress to be any different. The typical first line of treatment is to use synthetic cortisol (i.e. hydrocortisone). The goal here is to raise levels of cortisol to where they should be at specific times of the day to improve energy, mood, etc. This generally requires dosing multiple times per day and determining an optimal intake requires multiple cortisol measurements. Generally 4 or more are taken through the day either by blood or saliva sample. The only non-medication treatment I'm aware of that has been studied is DHEA, one of the adrenal androgens (that I will discuss more in Chapter 24). Another common change in hormone levels is a shift in the ratio of DHEA (the active form) and DHEA-S (the inactive form) and supplementation is aimed at normalizing that ratio. Studies have found that a dose of 25-50 mg of DHEA taken in the morning helps to alleviate at least some of the symptoms of hypocortisolism (52). However, as an androgen, DHEA can also cause many of the side effects seen in women with elevated testosterone such as oily skin, acne, increased body hair, etc. Ideally, testing of DHEA levels should be done so that a proper dose can be determined. Beyond those two treatments, adaptive hypocortisolism appears to be irreversible and this means that preventing it from occurring in the first place should be the goal. In the most general sense, this means doing one's best to avoid the chronically high stress levels that are so common in the modern world. If possible, this can be combined with other approaches that reduce stress. Within the context of this book, this would mean avoiding pathological dieting practices. This includes extreme calorie deficits unless they are modified in ways I will describe later in the book. Avoiding both excessive amounts of training along with not attempting to perform too much high-intensity training is also important. In all cases, the amount of exercise should be raised gradually to give the body time to adapt. Understanding that weight and fat loss is rarely linear, the limitations of body weight, the potential for water retention to cause short-term plateaus is equally important. If a great deal of psychogenic stress is present, the above becomes even more important. In some situations, therapy may be required. Even if all of the above is avoided, problems can still begin, especially for the Category 1 dieter going to the extremes of low BF%. Dieters must be on the alert for the first signs of problems. A constant muscular inflammation (the arms and legs may feel "heavy"), a reduction in the motivation to train, a decrease in performance itself, extreme fatigue or low blood pressure upon standing or a constant need to consume caffeine simply to maintain function are all signs that problems may be starting. Certainly there are times when athletes or dieters on a time scale must work through this but often it takes no more than a few days of reduced training with increased calories to reverse the problem. Ignoring the problem, as so many do, simply digs the hole deeper until the system shuts down, possibly permanently. If caught in the earliest stages of development, it is eminently reversible. Finally, for the majority of women, there are objectively ways to go about the process of dieting and fat loss in terms of how the diet is set up, nutrient intake, specific dietary strategies, the implementation of exercise, etc. These can both avoid the issues above as well as address all of the specific physiological issues I described earlier in this book. Because while there are many issues that women have to contend with, they can be addressed or even fixed. That discussion will start with the next chapter. 134 Chapter 14: Fixing the Problems In the previous 13 chapters of this book, I have covered a large amount of information which should make it clear that women's physiologies are distinct from men's in a number of ways related to fat loss, fat gain and altering body composition. While potentially disheartening, I would not have bothered to cover that information or write this book if there were not solutions to these issues. Some of those have already been addressed (i.e. the chapters on body composition should hopefully point out potential pitfalls with focusing on short-term changes in scale weight only) and I won't repeat those here. The specifics of diet set up, nutrient intake, etc. will be covered in detail in later chapters and will not be discussed either. Rather, in this chapter I want to look at solutions to the major physiological issues. A variety of different topics will be discussed including avoiding excessive fat gain (primarily important for women who are already lean), making gradual versus extreme changes, the Pre-diet phase, moderate versus aggressive dieting approaches, limiting hormonal disruption and enhancing fat loss. Many of my suggestions likely will seem counterintuitive or illogical compared to how women think dieting and fat loss should be approached. However, hopefully at this point it's clear that many of those approaches, whether chosen by or recommended to women, are not only ineffective but potentially damaging. Seemingly illogical or not, these strategies described are meant to take a woman's specific physiology into account and work with it rather than against it, something that must be done for any approach aimed at women (1). Much of the information in this chapter will be aimed primarily at Category 1 women (recall that leaner/normal weight women have far more problems overall) although much of it will apply to all women. Limiting Excessive Fat Gains In the most general sense, the ideal way to "fix" the problems that women have in terms of fat loss would be to avoid gaining fat in the first place. This would not only eliminate the need to diet to begin with (except for those women who reduce their body fat to very low levels) but also avoid the problems that come along with fat gain such as increased lower body fat or fat cell number. While theoretically ideal clearly this isn't particularly useful advice to women who have already gained some amount of body fat already. For them this section only becomes particularly relevant after they have first lost fat to begin with. Primarily here I want to focus on those athletic populations that spend at least some part of their year attempting to gain strength/power or increase their muscle size. Since this is best accomplished by combining proper training with as slight calorie surplus, it's common for at least some amount of fat to be gained. In addition to the many other reasons to avoid fat gain, as those groups usually need to reduce their body fat percentage (BF%) for appearance or performance reasons (or to make a weight class) it's important to avoid excessive fat gains. For those athletes who will eventually need to reduce their body fat to very low levels, keeping BF% at reasonable levels enormously reduces dieting time (discussed in Chapter 25). I'd mention that the above tends to not be terribly relevant for endurance athletes as, outside of injuries, it's rare for them to gain excessive amounts of body fat to begin with and they rarely target large scale gains in weight or muscle for their sport (rowing and swimming are potential exceptions). For athletes, certainly the risk of excessive fat gain is lessened by the large amount of training that is being done. Exercise, has what is called a partitioning effect, meaning that incoming nutrients tend to be stored within the muscle instead of in fat cells. High intensity exercise such as resistance training or interval training tends to have an even greater impact in this regard as they deplete carbohydrate stored within the muscle itself. The body prioritizes refilling muscular fuel stores before it stores extra calories as fat and this alone helps to avoid excessive fat gain. But this does not make it impossible as a large enough calorie excess can still cause fat gain even large amounts of training is being done. Even with reasonable surpluses, it's rare to make significant gains in strength, power or muscle mass without some amount of fat gain. The key here is to simply keep the total amount gained limited. This is accomplished by avoiding excessive calorie surpluses or intakes. Muscle mass, strength power and all other aspects of fitness improve relatively slowly and cannot be forced by eating more food. Any excess calorie intake beyond what is required for optimal results simply results an excessive gain in fat which must be lost at some later date (2). While this is true for both women and men, women's lower absolute rates of gain mean that an even smaller surplus in calories will be required. So while a man might be able to gain 2 lbs per month of muscle, a woman will be doing well to gain half of that (a hyperandrogenic PCOS woman might gain slightly more). As I'll show later in the book, one pound of muscle per month requires perhaps 100 calories per day over maintenance. Putting this into perspective, a recent book aimed at women recommended 500 calories per day over maintenance, an intake guaranteed to result in excessive fat gain. 135 In addition to limiting overall calorie surpluses, I'd remind readers that high-calorie/high-fat meals can cause direct fat storage in the thighs, fat that will be more difficult to lose later on. Whether an athlete or not, avoiding this is important for all women. Certainly this can be difficult in the modern world where many social and life events revolve around not only food in general but those types high-calories/high-fat In that situation where a woman knows that she will likely be eating these types of foods, either the total amount should be limited or offset by slight reductions in food intake the rest of the day. Performing an intense workout that day also helps to ensure that incoming calories are shuttled into muscle rather than fat. In addition to keeping a relatively tight control over calorie intake, I would recommend that female athletes (especially those who must diet to the middle of my Category 1 or lower for their sport) never allow their BF% to go above 24% (27% via DEXA). At this point, additional body fat gains are more likely to result in lower body fat gain and possibly increased fat cell number, making future dieting efforts more problematic. The exception to this would be women in sports where excess BF% is not a concern and/or there is no need to diet down. This would include superheavy weight powerlifting, Olympic lifting and some of the throwing events. For all other athletes. as soon as BF% has reached the top of my Category 1, body weight should be stabilized before a short diet is performed to reduce BF% to below that level. The goal here should be to lose only the gained body fat and the combination of proper training and sufficient protein makes this a relatively trivial task. As too low of a BF% makes gaining muscle, strength and power difficult, women should go no lower than 20-22% during this mini-cut. In practice, this means that females trying to gain strength, power or muscle will gradually gain weight (with some fat gain) until they hit 24% body fat, stabilize their weight, diet down briefly to 20-22%, stabilize, gain back up to 24%, etc. Over time, this will allow a gradual increase in performance or muscle mass while avoiding excessive fat gain. It also puts the individual in a good starting place to begin a diet when necessary. Making Moderate vs. Extreme Changes In the chapters on menstrual cycle dysfunction and stress, I looked at how both the combination of extreme calorie deficits and/or excessive amounts or intensity of exercise can cause problems in women, especially if they are not gradually built-up to over time. Cortisol levels may skyrocket (and this is worsened in the psychogenically stressed dieter who is so often drawn to the extremes), energy availability (EA) may drop below the critical threshold immediately and hormonal issues can start within as few as 5 days. This leads into a fairly general principle in terms of the overall benefit of making moderate versus extreme changes. This applies to the initiation of the diet, how it is applied and how changes should be made during the diet as fat loss slows (this will be discussed in detail in Chapter 27 rather than here). Once again, the above primarily applies to the Category 1 and possibly Category 2 dieter while the Category 3 dieter tends to have less overall problems physiologically. However, there can be adherence issues with extreme approaches even in that group. For example, someone coming from a relatively sedentary background who attempts to engage in a large amount of exercise may be too sore, too exhausted or too miserable to continue. At the same time, the Category 3 female with a large amount of fat to lose may find than the results from a more moderate approach are too slow to be rewarding or motivating. A balance between results and adherence must be struck here. The changes here tend to come through diet more than exercise and an extreme approach can be appropriate if implemented correctly. In that vein, the above is not universal for any Category dieter and there may be places where an extreme/aggressive approach may be appropriate. I will address these later in the book but they tend to revolve around relatively short-term diets where the goal is to lose a maximal amount of fat in a 2-4 week span before moving back to either a more moderate diet or maintenance. For example, the Category 1 female who is in a muscle or strength gaining phase may wish to do a short diet to reduce their BF% before returning to maintenance and then resuming their gaining phase. This tends to be in very specific situations and it only for fairly short-term use. This general principle of making more moderate changes really applies to long-term diets or fat loss programs and, with almost no exception, will starting at an extreme level or making extreme changes be anything but detrimental. The Pre-Diet Phase Moving from the fairly general information above to practical application, I want to start by discussing what I will call The Pre-Diet Phase (a similar concept to be used during active dieting will be discussed later in the book). This is a lead-in phase to the formal diet and is structured to allow women to avoid the stresses and physiological shock that can occur when they do too much too soon. The specifics of how the Pre-Diet Phase will be implemented depends on the specific situation and I want to look at each. 136 The Beginning Exerciser/General Dieter I'm going to group these two categories together since there tends to be overlap in the groups. Recall that this group can technically consist of women in any of the three categories and this does impact on how the Pre-Diet Phase should be implemented. For those women who are only looking to improve their general health and fitness (and not targeting fat loss), the Pre-Diet Phase is really more of a Pre-Training phase: a period of 4-8 weeks of gradually increasing exercise training should be done here and I cannot overemphasize the word gradual. Doing too much too soon can cause injury and burn-out in all beginners and long-term adherence tends to be superior when a more gradual approach is taken. If fat loss is the goal, different Categories of dieters will need to take different approaches. Since they don't need to worry about menstrual cycle disruption (and are often limited in the amount of exercise they can realistically perform to begin with), the Category 2/3 dieter can technically begin their diet at the same time as they begin their exercise program. In contrast, the normally cycling Category 1 non-exercising women does have to worry about causing menstrual cycle disruption along with the other problems I've discussed. In this case, a more typical Pre-Diet phase where exercise is gradually brought in (especially resistance training to limit lean body mass loss) with either no change in calorie intake or at most one of the smaller deficits/slower rates of loss I will discuss in Chapter 17 should be done. The Serious Trainee The serious trainee is my terminology for someone who is engaged in a large amount of exercise, typically some mixture of weight training, aerobic and/or HIIT work, but who does not compete in any specific activity. Typically their goals tend to revolve around maintaining their current fitness and/or appearance although specific fat loss may be a goal. Since the serious trainee is typically already performing fairly large amounts of aerobic/HIIT work to begin with, a true Pre-Diet phase is rarely needed. At most calories might be brought to maintenance for 1-2 weeks before beginning the diet. The Physique Athlete Since it diet or become disinhibited this is usually what is happening. Once athletes or dieters generate a low EA state, they typically maintain that for the long-term, generating all of the problems that I have described. But fundamentally there is no reason that this has to be the case. That is, despite how many conceptualize dieting or are instructed to diet, there is no reason that calories cannot vary from day to day or even be brought from below to above the critical EA threshold. Athletes in the physique community have used a similar strategy, typically called calorie or carb-cycling, for years and I will discuss these types of patterns in Chapter 23. For now I just want to look at the fundamental strategy and how it has the potential to reverse or at least limit some of the hormonal adaptations to a low EA state. Because it turns out that the basic "solution" to the problem of a low EA state is to reverse it for some time period. By that, I mean that calories are increased, activity reduced, or both so as to bring EA back above the critical threshold or to (or sometimes above) maintenance levels. In the short-term at least, this can reduce or even reverse the hormonal adaptations that are occurring. While this would seem to primarily be important to those women who are normally cycling, keep in mind that the other hormonal adaptations to dieting (thyroid, leptin, cortisol, etc.) occur in all women who cross the critical EA threshold. This makes it relevant to all Category 1 females (though it may still have benefits in Category 2 and 3). Despite this approach being counterintuitive or even detrimental to the fat loss process, it is not only profoundly effective but critical to avoiding or at least limiting the problems described above. This approach has not been studied extensively although a handful of studies do point to its effectiveness. In one, obese women were first dieted aggressively for 4 weeks before having their calories/carbohydrate intake gradually raised for one week. While they were still technically in a dietary deficit, their leptin levels started to increase, indicating hormonal recovery, while fat loss continued (10). In a study of males in the military, recruits were put through a severe eight week training course consisting of severe calorie restriction, enormous amounts of activity and sleep deprivation. This generated a tremendous loss of body fat but there were severe hormonal changes including lowered thyroid and IGF-1, a drop in reproductive hormones (testosterone fell to castrate levels) and a dramatic increase in cortisol levels. A single week of raised calories, even in the face of no change in activity caused all of those hormonal changes to reverse themselves (11). While limited, these studies support the idea that relatively long term increases in calorie levels can reverse the hormonal adaptations to dieting/low EA and I will discuss this topic further in Chapter 20. But what about shorter time periods of increased calories? In animal models, even one large meal can reverse the adaptations to low EA but animals also respond to changes in diet much more rapidly due to their shorter lifespan and faster metabolisms. One meal for a mouse or rat might be the equivalent of a day or more of eating for a human. Regardless, based on this data Ann Loucks, who had first identified the importance of EA and the critical threshold, examined whether a single day of extreme overfeeding could reverse those changes (12). First she exposed women to an extremely low EA for 5 days during which the standard set of hormonal adaptations occurred. The women were then fed 4100 calories (35 cal/lb or 77 cal/kg or roughly double most people's maintenance) over 24 hours. Surprisingly, this had no impact on reversing the adaptations to a low EA. Related to this, a second study made a fairly accidental observation. In it women were first fasted completely for 3 days, coincidentally this generated about the same net calorie deficit as the first 5 day study. Despite the shorter time frame, the same hormonal adaptations all occurred (13). Women were refed for 2 days. When hormones were retested, they had all reversed. LH pulsatility increased to normal, T3 increased, cortisol decreased, etc. When combined, these two studies suggest that a single day of increased calorie intake following 5 days of low EA cannot reverse the adaptations no matter how many calories are eaten. In contrast, following a similar energetic stress in terms of the total calorie deficit, two days of eating at even maintenance is able to do this. While unstudied to my knowledge, it is possible (and anecdotally this seems to be the case) that an even shorter period of time at maintenance might be able to reverse those adaptations. That is, while one day following 5 days of low EA cannot reverse the adaptations, perhaps one day following 2-3 days of low EA could. Recall from an earlier chapter that there appears to be a delay of 3-4 days between the decrease in leptin levels and when the brain "senses" the change and starts to adapt. If leptin levels were reversed to normal or near normal at the day 3 mark rather than the 5 day mark, this might be able to delay the changes. Even if a single day is not having a major hormonal effect, it has other potential benefits that I will discuss in Chapter 21. I will also provide specific schedule for how to implement this strategy at different stages of a diet later in this book. Limited data or not, the above hopefully makes it clear that this completely counterintuitive dieting strategy of raising calories to maintenance or above during a diet is absolutely critical to avoiding or at least minimizing the hormonal adaptations that occur to low EA in terms of the brain or menstrual cycle dysfunction. LH pulsatility can be normalized, T3 goes up and, if nothing else, the increase in calories will reduce cortisol levels. This can not only help to avoid cortisol mediated water retention (many find that their body weight goes down the day after raising calories) but it helps to ensure that a relentless level of chronic stress does not lead to adaptive hypocortisolism in the long run. Enhancing Fat Loss In multiple chapters throughout this book, I have looked in some detail at why women tend both to gain fat more easily while losing it with (generally) greater difficulty compared to men. This included the metabolism of nutrients during and after a meal as well as during exercise, the distribution of body composition and a biology that tends to defend against and adapt to fat loss more strongly than men. This inherent biology combines with the dietary and exercise approaches that women tend to be drawn to (or have recommended to them) which may exacerbate the problem. This includes the issues I discussed above in terms of combining extremely aggressive dietary deficits and exercise but this is not where the problems end in many cases. Specifically women tend to target fat loss with large amounts of aerobic activity, frequently done at a low intensity. If weight training is performed it tends to be done in a mistaken way, using very light weights and high repetitions. This is combined with a diet containing excessive carbohydrates and moderate-to-inadequate amounts of dietary protein and/or fat. But these choices combine with a women's inherent biology to generate suboptimal results. About the only place this isn't seen is in the physique community (who may still use too aggressive of an approach) or strength/power athletes (both of whom combine high protein intakes with proper weight training) which may explain their better results. As I discussed in Chapter 10, while women's bodies use a larger percentage of fat for fuel during aerobic exercise, a larger proportion of that comes from fat stored within the muscle (Intra-Muscular Triglyceride or IMTG) with relatively little of it coming from body fat to begin with (women also burn less total fat due to burning fewer total calories even if the percentage of fat burned is higher). Women's bodies also use less glycogen and rely more heavily on blood glucose during exercise. Essentially their bodies spare both body fat and muscle glycogen during certain types of exercise. Additionally, women's bodies shift back to using less fat and more carbohydrate for fuel the rest of the day. While what is burned during exercise would logically have the largest impact on fat loss, it is actually the calories and nutrients used during the other 23 hours of the day that are important. In contrast, men use less fat for fuel during exercise, relying more heavily on stored muscle carbohydrate, while burning more fat the rest of the day. Since it is the other hours of the day that are important, this contributes to men's generally greater loss of fat. 144 It is a general truism that the body in general and muscle in specific burns the fuel, for the most part, that is present in the largest quantities (it's slightly more complicated than this but I don't want to get into the details). A muscle that is full of carbohydrate tends to use more carbohydrate for fuel (a muscle full of IMTG will use IMTG for fuel) which means that less fat is used for energy. This works in reverse, if muscle glycogen and/or IMTG is depleted with intense exercise (and/or dietary changes), both lean and obese individuals will shift to using more fat for fuel the rest of the day (14,15). For years, I have recommended the combination of diet and specific exercise to deplete muscle glycogen/IMTG and generate this effect. This fact explains part of the gender difference in nutrient use during and after exercise. By depleting their muscle glycogen more effectively during exercise men use more fat for fuel the rest of the day while women who deplete their muscle glycogen less effectively do not. Supporting this idea, a recent study found that men who performed aerobic exercise after eating a meal used more carbohydrate during exercise, depleted their muscle glycogen to a greater degree and used even more fat for fuel the rest of the day compared to men who performed the aerobic exercise after an overnight fast (16). It's questionable if this also applies to women since they tend to rely on blood glucose more than muscle glycogen to begin with and I'm not sure if eating would cause muscle glycogen to be used to a greater degree (it would be interesting to see if it did). [illegible] often have disappointing fat loss results. Despite using relatively more fat for fuel, they are using less fat at all other time points of the day. This is in addition to all of the other problems that can occur when women perform excessive exercise in terms of a low EA state, hormonal and metabolic adaptations, etc. The second observation is that when women replace at least some of their endless low-intensity aerobic activity with some amount of High-Intensity Interval Training (HIIT) or properly done weight training, the impact is often enormous in terms of their ability to lose body fat and improve their body composition. I want to look at why as this leads to a way to enhance women's use of body fat for fuel. HIIT In Chapter 4 I described High-Intensity Interval Training (HIIT or simply interval training), referring to a type of workout alternating time periods (typically 30-90 seconds) at near maximal intensity with time periods of roughly the same duration of low-intensity exercise. While traditionally used by athletes, HIIT became popular when some research suggested that the fat loss might be greater compared to traditional aerobic activity. There was also a great deal of interest in HIIT for improving general fitness as it was often more time efficient than traditional aerobic exercise while generating similar or the same results. While the overall effects on fat loss were greatly overstated, there is no doubt that many women found that adding at least some to their training drastically improved their body composition and fat loss and I think that there are several reasons for this. [illegible] HIIT may also increase the amounts of the calorie burning beige/brown fat that I described in Chapter 5 (19). The hormonal response may also play a role here. HIIT raises Growth Hormone (GH) levels, which helps to mobilize fat, to a greater degree in women than men (20). Levels of ANP, the hormone that may sidestep the hormonal problems with stubborn fat mobilization, go up to a greater degree in women than men as well (21). HIIT causes a larger increase in adrenaline and noradrenaline compared to low-intensity aerobic activity and this has been shown to cause greater lower body fat loss (21a). I wrote about this years ago in my original Stubborn Fat Solution and two of the protocols utilized HIIT for this reason. Women using these protocols reported enormous improvement in their ability to lose lower body fat and I have reproduced them in Appendix 2 of this book. Of as much importance within the context of this chapter is that HIIT, like most high-intensity exercise, is fueled primarily by carbohydrate and specifically muscle glycogen (IMTG may also contribute during the rest intervals). Even here there is a gender difference: in response to one 30 second sprint, for example, women deplete less glycogen in Type I/slow-twitch but equal amounts in Type II/fast twitch muscle fibers (22). There are a number of reasons for this that I will detail in Volume 2 but it has been shown repeatedly that women generate less fatigue and recover more quickly than men in repeated sprints (23). Practically this means that optimizing HIIT for women's fat loss goals will require slightly longer intervals [illegible] than men. Women may also benefit from doing slightly more total intervals than men to ensure the same degree of glycogen depletion, enhancing the use of fat for fuel for the remainder of the day. 145 I want to make it clear the above is in no way meant to suggest that women should replace all of their traditional aerobic activity with HIIT. [illegible] I will discuss this in more detail in Chapter 27 and 28. Rather, it is meant to point out that replacing at least some of a woman's traditional low- or moderate-intensity training with HIIT can have profoundly beneficial effects on fat loss. Weight Training While HIIT alone can have potential benefits for women's fat loss efforts and body composition changes, perhaps nothing is as singularly effective as properly performed weight training. By properly here I mean using weights that are challenging to lift and which will actually have benefits in terms of increasing strength or muscle size. An additional, and equally important benefit, is the effect such training has on bone mineral density, either to stimulate it's gain or prevent it's loss. What is defined as challenging will depend on the trainee and the specific type of training being done and I will discuss the specifics of this somewhat in Chapter 27 and in far more detail in Volume 2. This type of proper weight training is in contrast to the type of training that many women do (or are recommended to do) which is to use very light weights, often for a very large number of repetitions. This is typically done to "tone" or for fear of "becoming bulky". As I previously discussed, being "toned" is a function of building some amount of muscle mass and losing fat while becoming muscularly bulky is simply not a fear for most women due to their lower levels of testosterone. At most, the PCOS woman with hyperandrogenism or subclinically hyperandrogenic women may build muscle slightly more quickly but even this is only in a relative sense. Invariably when a woman who is only performing enormous amounts of aerobic exercise introduces this type of lifting, or switches from the fairly common ineffective types of training to this type of training, the effects are almost magical. Shape and appearance improve drastically, fat loss frequently occurs quickly and/or becomes easier and this is true even if much less aerobic exercise than previously is being done. Done properly, no single type of exercise has as large of an impact on a woman's health, bone density, body composition or appearance. And I think that there are a number of reasons, some indirect and some direct, that weight training has this effect. Indirectly, women who start to engage in more productive forms of weight training often stop performing excessive aerobic activity. [illegible] Regardless, even reducing cardio from the often excessive levels seen has a number of positive benefits in its own right. One is that it may allow a woman who has also reduced her calorie intake by too much to go above the critical EA threshold. [illegible] Due to less overall fatigue, she might also find that her NEAT increases and this may potentially add up to an increased energy expenditure despite doing less activity. Anecdotally, many women report being able to eat more calories while doing less activity, while still staying lean or even losing body fat, when they cut out excessive aerobic activity. Additionally, the often chronically elevated cortisol levels will be allowed to drop which has more positive effects. [illegible] Like HIIT, the high-intensity nature of proper weight training means that muscle glycogen is the primary fuel. In men at least, multiple sets of weight training deplete both muscle glycogen and IMTG although it takes a fairly large number of sets to have a major effect (24). [illegible] 146 [illegible] The Impact of Diet [illegible] As much as I find the phrase trite and often misused or misleading, to put it in the common parlance, a woman becomes a "fat burning machine". [illegible] Physique athletes have always used a combination of resistance training and increased protein intakes (often with reduced carbohydrate intakes) and I think it is no coincidence that they have traditionally had some of the best success with altering body composition overall. 147 148 Chapter 15: Introduction to Dieting [illegible] Different Needs for Different Goals [illegible] The Pros and Cons of Popular Diets [illegible] Fitness Magazines [illegible] Commercial Diet Programs [illegible] Popular Diet Books [illegible] Dietary Restriction, Disinhibition and Dieting Attitudes [illegible] Structured Flexible Eating is often useful. These represent specific approaches to flexible eating that, while flexible, still have some specific rules and this can be a good transition in the early stages. I will describe these strategies in detail in Chapter 21. When Should a Woman Start a Fat Loss Diet? As a final general dieting concept, I want to address the question of when a woman should start her fat loss diet. Certainly most start their diets on a Monday (giving them a last chance to overindulge on the weekends) and there are considerations for those dieters who must reach a certain BF% by a given date but, beyond that, does it really matter on what week or month a diet starts? For the normally cycling woman, the answer is yes. Readers may recall from Chapter 2 that a woman's hunger and appetite is generally lowered during the follicular phase and lowest immediately prior to ovulation. This is reversed as hunger and cravings go up during the luteal phase with the late-luteal phase being the worst. Logically, it makes the most sense to start a diet after menstruation starts when hunger is lowest and the dieter is most likely to be successful to gain some positive momentum (13). This can be contrasted to attempting to start a fat loss diet in the luteal phase where the greatest difficulties in adherence will be encountered, possibly harming long-term adherence. For those women who must reach a goal BF% by a certain date, this means determining the predicted length of the diet (discussed in Chapter 25) and then adjusting the start date based on where in the cycle it falls. In some cases this may require starting the diet two weeks earlier than otherwise planned so that it will synchronize with the follicular phase; in others there may be no other option but to start during the luteal phase. The possible need to include the Pre-Diet phase and this adds another factor to consider in determining the start of the diet. For a 2-week Pre-Diet phase, it could easily be placed during the luteal phase so that the diet itself can be started during the next follicular phase; a 4-week Pre-Diet phase would have to start in the follicular phase for the fat loss diet itself to start in the follicular phase as well. I've shown this below. Follicular Phase Luteal Phase Diet Follicular Phase 2-week Pre-Diet Phase Begin Diet 4-Week Pre Diet Phase Begin Diet The above will not apply to any women with hormonal modifiers with the possible exception of women on some types of birth control with a withdrawal week. Since estrogen rebounds during this week, hunger may be relatively more controlled relative to the other three weeks of use and that would make the withdrawal week the best time to start the diet. For all other women, when the diet itself starts will be of little consequence beyond meeting the person's individual needs. 156 Chapter 16: Determining Maintenance Calories The first step in setting up a calculated diet for any goal is to determine, or at least estimate, maintenance calorie requirements. This is the number of calories that should ideally maintain both body weight and body composition without change and it is from here that any changes will be made to achieve different goals. I say ideally as there can be situations where body weight is relatively stable but body composition may change for the better or worse (i.e. some types of birth control can cause a slight gain of body fat and loss of muscle despite no weight change). I've referred to this value as Total Daily Energy Expenditure (TDEE) earlier in the book and will do so here. As discussed previously in the book, TDEE is made up of four components which are resting metabolic rate (RMR), the thermic effect of food (TEF), the thermic effect of activity (TEA, formal exercise) and Non-Exercise Activity Thermogenesis (NEAT). RMR is related mostly to lean body mass (LBM) but can be estimated with total weight while TEF is usually taken as 10% of total calorie intake. The calorie burn from TEA can vary massively and NEAT is incredibly difficult to estimate at this point. It's crucial to understand that any estimate of TDEE is only that, an estimate. Even with complicated equations, there is some variability between any two individuals at an identical weight and body composition with the greatest variation, outside of people performing a large amount of formal exercise, coming from variations in NEAT. While it's certainly possible to estimate good starting values for TDEE, real world changes in body composition will indicate if that starting point is correct or needs to be adjusted. It's equally important to remember that TDEE can change in both directions in response to dieting or overeating. For this reason it's better to think of it as a range than a fixed value. All components of TDEE adapt downwards in response to dieting and fat loss while they can be reversed or even increased in response to overeating and weight or fat gain. Women who have been on low calories for extended periods of time frequently report that their weight and body composition remains relatively stable even as they begin to increase their calorie intake. This is invariably due to changes in NEAT, coming above the critical EA threshold which may allow some degree of hormonal recovery, etc. Athletes often find that they can train more intensely which means that their TEA goes up along with those increasing calories. There are a number of ways that maintenance calories can be estimated and I will look at two different approaches, one of which I think is potentially problematic, especially for women. I'll also present a slightly more complicated but, in my opinion more accurate method of estimating maintenance. While I won't discuss them in detail, some of the new activity trackers may also be useful in obtaining a better estimate of TDEE as well. Tracking Calorie Intake and Bodyweight Method Used for years, one of the simplest approaches to estimating TDEE is to track bodyweight (and again I'd recommend a 7-day rolling average) and calorie intake for some period of time. Generally two weeks is taken as the minimum time frame due to daily variations and the assumption is that, if body weight is stable over this time period, the current calorie intake is equal to TDEE. That is, if someone gained weight over this time period, presumably their calorie intake exceeded TDEE and if they lost weight, their calorie intake was below their actual TDEE. In premise this approach makes a good deal of sense and determines an actual calorie intake level rather than trying to estimate it but I think there are some problems with it. The primary one is that it requires calories to be tracked accurately. This entails measuring and writing down all food and drink that is consumed This is not necessarily a bad thing and, as I will describe in a later chapter, is one of the most information exercises any person can go through to learn what real world portion sizes and calorie values are. At the same time, it can be a bit of a pain in the butt and, outside of the Category 1 dieter, may be overkill in the beginning. For the woman adopting a simpler diet approach it would be excessive. Those women are usually not setting calorie intakes to begin with. Even for the Category 1 dieter, this approach may be problematic. First and foremost is that weight isn't the same as body composition although the odds of any significant changes in body composition occurring over this time period are slim so that's really a non-issue. The normally cycling female have the additional issue of menstrual cycle related water weight changes. A woman tracking during the first two weeks of the cycle can't get an accurate idea of whether or not she has gained, maintained or lost weight when her weight may swing wildly due to water retention. She could track for a full month and compare similar weeks of the cycle and this would be reasonably valid. Women with a hormonal modifier won't have this issue for the most part but in most cases, I recommend the next method. 157 Calculation Method Although more complex than the previous method, I think that using a calculation based method to determine or at least estimate TDEE/maintenance calories is generally more appropriate. This is especially true for the normally cycling female who may not be able to use the above method due to shifts in her water weight every week. With this method, TDEE is calculated by estimating the four components of TDEE and then adding them together. While there are more complex methods that can be used (that I feel are generally unnecessary), the method I will present will be to first estimate RMR which will be increased by an activity multiplier based on both both activities of daily living (NEAT) and formal exercise (TEA). Since it is generally small, TEF is often ignored completely but I will factor it into the activity multipliers. As well, the NEAT value will only include overall daily activity as the unconscious part of NEAT (fidgeting, etc.) cannot be estimated in any meaningful way. Traditionally a single multiplier has been used based on overall or average activity levels during the week but I find this problematic. Most people's activity is not identical every day of the week and their work days may look very different than their non-work days. Athletes have the added issue of their training often varying significantly from day to day (some sports have training sessions that are more alike than not but this is not universal). An endurance athlete who used the same multiplier every day but who did 4 hours of training one day and only 1 hour the next would be vastly mis-estimating their true calorie requirements, making it impossible to match their nutritional needs. Practically this means that any day of the week might have it's own multiplier (and resulting TDEE) although most will probably end up with only a handful of different values for different types of training days. Women, as usual, have their own specific issues related to maintenance calories. The first is that their energy expenditure for all components of TDEE are roughly 10% below those of men. As discussed in Chapter 10, most of this is related to differences in body composition (1). This difference is also reflected in the calculations below and no further adjustments have to be made. There can also be slight adjustments to TDEE based on the hormonal modifier present and detail-oriented readers may wish to apply those. Do remember that all of these calculations are only estimates at best; no matter how close to TDEE they come, they may still have to be adjusted over time. Estimating RMR The first step in calculating TDEE is to estimate RMR, the number of calories that the body burns at complete rest. There are endless equations that have been developed over the years that range from simple to very complicated. Since they all tend to give results within a few hundred calories of one another, I prefer to use the simpler equations. For the most part, RMR equations have only been based around body weight (often including age, height, gender and others) but given the importance of LBM in determining RMR, these tend to become increasingly inaccurate for women with a very high or very low BF%. For example, a commonly used equation for women is to multiply bodyweight by 10 cal/lb (22 cal/kg). While this is accurate within a certain range of BF%, it becomes progressively more inaccurate as BF% goes up. For this reason, I prefer equations that take body composition into account and am presenting a simple one that I derived myself from other, more complex, equations. It requires body weight and some estimation of BF% so that the total amount of LBM can be calculated. I've shown the calculation for two women of different body composition below. RMR = (12 calories * LBM in pounds) + (2 calories *fat mass in pounds) or RMR = (26.4 calories * LBM in kg) + (4.4 calories * fat mass in kg) Example 1 Female at 150 lbs, 22% body fat with 117 lbs LBM and 30 lbs of fat RMR = 150 lbs * 10 cal/lb = 1500 calories RMR = (12*117) + (2*30) = 1404 + 60 = 1464 calories or 9.8 cal/lb Example 2 Female at 250 lbs, 50% body fat with 125 lbs LBM and 125 lbs of fat RMR = 250 lbs * 10 cal/lb = 2500 calories RMR = (12*125) + (2*125) = 1750 calories or 7 cal/lb You can see from the above calculations that while the 10 cal/lb value is very accurate for the leaner woman it drastically over-estimates the woman with a high BF% with the actual value for RMR dropping from 10 cal/lb to 7 cal/lb. For women who don't want to perform the above math, the following chart can 158 be used to estimate RMR from just bodyweight. Some estimate of BF% is still required but the values under each BF% can be multiplied by total body weight to estimate RMR. BF% 20 25 30 35 40 45 50 RMR (cal/lb) 10.0 9.5 9.0 8.5 8.0 7.5 7.0 20 19 17.5 16.5 15.5 RMR (cal/kg) 22 21 To use the chart, bodyweight is multiplied by the RMR value underneath the appropriate BF% value. The sample female at 150 lbs and 22% body fat would have an estimated RMR of 150 lbs * ~9.7 (halfway between the 9.5 and 10.0 values for 20 and 25% body fat) or 1455 calories which is effectively identical to the value I showed above. The 250 lb/50% body fat female would multiply her weight of 250 by 7 to get an RMR of 1750, identical to the value calculated with the first equation. This value for RMR will be modified by the activity multipliers, described next. Activity Multipliers If someone did nothing more than lay in bed all day, their TDEE would be equal to their RMR. Since most do not, this value will be increased based on the level of activity being done. Traditionally, activity multipliers have combined both TEA and NEAT but I find it more useful to split them up for better accuracy. This approach also makes it easier to take into account changes in each when activity levels are varying from day to day. Certainly only needing a single multiplier for every day would be simpler but this tends to be unrealistic unless someone's daily activity is extremely consistent. Daily Activity (NEAT) Since not everybody is involved in formal exercise but everyone (unless they are completely bedridden) performs at least some amount of daily activity, I will start with an estimation of that multiplier. In the modern world, someone's activity may range from completely sedentary to requiring extremely high levels of activity is their job or lifestyle is very labor intensive. For this reason, RMR multipliers from 1.2 to 1.9 are usually considered to be realistic with 2.5 times RMR being the maximum energy expenditure that can be sustained for extended periods (athletes may surpass this for short periods due to their incredibly high TEA values). For most people a realistic NEAT multiplier will be 1.4-1.7. In the chart below, I've shown multipliers for different activity levels and their general descriptions. Activity Level Description RMR Multiplier Sedentary Sitting, talking, reading, watching TV 1.3-1.4 Light Office work with moderate walking 1.4-1.5 Moderate Busy lifestyle w/ lots of walking 1.6-1.7 High Construction, hard labor 1.7-1.9 If no formal exercise is being performed, RMR can simply be multiplied by the value above to get the estimated TDEE. If our 150 pound female with a maintenance of 1455 calories had a sedentary lifestyle, she would use a multiplier of 1.3 to get a maintenance of 1890-2040 calories (1455 * 1.3 or 1.4). If she were moderately to highly active, she would use the 1.7 multiplier to get an estimated TDEE of 2475 calories/day (1455 calories/day * 1.7). If formal exercise is being done, it will have to be added to the above value. I will also provide a chart later in the chapter that will simplify all of the calculations. When using the above chart, I strongly encourage readers to be realistic about their daily activity levels. Someone who sits in front of a computer most of the day and does little else will be somewhere between sedentary and light activity even if they feel that is too low or dislike the relatively low TDEE value that is estimates. Someone on their feet all day will be in the moderate category and few will achieve the highest values unless they are moving continuously or working a very labor intensive job. While many older estimates put most people's multiplier closer to 1.7, I feel that changes in the modern world have made this too high for many people. Practically I would generally suggest erring on the side of too low of a multiplier than too high. Calories always need to be adjusted based on real world changes and it's better to be eating slightly too few and having to increase due to weight loss than the converse under most circumstances. 159 Exercise Energy Expenditure (TEA) Once the daily activity multiplier has been determined, the calorie expenditure from formal exercise, if it is being done, will need to be added to determine TDEE. The number of calories burned during exercise can vary enormously depending on the type, amount and intensity of the exercise done. In many forms of exercise, bodyweight also plays a role with larger bodies burning more calories. This is often offset by heavier individuals often being limited in the amount of exercise that they can perform. To put this into perspective, a relative beginner or untrained individual may burn only 200-300 calories in an hour of exercise (although this will increase as fitness improves) while a highly trained endurance athlete might burn up 650-900+ calories per hour and double or triple that for an extremely long duration workout. Observationally, female athletes report calorie intakes ranging from 15-23 cal/lb (33-50.6 cal/kg), representing a 1.5-2.3 RMR multiplier, depending on the sport and amount of training being done (2,3). Weight lifters tend to be towards the lower end of the range, high-intensity and team sports fall somewhere in the middle and only endurance athletes achieve the highest values due to the amount of training that they do. Due to difficulties in measuring actual energy expenditure, the above values are based on reported food intakes. As many female athletes undereat relative to their actual energy expenditure, it's possible and somewhat likely that actual values for true energy expenditure are somewhat higher. However, the American College of Sports Medicine (ACSM) position stand on the topic recommends a BMR multiplier of 1.7-2.3 (~ 17-23 cal/lb or 37.4-50.6 cal/kg) from moderate to heavy training and this is right in range of the reported calorie intakes (4). I'd note that these values are roughly 10% what is seen or recommended for male athletes in keeping with the differences in body composition, etc. As well, these values are for hard training athletes only. Recreational exercisers will not achieve all but the lowest of those values. Readers may see that the above values overlap with the general daily activity and some of this is due to the above representing total daily calorie expenditures rather than exercise alone. However, there is often an inverse relationship between the amount of exercise being done and other daily activities. The busier someone is, the less time or energy they have to put into exercise and athletes doing a large amount of training are often less active at other times of the day due to fatigue or simply recovering after a hard workout (in the case where a hard training athlete may be working many hours at a labor intensive job, their TDEE can skyrocket). A woman with a 1.9 multiplier for her daily activity is unlikely to do much exercise on that day and surpass a 2.3 multiplier. On a day off from work, when her daily activity is much lower, she might be able to fit in a much larger amount of exercise. Conceivably she could have similar activity multipliers for each, just accomplished through a different pathway: NEAT versus TEA. In the chart below, I've listed some general types of exercise along with their rough calorie burn per pound/kg per hour of activity. If 2 cal/lb is listed, a 150 lb woman would burn 150 calories in 30 minutes, 300 calories in an hour and 450 calories over 90 minutes. I've grouped the activities by intensity although more complete lists can be found online. In many cases, the values shown in those lists will be higher than what I have shown below. This is because I have factored out what a woman would burn doing no exercise at all. If someone would have burned 60 calories/hour sitting and burns 300 calories/hour during exercise, they have actually only burned 240 extra calories per day. This not only gives a more realistic indication of actual calorie expenditure from exercise but is the value that should be used to estimate energy availability (EA) if those calculations are being made. Activity Examples Per Hour of Activity Multiplier Low Intensity Aerobic (130 HR or lower) Brisk walking, slow cycling (