

The Second Law: Woolly Mammoth Training



The Way is in training.

—Miyamoto Musashi
from *A Book of Five Rings*

Now I am going to tell you how to lose body fat and keep it off for the rest of your life. In this segment, we will take apart the nature of training stress and how it effects the mobilization and utilization of the fuel substrates—principally carbohydrates and fats—a catabolic process. The mirror image of fuel mobilization, fuel restoration—which is an anabolic process—will be examined in the macronutrition segment. These two opposing processes, fuel mobilization and fuel restoration, comprise fuel regulation and control. Understanding them and their interrelationship is vital to your business productivity, quality of life and survival. Mastering them, through diligent practice of An Operating System for the Human Organism, is your life’s hope.

Endurance training is comprised of several very different types of training stimuli, but we are only going to focus on one type right now, prolonged sub-maximal endurance training. The training I am going to discuss is not some new millennium fad workout; on the contrary, it is the oldest and most proven training method known to the entire animal kingdom. It is so imperative to our existence that it is tattooed on our primordial genetic blueprint. For that reason, it is the Second Law of Biological Preservation. Unfortunately, it is a law broken by almost everyone and, if you are caught, the punishment is slow death by digital man disease. All I am doing is resurrecting it and making it very efficient for all inhabitants of the digital-age society.

Let's go back thousands of years again only this time we will only be concerned about our next woolly mammoth. Let's say you haven't seen a woolly mammoth in over three days and you have been walking so long that you can't keep up the pace. That's a big problem because you are going to be buzzard food unless there is a woolly mammoth coming your way soon. Thankfully, our primordial genetic blueprint has a script for us to adapt to the physical stress of the hunt in such a way that we become capable of going longer and longer between successful hunts. It is really ingenious how it works because it elegantly manages our fuel storage, fat and glycogen stores, catecholamine and insulin sensitivity and the combustion of the fuel mixture in the motor, the metabolic conversion of fuel into energy within the muscle cells of our prime movers—our legs and gluteal musculature.

To understand how to decrease and manage body fat storage you have to know how, where, why, and under what circumstances fat is utilized for fuel. Right now, sitting there, you are burning fat. When you sleep, you are burning fat. Why then do we get fat if we are burning fat all the time? The reason is that fat is a very efficient fuel in terms of how much energy is released per unit weight of fat—9 calories per gram. The secret to managing fat storage lies within the mechanics of the motor—muscle physiology—and the carburetor, whose biological equivalent is the principal hormonal regulator of lipolysis—the catecholamines and the adrenergic receptors (do you remember their caustic Dark Side from stressful encounters of the wrong kind?). If you had a car and you wanted to reduce the amount of fuel in the gas tank, would you put in a little four-cylinder engine or a big, V-12 gas guzzler? Obviously, you want the V-12. Let me build a V-12 for you.

Body fat is only lost when it is converted to energy inside a muscle cell organelle called the mitochondrion—the powerhouse of the cell. To build a V-12 we need to impose a stress on the muscle that triggers the alarm phase so that the adaptation during the resistance phase is making more mitochondria, among other things. The more mitochondria you make, the more independent channels of fuel oxidation you create, which are called respiratory chains. The more respiratory chains you make, the more body fat you burn when you train at the appropriate intensity. Furthermore, the quantity of respiratory chains, which can be measured by overall mitochondrial enzyme mass, and the quality,

which can be measured by the enzyme activity, correlates strongly with endurance capacity. It is endurance capacity that will profoundly impact your business productivity, your ability to function at high performance levels over long periods—instead of fading into digital oblivion. In other words, because woolly mammoth man morphed into digital man, you will produce more (and hopefully better) mouse clicks in lieu of lasting longer between woolly mammoth kills—just a minor change in occupation. Woolly mammoth man is designed to do a much higher training volume than what I am suggesting: I realize your time is valuable and so this training—which I call the woolly mammoth workout—is designed to modify your biology for optimal quality of life, longevity, and fuel management. There is no substitute. Not one second you do in this program is a waste of your time. First, I am going to outline how to do the woolly mammoth workout, and then, I am going to present a historical account of training as it relates to fuel substrate mobilization and utilization over digital man's lifespan.

Any cyclic activity, such as running, stair climbing machine, elliptical machine, cycling, or rowing will suffice, but I would recommend treadmill running or walking on an incline and stationery bikes (but not “spin” bikes because one of the inputs of chronotropic control (heart rate) comes from receptors in the joints, high joint speed feeds back signals to increase heart rate independent of metabolic demand) for now until you get a feel for the intensity and duration required to achieve the desired training effect. With cycling, you can be time efficient and talk on the phone with a headset, read the *Wall Street Journal* or watch the NASDAQ roll by while you are training. You are also going to need a heart rate monitor, your maximal heart rate, and your minimal heart rate to get started.

I have had as clients some of the best Tour de France cyclists in the world and the physicians that train them, and this is the type of training that most of them do in the off-season to build their endurance capacity. They earn their living from their endurance capacity and the training technology to achieve this objective is state-of-the-art. These cyclists, while in training can eat over 6,000 calories a day and still lose weight. Prolonged sub-maximal endurance training is foreign to you because it is not derived from the fitness or medical communities, but, instead, comes from the athletic performance world. Where did the

Tour de France physicians get it? The physicians, of course, adopted it from gold-medal winning woolly mammoth hunters! I picked it up from the cyclists, created a quantifiable termination point, redefined the protocol for determining minimum heart rate for this kind of conditioning, and then increased its efficiency by improving the adaptive yield through synchronized micro- and macronutrition and highly specific strength training. What I did was save you an incredible amount of time and futility. The woolly mammoth workout's protocol is a marriage of the primordial genetic blueprint's potential to the intelligent application of technology from An Operating System for the Human Organism. The physiological mechanisms and methodology of the woolly mammoth workout are in print for the first time in *The Digital Mantrap*. I want to share it with you because it is something every busy professional needs to embrace and prioritize in his or her lifestyle. Obviously, woolly mammoth man did not have heart rate monitors and their daily activity included treks of many hours while on the hunt in a wide range of "training" intensity. I have accelerated the process by increasing the intensity to the point that further intensification will not lead to the physiological events—and consequent adaptations—that we seek. The woolly mammoth workout is precisely what digital man needs to do to survive between hunts. You may have long forgotten about the hunt but your primordial genetic blueprint has not. Don't forget for one moment that woolly mammoth man inside controls your destiny—with or without your consent.

How to Do the Woolly Mammoth Workout

I strongly recommend you get a physical examination from your physician before you begin this, or any exercise program. Your physician is best suited to assess your present condition and what types of risks are tolerable for you. If you are considered high risk, you will need to modify your training until you achieve a higher level of fitness. From the stress test, you will find out what your maximal heart rate is. If you don't know your maximal **observed** heart rate, you will have to use the inferior, approximate value of 220 minus your age. Your minimal heart

rate you can determine yourself by observing your heart rate when you just wake up in the morning while still in a supine position. I don't want your resting heart rate while you are sitting around at 3 PM. I want the lowest value in a 24-hour period. This is closer to a *basal* heart rate than a *resting* heart rate. The difference between the two values, the maximal heart rate—which correlates with your maximal cardiac output—and the basal heart rate—which correlates with the cardiac output just to maintain your body under \emptyset activity—gives us a range of cardiac outputs that corresponds to the metabolic demands of locomotion from \emptyset activity to maximal activity. In other words, we have calibrated your body's ability to perform endurance work from \emptyset to 100% on an intensity scale—picture a car's tachometer from just above its idle rpm to the very end of the redline area. Find out your minimum heart rate first, otherwise if you do a max test and then find your minimum the following morning it might be artificially high by two or more beats per minute. You will need both these numbers to calculate heart rate training ranges. Your heart rate is how you measure intensity in endurance training. Without knowing intensity, you will be physically active but you might not be training.

Here is how the woolly mammoth workout is done. The first thing you do is calculate your target initial heart rate which defines the intensity. The second thing is calculate the target termination heart rate. Every couple of months you should obtain new max and min heart rates until they stabilize. Using what is called Karvonen's heart rate reserve formula, subtract your minimal heart rate from your maximal heart rate and multiply that by 60% (6.2, Sleamaker, 1989). Now add your minimal heart to that and you have your target heart rate. The termination heart rate is calculated by repeating the above equation except replace the 60% with 70% and add 5 to that. Here is an example. Let's say your maximum heart rate is 195 beats per minute and your minimum heart rate is 65. $195 - 65 = 130$. $130 \times 60\% = 78$. $78 + 65 = 143$. That is the target initial heart rate. $130 \times 70\% = 91$. $91 + 65 = 156$. $156 + 5 = 161$. You should notice one huge difference between this and other forms of endurance training. I took "endurance" out of the equation by throwing away the clock. I then replaced the clock with nature's reaction to the stress by using your own heart rate response. In other words, you don't train for 45

WOOLLY MAMMOTH WORKOUT HEART RATE EQUATIONS

$$HR_{\text{TARGET}} = ((HR_{\text{MAX}} - HR_{\text{MIN}}) \times 60\%) + HR_{\text{MIN}}$$

$$HR_{\text{TERMINATION}} = ((HR_{\text{MAX}} - HR_{\text{MIN}}) \times 70\%) + HR_{\text{MIN}} + 5$$

minutes or 60 minutes: you train until a specific fatigue scenario has been achieved independent of “time.” Although we are using beats per unit time, we are now directly connected to an authentic physiological response. You will find that listening to the body is a monumental advance in feedback compared to the meaningless gyrations of a clock.

The second order of business is managing your source of blood-borne fuel substrates prior to training. Ideally, you want to begin training with a minimum of a 6-hour fasted state to increase the ratio of fats to carbohydrates burned during the training session. In any case, avoid carbohydrates at all cost prior to training because this will increase insulin and attenuate adrenergic response. In other words, you would be burning carbohydrates instead of fat. To give you an idea how much you are shooting yourself in the foot, if you ingest 2 grams per kilogram of bodyweight of carbohydrate two hours prior, that will cost you an increased carbohydrate oxidation of 15% and, if four hours before, it is 13% (6.3, Montain, 1991). That is the opposite of what we are trying to do. In regards to low blood sugar, or better known as hypoglycemia, ingesting carbohydrates within 4 hours of exercise promotes a significant *drop* in blood sugar within 10 minutes of training. I have seen this all the time with racehorses and it is the kiss of death. The horse will finish dead last and behave like a sleepwalker. In both the trained and untrained state, minimal reduction of blood sugar will occur if your last carbohydrate source is 6 hours out or longer. What I am saying here, between the lines, is that you need to time manage your woolly mammoth sessions. But you should never adopt an attitude of not doing it because you ate 2 hours prior. Doing it is a lot better than making up excuses; just do the best you can with scheduling. Time to get on the bike!

You get on the bike and adjust the resistance or choose a gear that is

easy to push. This is the warm up. The warm up prepares your body for the stress of training. Over the course of 5 to 10 minutes, depending upon your condition and the projected duration of the training session, bring your heart rate up and readjust the load accordingly, if necessary. Once you reach the target heart rate in a graduated approach over the warm up interval, keep the load constant and go into auto pilot mode. For example, when you reach 143 bpm, you look at your velocity or power output and STAY LOCKED ON at that power level (or velocity) until you reach the termination heart rate. DO NOT vary your power output to maintain the target heart rate.

Several factors will affect your heart rate response during the woolly mammoth workout. One of them is the immediate change in heart rate when you move from an erect, seated position to a crouched position with hands or elbows braced on the handlebars. As the angle in your spine varies from horizontal to vertical to the floor, the work required to return blood (venous return) from the working muscles increases because of gravitational forces. The venous blood returning from the legs must now fight the force of gravity, which will reduce the preload, or the degree of stretching on the myocardium prior to contraction. The filling pressure affects the contractility which impacts the stroke volume, or the volume of blood ejected from the left ventricle, which then goes to the working muscles. Since a reduced stroke volume decreases cardiac output, the body compensates by increasing heart rate to keep cardiac output constant (6.13, Matthews, 1996). The reverse effect is noticed going from an upright posture to a prone posture. For our purposes, monitor your heart rate from as close to a prone orientation as possible to be consistent with the max and min heart rate calibrations. Feel free, of course, to alter your position during training to whatever position is comfortable for you, whenever you wish.

Another factor affecting heart rate is cardiovascular drift. It is the phenomenon of heart rate increasing over time while at constant load. Cardiovascular drift has four components: plasma volume decrease due to fluid losses to dissipate heat in order to regulate core temperature homeostasis, adrenergic response to maintain fuel supply, the progressive recruitment of lower efficiency muscle motor units as fatigue ensues, and decreased contractility due to myocardial fatigue. The last three factors are the ones we want to have as variables during the woolly

mammoth workout. They will give us feedback on how well our body is responding and adapting to the training stress. The first component, plasma volume, we will eliminate from the equation by drinking one liter of water per hour, assuming normal environmental conditions. This will slow the progressive heart rate increase over the course of the workout by nearly factoring out changes in plasma volume, which would negatively impact stroke volume (6.3, Hamilton, 1991; 6.3, Millard-Stafford, 1990; 6.3, Costill, 1974; 6.3, Fordtran, 1967). *Under no circumstances do you want to drink any fuel substrates during the workout.* If you consume fuel substrates, you are defeating the purpose of prolonged endurance training. The objective is to cause stress to lipolytic and glycolytic aerobic metabolism and the hormonal regulation of fuel substrates, *not* to artificially prolong sub-maximal endurance training duration by oxidizing external, or, better-known as, exogenous fuel sources. This is wonderful if you are competing in a race, but is counterproductive for producing adaptive response. *Water only*—no carbohydrates, bars or anything else. If you follow directions, you will find out how good of a fat burning machine you really are and you will accelerate the process of becoming a V-12.

If you have been sedentary for a long time, or significantly overweight, you might not be able to achieve a steady state heart rate at 143. This is because the intensity level where lactic acid begins to accumulate in sedentary people can be at 50% of VO_2 max or even lower and the target heart rate is about 55% of VO_2 max. If this is the case, you will feel like you are working hard just to maintain 143 and you will notice that your heart rate will quickly rise to the 161. If this happens to you, you have two options. The first option is to begin a walking program. I won't go into details on walking, but depending upon your condition, you may have to limit your initial walking duration to less than five minutes. *If your physician has assessed you as high risk do not do attempt this training program at all right now.* Instead, let your physician manage your fitness goals until he or she says otherwise. If you are not high risk, gradually over the course of four to six weeks increase the intensity of your walking sessions beginning with walks every other day and progressing to every day. When you can walk at a brisk pace for 45 to 60 minutes you will be ready to do this training. The second option is go back to the Karvonen heart rate equation and substitute 45% for the

60% and 55% for the 70% and recalculate the initial and termination heart rates. When you can go 45 minutes before reaching the termination heart rate, go back and do the 60% intensity again.

If you are sedentary and overweight, you have several obstacles in your path. Number one, you are a four cylinder engine so you don't have much capacity to burn fat either while training or sleeping which means it is physically impossible for you to burn much fat. That's why the fat get fatter. Number two, at the same relative intensity of 65%, a fit person will burn ~53% fat whereas an untrained person will burn ~33% (measured after 1 hour) (6.3, Jansson, 1987). Number three, the perceived effort to perform this type of training is going to be tough on you. Number four, if you are high risk it may take awhile just to progress from high risk to unfit. Number five, because this is hard for you in the beginning, the temptation of yo-yo diets and thermogenic or ephedra or fat-blocking miracle pills will be overwhelming. This snake oil is so absurd that it can't even stand in the little Dutch boy's shadow. There are no shortcuts. This is a two-year strategic plan, not a two-week miracle prayer. Be patient and do not give up. Set little goals like going from 5 minutes walking to 10 minutes. You did not become overweight overnight and you won't become lean overnight. You will want to quit. You will astound yourself with incredibly creative and inspirational excuses why not to do it today. You will tell yourself you will do it later. You will say this does not work for me, my metabolism just doesn't respond, I have bad genetics, I will find another way, an easier way. You will buy the latest diet and fitness books on the best-seller list that every one is just raving about. Just remember this: This is the fastest, most efficient way to lose body fat and keep it off for life. Mother Nature tattooed it to your genome for a reason. The only way to burn fat is through oxidative enzymes in the mitochondria, and the fastest way to burn more body fat is progressing from a four-cylinder engine to a six to a V-8 to a V-12. No drug or diet can build an engine and you only have a four-cylinder gas miser with a huge gas tank right now. Weight loss should not be an obsession and isn't even the focus; the focus is implementing the engine-building process of progressive woolly mammoth workouts. The byproduct of the engine-building process is fat loss; or, in other words, body fat loss is an effect of, and secondary to, the engine-building process. The weight scale does not

measure progress; *progress is measured by the rate of change of fat calories burned in a 24-hour period.* It will take time and consistency. Habits are processes. Indoctrinating new habits requires time management and abolishing bad or non-productive habits. The woolly mammoth mantra must become habit-forming.

For most people, and that includes the sedentary people mentioned previously who didn't quit and progressed to the initial starting intensity, the training workload should feel very easy, in fact you might think it is so easy that you are just wasting your time. The fact is that what you feel and the training effect we desire are in direct conflict and the mistake that everyone makes is: "I can go harder than this and let's just get this over with." Big mistake. You cannot substitute intensity with duration. I have already established an intensity level at the upper bound to achieve the V-12; a V-12 cannot be built faster than I have outlined in *An Operating System for the Human Organism*. Now that would be wasting your time. The alarm response we seek only comes through progressive fatigue and I will explain why soon enough.

When your training heart rate reaches and stabilizes on the termination heart rate, begin your cool down. The reason you stop here is if you continue training you will be in a catabolic state longer than you need to be and adrenergic response (being chased by a pack of wolves) escalates rapidly along with the odds of post-exercise immunosuppression (7.10, Hoffman-Goetz, 1996). You want to cause the minimum stress to trigger an adaptive response; you don't want to become totally glycogen depleted and venture into a ketotic, catabolic state burning more and more amino acids (8.17, Lemon, 1980). The cool down is where you gradually go from active training back down to a more normal state. Slowly bring your heart rate back down while reducing effort on the pedals over the course of five to ten minutes. Now that the workout is over and you have showered and cooled off, it is time for a carbohydrate meal. Now let's examine what you accomplished.

What you will notice from this training process over several weeks is that you will go longer and longer before reaching the termination heart rate. You will have to increase the load to achieve the target heart rate. You will feel better and better. Your productivity will increase and your energy level will soar. Your boss will ask what you have been doing. If nothing were physically changing, obviously this would not happen. At first, you should do this workout two to three days a week. As time goes

on, do this workout only twice a week and add a 60-minute session at the same intensity once a week. This training yields fantastic returns on your investment, your time. Don't get zealous and overdue, however, because overtraining can lead to overuse injuries that can become chronic. Cross training, which is doing different activities like rowing and stair machine instead of just the stationary bike, is an effective means, along with the Third Law's micronutritional profile, of reducing the odds of suffering overuse trauma.

When you get to the point where your body fat level is where you want, you can back off from this training regimen and switch into maintenance mode and just live off the dividend—true quality time. Going back to Karvonen's heart rate reserve method to re-calculate an initial heart rate, substitute 65% for 60% and do one hour at this target heart rate three times a week. Don't switch into maintenance mode until you have trained for at least six months doing progressive woolly mammoth workouts, however. The reason for the change in intensity is our goal has changed. In the beginning, we wanted to train in the most efficient way possible to build a bigger engine. Now, in maintenance mode, we want to continue to maximize the amount of fat oxidized during training while maintaining adrenergic and insulin sensitivity and as great as a percentage of our newly created endurance capacity as possible. In addition, we don't want to increase your daily maintenance caloric intake very much because there is a proven relationship between caloric intake and aging (this will be explored in great detail later). In other words, build the V-12 and live off the interest; your goal is not to train four hours a day, seven days a week for the rest of your life.

Historical Aspects of Endurance Training and a Primer on the Biochemistry of Energy Metabolism

Now I want to discuss why the woolly mammoth workout is the best form of training for mobilizing and utilizing body fat stores over the course of a digital man's lifespan. Moving forward from the days of woolly mammoth man by hundreds of millennia, we beam down into the modern times of ancient Greece, *circa* 1500 B.C. Around this time

we witness the first organized athletic events, with particular emphasis on the Olympic Games in Olympia, Greece around 776 B.C. (6.18, “Olympic...,” 1999). Athletes trained for the Olympic events, which were held every four years, but, of course, they had no technical understanding of what they were doing. Training became formalized for Greek youths for military purposes around 335 B.C. During the Roman Empire, there was a strong focus on preparation for war rather than competitive sport. They believed in training the body for combat, including swimming across torrents and other difficult feats. No real technology of training developed throughout most of written history because the unfolding of physiological and nutritional knowledge did not occur until the twentieth century.

In the late nineteenth century, there was more fear of too much trauma caused by athletic stress than one of blind daring. In 1874, Archibald Maclaren wrote a handbook entitled *Training in Theory and Practice*, which cautioned about making extreme and exceptional demands upon the energies of the human body. In 1881, Emil Du Bois-Reymond, the German founder of modern electrophysiology, wrote an essay on the science of “exercising” *all* of the human organism’s capabilities—and he was not referring to just athletic or even physical potential, but *all* abilities (6.18, Hoberman, 1992).

Moving into the present century and narrowing my focus to endurance training milestones, the first scientific study of circulation and respiration using a bicycle ergometer was published in 1913 by August Krogh, the Nobel prize-winning Danish physiologist (6.16, Krogh, 1913). In 1929, ATP, the body’s ultimate fuel, was discovered by Fiske and Subbarow in the U.S. and by Lohmann in Germany (6.18, Lehninger, 1975). The very first structured training program based on physiological knowledge was constructed by physiologist Hans Reindell for the 800-meter runner Rudolf Harbig (6.17, Boulay, 1995). Arthur Newton, the great English-born distance runner, published four books, in 1935, 1940, 1947, and 1949, that established a commonsense training method that is still valid today. His proposals of long slow distance training, training all year, and mental preparation, form the foundation for everyone today seeking increased endurance capacity, which will, hopefully, soon include you, the enlightened business professional (6.18, Noakes, 1991). In 1941, Fritz Lipmann hypothesized that ATP is a cyclic

carrier of chemical energy used universally to transfer energy in living cells (6.18, Lehninger, 1975). This was a giant leap forward for biochemistry.

Biochemists in the early half of the twentieth century burned the midnight oil to figure out how the body used carbohydrates and fats as fuels and under what conditions. Fiske and Subbarow, the discoverers of ATP, also discovered phosphocreatine two years earlier in 1927. But it was R.E. Davies, several years later, that discovered that an enzyme called creatine kinase catalyzes the conversion of phosphocreatine and ADP, which is like ATP, except only has two phosphate groups instead of three, into ATP and creatine. In other words, ADP is regenerated to ATP to be used for energy by the body by borrowing a phosphate group from phosphocreatine (6.18, Lehninger, 1975). When you hear in the news of strength athletes and baseball players using creatine to boost their performance, what they are doing is increasing their stores of phosphocreatine so that they can supply energy longer without fatigue. This is the first of four energy systems in the body and it is classified as anaerobic and alactic. This means that the CP-ATP system produces energy without the use of oxygen and does not produce lactic acid. In the days of woolly mammoth man and in the caveman game of football, it is used for immediate bursts of speed supplying high power for around five to seven seconds, just long enough to evade the saber-tooth or a linebacker or fire up the second energy system, called glycolysis.

The glycolytic pathway was heavily investigated by German biochemists in 1930s. Gustav Embden and Otto Meyerhof were given credit for the discovery, but Parnas, Cori, and Warburg helped elucidate the process. In glycolysis, or also known as the Embden-Meyerhof pathway in honor of its discoverers, glucose from the blood, or glucose cleaved from the glucose polymer glycogen, enters a series of reactions that results in pyruvate. Under conditions of sustained high power output, your muscles produce far more pyruvate than they can possibly accommodate, so pyruvate is rapidly converted to lactate. When you hear the term lactic acid used by athletes, just remember it came from pyruvate via glycolysis. Glycolysis, like the CP-ATP energy system, is an anaerobic process, but it produces lactic acid. Most people think lactic acid is only produced when exercise commences or when energy cannot be met from aerobic sources, but that is not true. It is a myth that you “go anaerobic” at some given exercise intensity. Actually, just sitting there and even

while you sleep, lactate is being produced, but in very small quantities. At low intensities, lactate and pyruvate are rapidly utilized for fuel by the third energy system, aerobic glycolysis, so lactic acid does not accumulate. There is a dynamic balance between lactate's production and utilization so there isn't a net increase in its concentration. The idea of "going anaerobic" is absurd because the third energy system, aerobic glycolysis, depends upon anaerobic glycolysis for its fuel! In fact, many physiologists believe the main purpose of glycolysis in slow twitch muscle is for feeding substrates into aerobic glycolysis. In other words, glycolysis begins with glucose and ends with pyruvate. Aerobic glycolysis takes the end-product of anaerobic glycolysis, pyruvate, and transforms it into CO_2 and water in the presence of oxygen. When you look at anaerobic and aerobic glycolysis as a single process, which is how the body looks at it, the body starts with glucose and ends with CO_2 and water producing 36 molecules of ATP for every molecule of glucose and six molecules of oxygen. Aerobic glycolysis is actually very complicated compared to anaerobic metabolism and I am only giving you a high altitude bird's eye view. This entire process of aerobic glycolysis is called respiration and is comprised of three sequential steps. The first step, the mobilization of acetyl-coenzyme A, begins with not only pyruvate as I told you before, but also with fatty acids and amino acids. The next phase, first proposed by Krebs in 1937, is called the Krebs' cycle, citric acid cycle, or tricarboxylic acid cycle. The Krebs cycle then feeds into the third stage, called the respiratory chain, where a series of electron carriers comprised of coenzyme Q and five cytochromes form a chain with the last cytochrome complex, called cytochrome oxidase, transfers electrons to oxygen forming water. CO_2 and water are the end products of respiration with the generated ATP being used to fuel muscle contraction and other processes requiring energy and the CO_2 enters the venous blood supply and is eventually exhaled as an exhaust gas from the lungs. [General overview of the integration of fuel supply with demand: (6.18, Hochachka, 1994). For general review of glycolysis and the tricarboxylic acid cycle: (6.3, Lehninger/"Glycolysis," 1975; Lehninger/"Carboxylic..." 1975). For in depth study, reference 6.2 and 6.3.] All of this metabolic machinery I have been talking about deals with carbohydrate metabolism — what about fats?

Figuring out fatty acid oxidation took a little longer and credit goes

to the Argentines Leloir and Muñoz and the Americans Lehninger and Kennedy between 1943 and 1948. We will examine fat metabolism in more detail later. Now we know how digital man gets his energy, the anaerobic processes, CP-ATP and glycolysis, which occur in the cell's fluid media called the cytosol, and the aerobic processes, respiration and fatty acid oxidation, which occur within the mitochondrion. Now, in the classical view of exercise metabolism, meaning as it is taught to physiologists, physicians, and fitness trainers, only three energy systems are described: the CP-ATP system or anaerobic-alactic, glycolysis or anaerobic-lactic, and the aerobic system. The governing position stand on aerobic fitness, published by the American College of Sports Medicine in 1978 and revised in 1990, is predicated on this view of metabolism (6.19, "American..." 1978; 6.19, "American..." 1990). I, and many others, think this is an oversimplification and has great ramifications in our digitally obese world today. What I mean is, by grouping fat and carbohydrate metabolism in a single pot called "aerobic energy system," it has left the door wide open to training practices and myths that are exercises of futility. Seneca, the great Roman philosopher, around A.D. 40, must have had a vision of health clubs in the 1990s when he said: "It is the superfluous things for which men sweat." Allow me to take you on a tour of some insights and discoveries that will show you what I mean.

Now that you endured the biochemistry lesson, you are prepared to know how all of these technical things apply to improving business productivity and quality of life. The first insight on fat metabolism occurred in 1974 in an experiment conducted by Linnarsson. He had a special room where he could control the concentration of oxygen in the room where a cyclist was working at an intensity of 65% of VO_2 max, a little harder than the woolly mammoth workout. The cyclist was subjected to three oxygen concentrations, 14%, 21% and 30%. For reference, 21% is normal air. When oxygen was in relative short supply at 14%, the respiratory quotient, or RQ, the ratio of CO_2 gas produced divided by the oxygen gas being consumed, was 0.965. A value of 1.000 describes a scenario where all fuel being oxidized is carbohydrate and a RQ of 0.707 is where 100% fats are being used. With normal oxygen concentration, 21%, the RQ was 0.889. Under hyperoxic conditions, 30%, the RQ dropped to 0.874. Converting the RQ values to fuel mixtures we

have as percent fat burned 11.2, 35.8, and 41.2 for oxygen concentrations of 14%, 21% and 30%, respectively. What this means is that in the presence of abundant oxygen at submaximal workloads, the body prefers to oxidize a higher fat to carbohydrate fuel substrate ratio. This same relationship occurs when ambient oxygen concentration is a constant and workload, as a function of VO_2 max, is varied (6.13, Linnarsson, 1974). In other words, at low intensities, such as walking, a greater percentage of fuel being oxidized is fats than at high intensities, where the fuel source is predominantly carbohydrate. From these experiments, it can be concluded that oxygen availability is an important regulatory control of the aerobic energy system.

The next insight was published in 1986 in the *Biochemistry of Exercise Series* in a paper entitled “Metabolic Changes Limiting Muscle Performance” by Kent Sahlin. In this paper, he describes the limitations of the three energy systems to produce ATP as a function of VO_2 max. He calls these limitations the carbohydrate threshold, the anaerobic threshold, and the PC threshold. This is not to be confused with the earlier German research describing the aerobic and anaerobic threshold by Hollmann, Wasserman and McIlroy between 1957 and 1964. The early German work, which was made famous by its application to the hour record in cycling by Francesco Moser in 1984 by the Italian sport scientist Conconi and his pupil Dr. Michele Ferrari, describes a method of training based upon plasma lactate concentrations as a function of training load. What Sahlin did was map the capacities of the energy systems from \emptyset to supramaximal loads and display the interrelationships between the systems. My focal point here is the concept of the carbohydrate threshold, which is the training intensity of maximal free fatty acid (FFA) oxidation. He cites a value of about 30% of VO_2 max for untrained subjects and 50% for elite marathon runners. The idea here is to define the training intensity where you are burning the *maximal* amount of fat, not just the greatest *ratio* of fats to carbohydrates. The highest ratio is when you are doing nothing or starving! Sahlin brought us one step closer to pay dirt (6.3, Sahlin, 1986).

Sahlin's paper outlined the limitations of the energy systems but it did not describe adaptive response to stress caused by training. Let's not forget stress and adaptation, homeostasis and survival. George Brooks *et al*, in 1994, published a paper in the *Journal of Applied Physiology*

entitled “Balance of carbohydrate and lipid utilization during exercise: the ‘crossover’ concept.” They defined the crossover point as the power output where carbohydrate substrate exceeds fat substrate, as a function of energy contribution, where higher intensities than the crossover point only broaden the relative contributions from carbohydrates and fats. A RQ of 0.85 is roughly the point where carbohydrate exceeds fat, as a function of calories burned. The most important contribution to physiology from this study, in my opinion, are their references to specific lipolytic adaptations, as opposed to general “aerobic” adaptations, from training protocols confined to training intensities approximating the crossover point (6.3, Brooks, 1994). The carbohydrate threshold and the crossover point, although not identical, are hinting at the same thing: the aerobic system should be treated as two systems, even though their processes overlap.

In 1995, John Hawley and Will Hopkins published a seminal paper on exercise metabolism in the *Sports Medicine* journal entitled “Aerobic Glycolytic and Aerobic Lipolytic Power Systems: A New Paradigm with Implications for Endurance and Ultra-endurance Events.” They came right out and said what needed to be said: the aerobic energy system should be divided into two functionally distinct aerobic power systems. There is an aerobic glycolytic system, just as I described earlier, which deals with carbohydrate metabolism. But now, there is a second aerobic system, meaning a fourth energy system overall, which is the lipolytic power system (6.3, Hawley, 1995). You, as a business professional, need to embrace this fourth system because the specific training of this system defines the process of *optimally burning fat* and impacts endurance capacity, which, as you will see later in the business segment, is the launching pad for your business productivity.

Now that you know how and why the lipolytic system was born, and who and where it came from, it is time to wield its power and lay waste to existing exercise protocols and their attendant myths. In order to optimize the process of burning fat using exercise, we need to examine three different metabolic circumstances of fuel substrate use. We also need to examine the chronic effects of the process, or, in other words, how does the body adapt to training stress over weeks, months, and years? It is the long-term structural benefits accrued from life style change that we seek—not the acute, finite, cause and effect approach

that is in vogue in the current fitness mania. The three circumstances are fuel use during exercise, fuel use immediately after exercise until metabolic rate resumes pre-exercise level, and fuel use under resting metabolic conditions. The net effect of these three conditions, as a long-term adaptive process, is the only measure of judging the relative worth of different exercise protocols. Let's begin with fuel use during exercise.

How Do I Leverage My Time To Optimally Increase Endurance Capacity?

First of all, a muscle cell does not know what exercise is, what 55% of VO_2 max is, how fat you are, or what you are trying to accomplish. It is just going to relax and contract on demand as long as it is able, and, if homeostasis is sufficiently disturbed and the right nutrients are supplied in time, it is going to adapt in a manner to minimize a specific stress or stresses should they happen in the near future. The sooner you realize that, the sooner you will stop equating your perceived hard exercise efforts with the outcomes you feel you deserve from such efforts. From the cell's point of view, it doesn't care how hard you work and cares even less about your grandiose expectations; it only cares about what happens to *it*, and *it* will respond to specific stresses according to *its* primordial genetic blueprint, not to your wishes. The point is, be concerned about what happens to *it*—*not to you*. Now, it is obvious that at low intensities, as expressed by a percentage of VO_2 max, that a greater contribution of energy is derived from fat than carbohydrate. For example, walking is a good way to conserve carbohydrate stores while burning mostly fat for fuel. For some people, walking is what they should do because higher intensity exercise is contraindicated for safety reasons and walking is vastly superior to doing nothing. The number of people that have lost significant amounts of fat through walking alone is legendary. It just takes a long time to get the results you seek.

In a study published in 1999 by *The New England Journal of Medicine*, 72,000 female nurses were analyzed on exercise habits. Nurses who walked one to three hours per week at a three mile per hour pace reduced their risk of a heart attack by 30% and those exceeding five hours

per week reduced their risk by 40% (6.8, Manson, 1999). No revelation here. The question is—is walking the optimal means of burning fat? The answer is no. The problem with walking (walking, *not* race walking, the Olympic sport) is fourfold: it does not burn the most fat per unit time, it does not cause sufficient stress on the lipolytic aerobic system to maximize adaptations to burn more fat the next time you do it unless you walk yourself into oblivion, it is not intensive enough to cause significant intramuscular triglyceride adaptations, and fast twitch fiber will never be recruited. Woolly mammoth man probably walked over three hours *per day* (I am being conservative here incredibly beyond reason) in order to survive, but digital man wouldn't survive in the digital age because he *would not have time* to meet all the deadlines screaming out in his cranium. The point being, what is the intensity, if there is one, which maximizes fat burning per unit time *and* causes sufficient stress to produce maximal lipolytic adaptations? The purpose of *The Digital Mantrap* is to elucidate an optimal approach—An Operating System for the Human Organism—that will exponentially improve business productivity *and* extend biological lifespan by switching the internal milieu from a competitive-survival pathway to a cooperative-synergistic pathway. *We deserve to live a life that the primordial genetic blueprint can potentially provide us.* I am *not* interested in, and you should not be interested in, finite, incremental, linear improvements to a fundamentally unsound, deleterious, self-defeating *modus operandi*. The motivation behind recent medical studies appears to be: what is the *minimum* the sedentary, obese masses have to do to reduce their exposure to cardiovascular disease, adult-onset diabetes, cancer, hypertension and so on (digital man disease) in terms of the *least* intrusive, innocuous forms of exercise, dietary modification, and stress reduction to minimize disturbance to their hypnotic, virtual reality universe so that the risk of biological Russian roulette is decreased from one in two to one in three or four by the time they are 50 years old. In other words, a focus on minimum wage physical activity, like walking, and minimum wage micronutrition, like the USRDAs, as a means to implement managed, chronic illness care—the heralded medical paradigm of the next millennium. Is this what the omnipotent creature called *Homo sapiens* has stooped to, the act of begging the digitally obese to walk three miles per hour one day per week to slightly loosen digital man diseases?

inevitable noose? If all you want out of life is postponing digital man disease while leading a life of digital bliss for about four or five decades or so, then a legion of little Dutch boys and vitamin hunters are at your beck and call. If you expect more out of life, then you need to be more mindful of how your body is designed to work. Let's move on to the discovery of optimal fat burning.

The study by Brooks *et al* in 1994 describes a metabolic environment conducive to our goal, and the earlier work by Sahlin does, as well. Woolly mammoth man obviously accomplished the goal, albeit inefficiently, but Arthur Newton, in the 1920s codified a long slow distance process before ATP was even discovered! In other words, he found the answer pragmatically instead of scientifically, by trial and error. To find the answer to this question using a scientific platform, you will not find the answer in the fitness world nor in the medical community, but in the athletic performance world, as I said earlier. The earliest technical roots probably were developed in Germany by Wasserman *et al* in the 1950s and 60s in their analysis of plasma lactate levels versus power output. Their findings were applied to long distance Nordic skiing events in Russia and the Scandinavian countries and to professional cycling with great success. A few decades later, this method was applied to Ironman distance triathlon events. Italian sport scientist Conconi influenced the European cycling to a profound degree after Francesco Moser's smashing of the hour record. In 1987, the Danish medical doctor Peter Janssen, who was the official physician for the cycling team PDM and then Panasonic, published a book called *Training Lactate Pulse-Rate*. He described training methods that specifically stress the four energy systems based on correlating plasma lactate levels to heart rate. For elite endurance runners, for example, he outlines training programs where 55% of the training time is conducted at heart rates below 140 beats per minute and 85% below 160 (6.3, Janssen, 1987). In 1989, Sleamaker, who had roots in Nordic skiing events with athletes from Scandinavia, Germany, and New Zealand, published a book called *Serious Training for Serious Athletes* (6.2, Sleamaker, 1989). Once again, the emphasis is placed on the vast majority of training time spent at 55 to 65% of VO_2 max. In 1995, Boulay wrote a paper in the *Sports Medicine* journal entitled "Physiological Monitoring of Elite Cyclists." Once again, the majority of the time is spent at an intensity of 60% of VO_2 max for extended periods (6.17, Boulay, 1995). The professional cyclists

that I have been associated with spend almost all of their off-season training time around 50 to 60% of VO_2 max, hour after hour. In 1975, Fitts *et al* published a paper in the *American Journal of Physiology* on endurance capacity as a function of training duration using rats. One group ran 10 minutes per day, the second group ran 30 minutes a day, the third group ran one hour per day, and the fourth group ran two hours per day, all at the same intensity for a total of 13 weeks. At the end, they were all tested for endurance capacity and the two hour a day rats blew away the one hour a day rats, and so on. There was a close relationship between muscle mitochondrial enzyme content and endurance capacity (6.2, Fitts, 1975). Remember about respiratory chains? In 1981, Davies *et al* published in the *Archives of Biochemistry and Biophysics* a paper on mitochondrial factors and endurance capacity. The exercise group, for the last six weeks of a 10-week training period, was running two hours per day and the sedentary group just sat around admiring their cage. At the end of the period, the sedentary rats lasted around 36 minutes and the trained rats had an endurance capacity of 183 minutes, 403% higher (6.2, Davies, 1981). In the athletic community of endurance athletes, why do they spend the lion's share of their precious time at such a low intensity?

The reason is very simple. This is the optimal process of increasing endurance capacity and of maximizing the rate of fatty acid oxidation while performing in competitive endurance events. These athletes do not compete at this low intensity; they *train* at this intensity. Of course, they train at other, higher intensities at the appropriate point in their yearly training cycle. I am not trying to convince you to become a Tour de France cyclist; instead, I am presenting you the training method that, when properly executed, is unequivocally the best and safest training method (when synchronized with micro- and macronutritional practice) to lose body fat.

Fat Oxidation During Exercise

How much fat do you burn when you exercise? Funny you should ask that. All of these great athletes I have been talking about don't do this training to lose fat, they do it to win races, and to win races of this type

you have to burn an enormous amount of fat or else you will run out of gas and hit the wall. The primordial genetic blueprint is designed to respond this way because in the days of the woolly mammoth, if it didn't, the vultures and hyenas would be picking your bones clean in a New York second. In other words, if low intensity prolonged endurance training did not cause a dramatic shift of fuel source from carbohydrate to fat, your liver would become glycogen depleted, your blood sugar would drop like a rock and you would get light headed, then you would slow to a crawl and the circling buzzards would initiate their landing procedure. It is all about survival, stress and adaptation, pure and simple.

There are many myths about fat burning while exercising that need to be clarified. Many factors enter into the equation including intensity, duration, fitness level, and glycogen levels. I want to examine three exercise protocols, the woolly mammoth workout, high-intensity aerobic exercise being defined as 80% or greater of VO_2 max, and resistance training. Resistance training, meaning heavy weight training or circuit training, for all intents and purposes oxidizes almost 0 fat for fuel during exercise but has benefits during the other two metabolic circumstances, namely after exercise and effects on resting metabolic rate. Let's separate fact from fiction by looking at some real measurements. In 1970, Pruett published a paper in *The Journal of Applied Physiology* on FFA mobilization in moderately well trained men. Using a bicycle ergometer, he examined RQ values at 20, 50, 70, and 85 to 90% of VO_2 max. During the first hour at 20%, the percent energy from fat was almost 60% and at six hours was nearly 74%. At 50% of VO_2 max during the first hour was 53% and during the last hour prior to exhaustion was 63%. For 70% of VO_2 max, during the first hour was 36% and the last hour 49%. At 85 to 90% VO_2 max the numbers varied between 3 and almost 13% (6.3, Pruett, 1970). David Costill conducted a study in 1970 in the *Journal of Applied Physiology* using college and national level distance runners. In a two-hour treadmill test at 55 to 67% of VO_2 max, the runners used 39% fat during the first 10 minutes, which then gradually shifted to 67% fat at two hours (6.5, Costill, 1970). In 1974, Ahlborg *et al* in *The Journal of Clinical Investigation* examined six untrained men in a four-hour bicycle session at 30% of VO_2 max. After 40 and 90 minutes, 37% of the fuel used was FFA, and after three hours the fat contribution rose to 50% and then at four hours increased again to 62%

(6.3, Ahlborg, 1974). These three studies are only validating the applicable models of the energy systems stated previously. We already knew that fat contributes more energy on a percentage basis when we walk or exercise at extremely low intensity than compared to any higher intensity we care to evaluate. These studies also show the significant difference between fat contributions in the trained versus the untrained state. All of these studies used a method called indirect calorimetry, which is not as accurate as methods using isotope tracers developed in the 1990s.

In 1993, Romijn *et al* published in the *American Journal of Physiology* a study of fat and carbohydrate utilization at 25, 65, and 85% of VO_2 max for different durations. They found at 30 minutes, an intensity of 65% of VO_2 max burns nearly 45% more fat than at 85% of VO_2 max. In other words, not just the fuel proportions, but also the actual *amount* of fat is drastically reduced at high intensity as opposed to maximal fat burning. At high intensity, fuel regulation favors aerobic glycolysis using pyruvate instead of starting with fatty acids. Using the new isotope tracer technology with five endurance trained cyclists, they discovered that *after 30 minutes* of exercise at 25 or 85% of VO_2 max, fat burning *quantities* were equal. This is actually a rather important fact to know because this study is using trained athletes who can oxidize *far more fat* than digital man can at 85% of VO_2 max (6.3, Romijn, 1993). In other words, in terms of how much fat is actually being burned, *not as a percentage*, a digital man walking at a moderate pace is burning more fat than that guy in the gym on the treadmill nearly having a heart attack and sweating bullets.

Remember that guy running the two miles at a 12 minute per mile pace four times a week earlier? Based on his level of fitness, what he is doing is essentially a waste of time. Yes, he will maintain systemic cardiopulmonary fitness and marginally increase insulin sensitivity, but it would do absolutely nothing to increase endurance capacity. The intensity is too low and the duration is too short. If you do the same training routine all the time after awhile there is no further improvement in your engine—the stress caused by training must be progressive. This is one of the biggest problems with walking. You are always *time constrained* which guarantees no progression. You violate the most fundamental law of physiology. Without an alarm phase, there can't be a resistance phase. In fact, the runner may even **detrain** on his four

times a week running regimen. He is only burning carbohydrate and a miniscule amount of fat! The sad thing is, most Americans, if they exercise at all, do something like he does and they wonder why they keep getting fatter. In gyms all across the country, you see signs above the exercise machines that say, “20 minutes maximum use.” This policy guarantees your failure.

At the other end of the spectrum, high intensity aerobic training or sprint training without a solid base of woolly mammoth training is a sucker bet. It is tempting because it has great benefits in the short term, including excellent fat burning adaptive responses for a while, but it is a diminishing returns process. In other words, all of its upside is given to you up front, but long term it has a ten-foot thick glass ceiling, an eternal plateau. For beginners it is a cardiovascular high risk gamble, and if you survive the initial stress you will eventually be stricken down with overtraining syndrome caused by nervous system over-stimulation and damage to mitochondria and enzyme systems from chronic acidosis. This would happen even to the world’s most gifted kilometer cyclists, 400-meter runners, and 1000-meter speed skaters that do this at the Olympic level. *Nobody* can maintain compliance of a steady diet of high lactate sprint training. Why even start?

How fast do changes occur to fat metabolism when you start doing the woolly mammoth workout? In 1996, Phillips *et al* published a paper in the *Journal of Applied Physiology* outlining the longitudinal effects to metabolism over a month’s time doing an endurance program like the woolly mammoth with untrained but active males. They used an even more refined method of isotope tracers than the Romijn study and examined in great detail the adaptations to fuel usage while performing two hours per day of ergometer cycling at 60% of VO_2 max with a day off every sixth day, which coincides very closely with the woolly mammoth workout’s intensity but not its frequency. They found that after only 31 days of training, fat oxidation at rest had increased 11% and fat oxidation during exercise had increased an incredible 58%. They found large decreases in lactate production, attenuation of adrenergic drive (do you remember adrenaline resistance?), and reduced reliance on glucose and glycogen stores (6.3, Phillips, 1996). You should be aware of two things that happen to you as you progressively adapt to the woolly mammoth workout: as you are able to go longer you burn more

fat with more fat being burned at the tail end of your workout than at the front end, and the fuel substrate ratio of fats to carbohydrates increases dramatically. Given these recent studies, and all of the older studies too, when it comes to burning fat while exercising, in just one month's time the woolly mammoth rules the entire fitness kingdom by a long shot and the advantage only widens the longer you invest your time. It is an increasing returns process, as opposed to a diminishing returns process, which means the more and longer you do it, the more and more dividends you get back in return. It also makes vividly clear, with volumes of impeccable research as support, that *if you exercise for less than 45 minutes, you will burn only a miniscule amount of fat, no matter what you do. Once you get into shape, life begins at one hour.*

Where Does Fat Come From While Exercising?

Prior to the 1970s, most muscle physiologists thought the majority of fat oxidized during exercise came from adipose cells and, today, the lay public today believes it comes from adipose cells. As evidence, the next time you are in the gym, ask a fitness trainer where fat comes from when you do moderately intense, aerobic training, or ask your doctor. They will tell you it comes from fat cells. In the 1970s, at least two studies discovered that a substantial proportion of FFA used for fuel at moderate exercise intensity were derived from intramuscular triglyceride storage (IMTG) (6.3, Carlson, 1971; 6.3, Essen, 1977). In terms of adaptation to woolly mammoth training, it is logical, but erroneous, to assume that, over time, adrenergic response increases which mobilizes more fat from fat cells than before, thus sparing carbohydrates. It certainly fits the gross model of energy substrate utilization that we know to be true and is still taught in universities—but it does not work that way. In reality, the primary training effect of endurance training on plasma FFA kinetics is to *decrease* fat mobilization from adipose cells (6.3, Martin, 1993).

In 1986, the first study to focus on IMTG depletion as a function of adaptation to training was conducted. Previous studies had discovered

that adrenergic response and plasma FFA concentration *decreased* with training. If fat mobilization from adipocytes decreased, overall fat oxidation increased and carbohydrate utilization decreased, then where was the fat coming from? Nine male subjects that had not performed endurance training for six months performed a 12-week endurance protocol. Cycling at 64% of VO_2 max for 90 to 120 minutes, prior to training 35% of the calories were derived from fat, and after completion of the program, 57%. By muscle biopsy, glycogen utilization dropped 41% and IMTG utilization increased 106% (6.3, Hurley, 1986).

In the 1990s, using the more sophisticated stable isotope tracer methods cited earlier, more accurate assessment of IMTG's role in metabolic process became accessible. In 1993, another 12-week longitudinal study was conducted to investigate, in detail, whole body fat oxidation, before and after training, during cycling for 90 to 120 minutes at 63% of VO_2 max. Fat oxidation increased by 41% but during the last 30 to 60 minutes of exercise, plasma palmitate (fat) oxidation was 27% lower than in the untrained state. The human body adapts to endurance training by sharply attenuating sympathoadrenal response (55% lower catecholamine concentration under these conditions) during training for up to 90 to 120 minutes, increases IMTG storage sufficient for two to three hours of exertion, and increases reliance on IMTG as a fuel substrate from approximately 50% (untrained) to 75% (trained) reliance at 63% of VO_2 max (broadly addresses moderate intensity applications) (6.3, Martin, 1993). A probable reason the primordial genetic blueprint implements these adaptations is to reduce adrenergic response (which is a systemic catabolic tactic) and compensates by increasing local IMTG storage that can be triggered by post-adrenergic mechanisms (a global to local shift which is less stressful on the organism). For example, at low intensities, like walking, it is fact that fat substrate is nearly exclusively derived by mild adrenergic stimulation to produce plasma-borne FFA from adipocytes. As intensity increases, fats mobilized from adipocytes *decreases* and is progressively supplemented by increased reliance on IMTG as a function of energy demand (6.3, Romijn, 1993). Up until the point of maximal fat oxidation, IMTG are providing the additional fat substrate, and then, beyond that point, glycogen must carry the load. As intensity reaches the mid-70s to 80% VO_2 (many variables here), both the utilization of IMTG and blood-borne FFA *decrease*, and

carbohydrate oxidation (glycogen) dominates (once again, many variables; I just want you to understand what the primordial genetic blueprint is doing, or attempting to do). During prolonged (2–3 hours) moderate work (45–65% VO_2 max), regulation and control priority shifts *again* to increased sympathoadrenal stimulation and increased plasma FFA concentration as the IMTG depot (and liver glycogen) is depleted. Survival is enhanced with nature’s four-tiered strategy: (1) At low intensity, woolly mammoth man could hunt and gather for hours and hours with minimal systemic consequence (low sympathoadrenal stimulus); (2) At moderate intensity, catabolic consequences are blunted by progressive reliance on IMTG instead of just “goosing” adrenaline to linearly mobilize more FFA from remote adipose cells (a brilliant non-linear solution nature has come up with!); (3) At high intensity, fat utilization is metabolically turned off and aerobic glycolysis is turned on because fat metabolism doesn’t get the job done; (4) After three hours at moderate intensity, IMTG, IM glycogen, and liver glycogen are running low and the sympathetic nervous system progressively “turns up” the catecholamines to mobilize body fat from the large energy depot available. Stress and survival are optimized by nature’s strategy.

Unfortunately, this doesn’t apply to digital man because his sympathetic nervous system and adrenoceptors are not calibrated by woolly mammoth man’s means of existence for any of the above to happen. Instead, harmful stress is magnified because of no endurance training (or is it no hunting and gathering?) to attenuate adrenergic response to stress (the pack of wolves in his mind), no mitochondria to burn fat because they atrophied away years ago, and enough fat stored around his waist and buttocks to walk around the equator one-hundred times without refueling.

What are the consequences of nature’s fat management strategy and how do we leverage nature’s wisdom? To answer that, we need to examine the fuel restoration process of IMTG. Understanding the relationship between IMTG metabolism, woolly mammoth training, and macronutrition is crucial to optimizing body fat management. First question: Can the muscle cell “reload” IMTG by converting carbohydrate into triglycerides? In adipocytes and hepatocytes, yes, *de novo* lipogenesis can happen (see pages 175 and 189), but in *cardiac and skeletal muscle cells*, IMTG replenishment is regulated by the lipoprotein

lipase enzyme, which increases clearance of **plasma** triglycerides for **intramuscular** lipid droplet storage (6.3, Oscai, 1990). That leaves two options: endogenous fat (from adipose cells) or exogenous fat (dietary source). The easiest way to restore IMTG is to just eat fat and the body will be very happy to shunt the dietary fat into the depleted muscle fibers and you are quickly reloaded to go train again (survival programs would be very happy by such a generous favor). Of course, being an intelligent, digital man, your cranium is going to be overwhelmed by a eureka effect of the highest order: Why did I just do that woolly mammoth workout for if I used very little fat (~25%) from adipose cells and I just reloaded my IMTG (~75%) from my high fat diet? It should be no surprise that stress, adaptation, and nutrition are interwoven. It elegantly explains why many endurance athletes, even though they train hours per day, can still have love handles to spare (imagine what happens if you consume a 40% fat diet and don't exercise hours per day, like a digital man). What you want to do is option B, the shell game; *force the survival programs to execute the adaptation scripts of mobilizing fat from adipose cells to muscle cells instead of from a bone-headed, high fat diet to muscle cells*. This is one of the most important insights of *The Digital Mantrap*: high fat diet = chase your tail. The primordial genetic blueprint Herself leaves no wiggle room here for "interpretation," you either do option B or you don't, your choice. The point being, to achieve the desired results, executing the most physiologically-sound exercise protocol is not enough: consummation of the benefits is only achieved by understanding how the organism responds to a given stress as a *system*. Now that we know how IMTG metabolism functions, we must demonstrate a modicum of intelligence and discipline to assure that dietary fat intake is kept a million light-years away from the primordial genetic blueprint's presence, or else you can expect squat for results. Let me ask you, what would be your reason for expecting results; your time, heart, effort, sweat, fatigue, courage and sacrifice? One more time, all of that counts for exactly \emptyset . All that gives you is the ammunition of rationalization to quit because you believe that your body, being so radically different than billions of other *Homo sapiens* (actually, mammals) that have walked the Earth since emerging from the primordial ooze, doesn't respond to the woolly mammoth workout due to bad genetics or some other lame excuse.

If you decide to execute option B, the primordial genetic blueprint is

forced to play Her hand. Nature is no stranger to a low fat diet; the primordial genetic blueprint already has the answer by increasing the plasma glycerol and FFA *at rest* two to three fold over sedentary, digital levels (6.3, Romijn, 1993). Nature is so skilled at this that She accomplishes this feat without increasing resting catecholamine concentration. How is this accomplished? Let's look at the source and the sink as a single system—just as the primordial genetic blueprint does. At the source end—the adipose cell—adaptations occur to the β -1-adrenoceptors (upregulation) on the adipocyte's membrane and/or to post-adrenergic receptor mechanisms like lipase activity (the enzyme that breaks apart stored fat and gets it ready to be released in to the blood). At the sink end—the muscle cell—the lipoprotein lipase enzyme (LPL) can greatly increase activity (opening the barn door for plasma FFA to enter the muscle cell where the fat is re-esterified as a triglyceride droplet) (6.3, Oscai, 1990). Nature mirrors this process with the relationship of hormonal regulation and control of glycogen resynthesis via the finely tuned relationships between β -cells in the Islets of Langerhans, muscle cells, insulin, plasma glucose, insulin receptors, and glycogen synthase (see “Woolly Mammoth Man, Digital Man, and the Metabolism of Fuel Restoration” on page 187). As evidence of the role in LPL in plasma triglyceride clearance, LPL-deficient mice (which, grimly, function similarly to the woefully dysfunctional digital man) are unable to transport fats into the muscle and have no triglyceride droplets. Resultantly, they suffer *plasma* triglyceride levels of 20,000 mg/dl (100 times normal, with digital man's blood tests not far behind *vis-à-vis* slothful diet/sedentary synergies in lieu of the mice's bona fide genetic aberrations), whereas normal mice are happy souls and have numerous droplets (6.3, Oscai, 1990). (For detailed information about resting FFA kinetics, see “Fat Oxidation at Rest” on page 89).

So nature has it all figured out. If you hunt, gather, and move all day long and eat a low fat diet (I hope you can see now why it is imperative that it is a low fat diet, don't you?) consisting of lots of fruits, vegetables, and micronutrients and you rest, relax and get good, sound sleep, then the primordial genetic blueprint is like a fish in water. Deviate from these delicately balanced expectations in anyway, and you are going to pay with digital man disease, as over 200 million Americans walk the plank—a very long plank—as you read this. And the destination? The digital mantrap, of course.

Energy Expenditure After Exercise, or EPOC

Fat burning once exercise is completed is probably a new concept to you and must sound like a dream come true. Imagine, doing nothing and burning fat! It is true, and let's find out how much and why. It is called excess post-exercise oxygen consumption, or EPOC. Many studies have been conducted in the last 15 years and I will share the results with you. This is actually a rather complicated area of research. Because of the complexity, many exercise equipment companies and popular diet programs have preyed upon certain findings and have applied them out of context by making erroneous claims. They will say just use XYZ machine for 20 minutes a day and you will burn millions of calories after exercise, and so on. Unfortunately, it does not work that way. When you look at the entire field of EPOC research, you arrive at a definite consensus—repeatability of predicted results given certain types, intensities, and durations of exercise protocols. As you can already imagine, there is no free lunch. If you perform endurance training at less than or equal to 80% of VO_2 max for less than or equal to 40 minutes, weight training or circuit training, the degree of EPOC is very low. I will summarize some examples from six different studies. Cycling or treadmill at 60 to 65% of VO_2 max for 30 minutes produced an EPOC of 15 to 17 calories (6.9, Sedlock, 1992). Cycling at 80% of VO_2 max for 40 minutes yielded an EPOC averaging 32 calories. 40 minutes of circuit training resulted in 49 calories. 40 minutes of heavy resistance training was good for 51 calories (6.9, Elliot, 1992). Many people have thought in the past that if you do endurance training and expend 300 calories then that will generate a handsome EPOC. Well, 300 calories of cycling at 75% of VO_2 max was worth 29 calories, 300 calories of cycling at 50% of VO_2 max was worth 14 calories, and 600 calories of cycling at 50% of VO_2 max was worth 12 calories (6.9, Sedlock, 1989). Walking or running the same distance of 3.2 km at 18, 33, 50, and 68% of VO_2 max resulted in EPOC ranging from 3 to 17 calories (6.9, Brehm, 1986). Treadmill at anaerobic threshold for 20 minutes and 1 mile per hour faster than anaerobic threshold for 40 minutes produced marginally elevated oxygen consumption rates over baseline for 40 and 100 minutes after exercise, respectively. They concluded there was no appreciable caloric loss beyond that produced by the training itself (6.9, Freedman-Akabas, 1985).

These are quite a few studies producing the same results. The exercise programs that the average digital man does produces only a very limited EPOC. I agree totally. Only the most blind optimist follows logic like—if you do 233.3 aerobics sessions with an EPOC of 15 calories each, that equals the amount of energy in 1 pound of fat. That is just the kind of linear thinking you can live without. Let's get a little smarter about this and start thinking the way the primordial genetic blueprint thinks.

In the EPOC research, there are two types of exercise protocols that have produced satisfactory results. These studies have focused on extremely high intensity aerobic exercise and prolonged sub-maximal endurance training. These studies go farther than just the average EPOC study by examining alterations in respiratory quotient and longer term effects such as modification to body composition.

In 1994, Tremblay *et al* compared a moderate to high intensity aerobic program against a sprint-training program with timed intervals. None of the subjects had been previously engaged in an exercise program or regularly was active in any physical activity. The first group increased in intensity to 85% of VO_2 max for 45 minutes with an overall training duration of 20 weeks. The sprint group trained for 15 weeks and performed multiple 30, 60, and 90-second sprints at very high anaerobic power levels, meaning greater than VO_2 max. Neither group lost any body weight but both lost body fat as measured by skinfold evaluation. The sprint group had a greater body fat loss than the high intensity endurance group. Both groups had increases in a fat burning enzyme marker with the sprint group producing a greater increase (6.9, Tremblay, 1994). This may seem surprising to you that a group that principally sprint trained lost more body fat than a high intensity endurance group, but it does not surprise me at all—especially with the test subjects being untrained. I used to train track-cycling sprinters and kilometer riders, they did this type of training, and they did lose body fat. However, with firsthand experience in this training and designing training plans with these methods, I would not wish this on my worst enemy. As I stated earlier, this is very high risk for the average digital man, it would have extremely poor compliance for anyone, and the odds of overtraining and immunosuppression are eminent. Physiologically, I understand the stress it imposes but it has no place in digital man's training plan or cerebrum. I personally do not prefer one training

program to another; I just want to choose what is safest, most effective, and sustainable over a lifetime, while being an increasing returns proposition. Theoretically, sprint training is effective, but I think I have shown that it is a very good example of what not to do.

In 1986, Maehlum *et al* published in *Metabolism* a study that examined the effects of cycling at 70% of VO_2 max for a total of 80 to 90 minutes, dividing the exercise periods into 10 to 30 minutes blocks with 5 minutes rest. The subjects were active but not trained so they were unable to maintain 70% of VO_2 max continuously. Six of the nine were unable to complete the full 90 minutes in spite of the rest periods. Respiratory exchange values, which are slightly different from RQ, were lower than control values for 24 hours post exercise. In the 12-hour period after exercise, oxygen consumption was elevated 14% over control values and with a significantly lower R-value, quite a bit more fat was being burned post exercise for a long time relative to the controls (6.9, Maehlum, 1986).

The Maehlum study demonstrates what happens to fat burning post-exercise while in a glycogen-depleted state, but the intensity is higher than the woolly mammoth pace. In 1985, Bielinski *et al* in *The American Journal of Clinical Nutrition* did a similar study at 52% of VO_2 max on a treadmill. The woolly mammoth is done at about 55% of VO_2 max. The subjects in this study were all active in sports, which differed from the Maehlum and Tremblay studies. This study resulted in an increase in resting metabolic rate, or RMR, of 4.7% 24 hours following exercise and a drop in RQ of 14%. Once again, the same results—significant acute rise in RMR and a big increase in fat to carbohydrate oxidation ratio (6.9, Bielinski, 1985). A third study by Bahr *et al* in 1987 duplicated the findings of these studies using cycling at 70% of VO_2 for 20, 40, and 80 minutes. They found that 12-hour EPOC was proportional to exercise duration and on the average amounted to 15% of the oxygen consumption during training. It is saying the same thing, the longer you go, the more the benefits. Changes to R-values were far more significant for the 80-minute session than either the 40 or 20 minute sessions (6.9, Bahr, 1987). In conclusion, for EPOC research, both sprint training and woolly mammoth training are the winners. Another mode of exercise not discussed here due to lack of peer-reviewed evidence is body building training. Since body building is also a means of

glycogen depleting most of the body's musculature, it would probably yield similar EPOC results to sprint training. If you started with a digitally inactive lifestyle, I would have to admit that EPOC from sprint training would be superior to the woolly mammoth for probably two to four weeks, but then after that the woolly mammoth would pull away. This is a tortoise and the hare race with the hare fast asleep beside the first mile marker.

Fat Oxidation at Rest

The third circumstance is effect of exercise on fat burning at rest after the EPOC period. We will now examine resting metabolic rate, or RMR, and changes to RQ while at rest. This is very important because 60 to 75% of daily (24-hour period) energy expenditure occurs while in the resting state. Even a small percentage change to either RMR or RQ has great impact on weight management on the catabolic half of the energy balance equation. The research findings on endurance training's impact on RMR are very mixed; some say it increases, others say it decreases. A very important fact to understand about RMR or BMR is that the liver, brain, heart, and kidneys comprise 58% of the RMR and they only compose 5.5% of the body mass. In other words, these four organs have a RMR 15 to 40 times as great as an equivalent mass of resting skeletal muscle (6.10, Elia, 1992; 6.10, Smith, 1999). What this means is that a 300 pound competitive body builder with veins bulging everywhere has a much *lower* (higher burns more calories) RMR per kilogram of lean body mass than a scrawny cyclist. Given the massive weighting factor imposed by these four energy-hog organs, don't expect large decreases or increases in RMR from any form of exercise, but it is possible to influence RQ and that is just as good.

You might be asking the question about now on why I included weight training in this comparison because it didn't burn any fat while training and only a few calories during EPOC. The reason is twofold: weight training is the best means available to maintain lean body mass while in a negative caloric balance, and while in neutral caloric balance it provides a means to increase lean body mass at the expense of fat

body mass. In fact, advocates of weight training have declared that lifting weights is a better weight management tool than aerobic training. Their position is based on the idea that an increase in lean body mass increases your body's net daily energy expenditure which obviously impacts caloric balance in your favor. This is true; the magnitude is erroneous. The temptation is to divide your daily energy expenditure by your lean body mass to arrive at a figure of caloric expenditure per kilogram of lean body weight. A typical figure is 25 calories per kilogram per day (this accounts for 60–75%, or 33–42 kcal/kg, of the daily total expenditure less exercise (6.10, Broeder, 1992; 8.15, Calloway, 1975)). If you gain three kilograms then you are burning an extra 75 calories per day, right? No! It's just another fable! In 1992, Elia published the breakdown of RMR, which included the 58% contribution by the four energy-hog organs. In other words, for each incremental kilogram increase in muscle mass, the increase in overall RMR is much lower than a linear, non-weighted average of 25 calories per kilogram. For the business professional that has no aspiration of becoming the Incredible Hulk, this is not the answer. It is, however, vital to your quality of life for many other reasons and is for those reasons just as valuable as the woolly mammoth workout. One important reason is that weight training impacts the production of the anabolic hormone testosterone and the woolly mammoth workout tends to attenuate testosterone secretion. The Six Laws of Biological Preservation are an interwoven network of checks and balances and the weight training aspect, called the saber-toothed tiger workout, balances the woolly mammoth workout in many ways. I believe that one of the findings in the Broeder *et al* study in 1992 published in the *American Journal of Clinical Nutrition* hit it right on the head. They said doing both endurance *and* resistance training prevented an attenuation of RMR while in a negative caloric balance (6.10, Broeder, 1992).

If we can't make big increases to RMR, than what else is there? We can affect the rate of fat oxidation in a big way. Let's see by how much. I will begin with studies that show what is possible, then I will look at what the average digital man can realistically expect in a short time period and include research confined to older populations such as 65-year old business professionals. In 1992, Tremblay *et al* published a paper in the *Canadian Journal of Physiology and Pharmacology* that studied RQ

at rest of endurance trained subjects versus sedentary to moderately active subjects. The RQ values were 0.83 for the trained and 0.86 for the untrained, corresponding to 56.2% and 45.9% energy derived from fat, respectively (6.10, Tremblay, 1992). Romijn *et al*, in 1993, published in the *Journal of Applied Physiology* a paper comparing resting fat oxidation of endurance-trained cyclists versus untrained controls. The athlete's free fatty acid levels were 38% higher than the controls even though RMR, when expressed on a per kilogram lean body mass basis, was not significantly greater. Furthermore, the rate of triglyceride and fatty acid cycling was almost four times higher and plasma glycerol and FFA levels were two to three times higher in the athletes (6.10, Romijn, 1993). In other words, if you are endurance-trained, the body at rest is releasing far more fat than it uses at rest, so it takes it out of circulation by a process called re-esterification. It takes energy to drive this recycling process and the primordial genetic blueprint doesn't want to do *anything* to waste energy for no reason. We will come back to this soon. In 1994, Klein *et al* in the *American Journal of Physiology* evaluated physically active subjects with no exercise program versus competitive distance runners. The runners had trained one to three hours per day, five to seven days per week for three to eight years. At rest, the runners burned 70% more fat per kilogram of bodyweight than the normal group (6.3, Klein, 1994). If you burned 70% more fat at rest than you do now, what would be the result?

The doubting-Thomas argument against the previous papers is that the athletes are gifted, that they have superior genetics, and, therefore, the data does not apply to doughboy digital men. Okay, then let's approach it longitudinally. In 1994, Eric Poehlman, one of the leading researchers in the field of RMR, and colleagues published in the *Journal of Applied Physiology* a paper that involved 18 subjects of approximately 65-years of age that were not involved in an exercise program. They did 8 weeks of endurance training only beginning with 200 calories worth of 60% of VO_2 max, then 65%, and finally 300 calories worth of 75% of VO_2 max. Resting fat oxidation increased by 22% with an increase of 7% in RMR (6.10, Poehlman, 1994). The 7% increase in RMR would be an added plus, and may be due to the age of the subjects, but the 22% increase in fat oxidation is remarkable. Granted, the 22% is not 38% or 70%, but you move in that direction one woolly mammoth workout at

a time. Sort of like each year, an oak tree adds one more ring to its trunk. The training program used here is not nearly as effective as the woolly mammoth and look what happened to these 65-year olds. Two other recent studies using older subjects had mixed results as far as modification of RMR relative to lean body mass for endurance trained versus untrained, as I would anticipate given the four energy-hog organ information stated earlier.

Adaptations to the Woolly Mammoth Workout

I want to discuss the disturbances to homeostasis and the consequent adaptations dispatched by the primordial genetic blueprint. First, you need to know a little bit about muscle fibers because they are not all created equal. There are two types of voluntary muscle cells—slow twitch and fast twitch. Slow twitch fibers do not supply much power but they have great tolerance to fatigue. Trained slow twitch fibers prefer fat for fuel. Fast twitch fibers are recruited when we need to exert great force or need to move explosively, but they cannot do it very many times or for very long before they fatigue unless properly trained. Fast twitch fibers are actually of two sub-types. The first sub-type are called fast oxidative glycolytic, or FOG, and the other are called fast glycolytic, or FG. FOG fibers in humans can be trained to have similar endurance properties as slow twitch and actually have greater oxidative capacity than slow twitch (6.2, Saltin, 1977) as opposed to rats (6.2, Baldwin, 1972). Although more difficult to stimulate with this kind of training, FG fibers will change in similar ways as FOG fibers. Now, when we were between woolly mammoth hunts, at first we only needed slow twitch because they could supply sufficient power and they had sufficient glycogen stores to last between hunts. You might ask—why not just have all slow twitch so we could last forever between hunts? We couldn't do that because we wouldn't have the speed or strength to get away from the saber-tooths! We need a balance between the two, thus another reason to perform the saber-toothed tiger workout.

As the hunt transpired, the slow twitch fibers that they were using in the beginning ran out of glycogen (6.12, Saltin, 1977). Even though slow

twitch fibers are rich in fat burning enzymes, fatty acid oxidation alone is insufficient to produce more than about 50% of VO_2 max in all but the most elite endurance athletes and about 30% in the unfit (6.3, Sahlin, 1986; 6.3, Newsholme, 1986, 2 refs). Even at a walking pace, some carbohydrate is being utilized. If you recall from the strength section earlier, there are two stressors that trigger motor unit recruitment—tension and fatigue. Prolonged low power output to fatigue is where all the magic is. The peripheral nervous system will then recruit other motor units, or groupings of muscle fibers, to use in their place as they fatigue. After awhile, you don't have enough slow twitch fibers to keep going, so the nervous system is forced to recruit up and supplement with FOG fibers (6.7, Gollnick, 1974). This is another unique reason why the woolly mammoth workout is superior to everything you have ever heard or done before. If you go long enough, FG fibers will be needed for you to continue and they will begrudgingly adapt just the way we want. When the FG fibers are recruited, it is kind of like having to use a Porsche to finish tending the crops because your John Deere tractor ran out of gas (I admit I robbed this analogy from a lecture I attended in 1990 from the esteemed German physiologist Dr. Deitmar Schmidtbleicher). The tractor is designed for the job, but the Porsche can do the tractor's work in an emergency. The evidence for this progressive recruitment during the woolly mammoth workout can be found from glycogen depletion studies measured at different stages of fatigue and with different exercise modalities, such as cycling and running. An important point to make is that progressive fatigue and recruitment occurs just as I have been outlining with cycling, **but not with running** (6.7, Costill, 1973). Even with trained distance runners, fast twitch glycogen stores are barely utilized even at 83% of VO_2 max going to exhaustion. It is difficult to say exactly why this is; it probably has something to do with survival, perhaps allowing for sufficient power to escape from a predator while in a fatigued state. Woolly mammoth man did not have bicycles! Cycling, on the other hand, will deplete almost all fast twitch glycogen stores at 64% of VO_2 max within 2 hours (6.7, Gollnick, 1973–4, 4 studies). That is a monstrous difference. This is very important to know because the preeminent factor in driving EPOC and RQ during rest is chronic and massive depletion of glycogen storage (with IMTG replenishment right behind). The average digital

man is composed of 45 to 55% slow twitch fibers, although this can vary greatly depending upon the specific function of a muscle. This means that if running is your principal exercise modality, you are leaving a tremendous magnitude of potential upside on the table compared to cycling. That is why cycling is the preferred exercise for the woolly mammoth workout. It is unconditionally guaranteed to work as advertised.

For all but the unfit, heart rate response during the woolly mammoth workout is very stable for a long time provided you maintain plasma volume by being adequately hydrated. You may not feel much through the majority of this workout, but a lot is happening. As fatigue progresses, fast twitch fibers become recruited in greater and greater quantities and heart rate will start to rise. This is what we have been waiting for. One of the reasons for the increase in cardiac output at constant power, or cardiovascular drift, is that the recruitment of higher threshold, lower efficiency motor units produces power with lower economy of oxygen utilization. For example, FG fibers are 18% less economical than slow twitch fibers (6.4, Willis, 1994).

There are several adaptations to the woolly mammoth workout and they all have great impact on our health and well-being. I am sure you will feel a lot better after one of these workouts than you would feel taking ephedra and buzzing around the ceiling like some wired mosquito. Your body interprets this training as a survival threat because exhausting your glycogen and IMTG depots and recruiting fast twitch fibers in the role of slow twitch fibers your body just doesn't want to have happen again. The training intensity of the woolly mammoth workout is just higher than the carbohydrate threshold, which means that the power demand barely exceeds the body's ability to supply the energy from aerobic lipolytic metabolism. In other words, you are burning fat at the maximal rate along with a minimal amount of glucose from the liver and glycogen from the muscle. Technically, the intensity can be derived by knowing the target heart rate as a function of the resting lactate level plus an increment, or decrementing from the heart rate corresponding to a plasma lactate concentration of 4-millimolar, which some researchers call the anaerobic threshold (6.5, Weltman, 1995; 6.3, Janssen/endurance training, 1987).

If you recall from the biochemistry lesson, if aerobic glycolysis cannot metabolize the pyruvate from glycolysis, lactate levels increase and

diffuses into the blood. An increasing lactate level guarantees you are burning carbohydrates and lactate also promotes re-esterification of plasma fatty acids. Not good. The stress being caused by this power demand is to principally improve aerobic glycolysis, not aerobic lipolytic metabolism. In other words, instead of lipolytic aerobic metabolism feeding acetyl-coenzyme A into aerobic glycolysis, carbohydrate is the source for acetyl-coenzyme A. The oxygen availability, measured by the oxygen tension inside the muscle cell, is involved in the regulation of this process. The front end of aerobic lipolytic metabolism is called the fatty acid cycle, and it consumes oxygen via the respiratory chain to produce acetyl-coenzyme A. This then feeds into the Krebs's cycle. When oxygen availability is insufficient to drive the oxygen demand of the respiratory chains for the fatty acid cycle *and* the third stage of respiration, there is a shift toward anaerobic glycolysis instead of the fatty acid cycle because *the pathway of glucose to acetyl-coenzyme A does not require oxygen* (6.4, Connett, 1990; 6.4, Gayeski, 1985; 6.4, Gayeski, 1987; 6.4, Wittenberg, 1989; 6.2, Davies, 1981). Furthermore, aerobic glycolysis produces twice as much ATP *per unit time* as aerobic lipolytic metabolism (0.5 vs. 0.24 millimole ATP per kilogram per second) and produces 11% more energy *per liter of oxygen* (18.9 vs. 21.0 kilo-Joule per liter) (6.3, Janssen, 1987). Don't confuse these more important parameters with fat producing 2.3 times as much energy per gram (4 vs. 9 kcal/g) as carbohydrate; *that is irrelevant while training beyond the carbohydrate threshold*. In high intensity aerobic exercise, there just aren't enough respiratory chains to go around because the demand for ATP exceeds the supply. Something has to give when ATP needs to be produced faster. What happens is fat metabolism is left holding the bag.

But what if you increase the quantity of respiratory chains? What if you increase capillary density? Glad you asked. In that case, the demand for any given respiratory chain decreases thus allowing the aerobic lipolytic pathway to function at a higher carbohydrate threshold. More capillaries—*and miles and miles of capillaries are going to be created*—means more oxygen is extracted from the arterial blood supply, which increases what is called arterial-venous difference (there is less oxygen in the venous return because it has been extracted). It simply means that there is more oxygen available to the increased number of respiratory chains (6.1, Andersen, 1975; 6.1, Andersen, 1977; 6.1, Ingjer,

1978; 6.1, Ingjer, 1979; 6.1, Brodal, 1977). You go from a two-lane goat path to a ten-lane fat burning superhighway. You did it. You built your very own V-12. I know this stuff is technical and may sound boring. You don't need to know all the details. However, what you do need to know is how to use the formula provided earlier because that will place you close to the right heart rate in the simplest way and how to coordinate pre-training and post-training macronutritional content. Remember, Einstein said: "Everything should be made as simple as possible, but not simpler." In response to this low intensity, progressive muscular fatigue, the body responds as follows: Mitochondrial density is increased in the exhausted fibers, glycogen storage is super compensated, capillary density increases, myoglobin, cytochromes and fat burning enzyme concentration increases, IMTG storage increases and the fat to carbohydrate burn ratio increases, which decreases your RQ on a **24-hour basis**.

I have used the term adrenergic drive several times in this segment and it means the same thing as the sympathetic nervous system. If you recall from the stress management segment, the catecholamines, adrenalin and noradrenalin, we already know they are triggered in the fight-or-flight response from stress caused by an attacking saber-tooth or a traffic jam; they also are the body's principal means of regulating the release of fats from the adipocytes into the blood to be used for fuel by the working muscles. Besides the adaptations that occur to metabolism inside your muscles, the most important thing to remember about the woolly mammoth workout is the radical changes that occur to the regulation of lipolysis, the release of fats from the adipocytes. To burn fat from stored fat around your waist or anyplace else, you need to do two things, you need to get it mobilized from storage and then you need to get it burned by aerobic lipolytic metabolism. This same mobilization process is how the IMTG depots are "reloaded" during the refractory period called rest, relaxation, and sleep (remember the First Law?) **provided your diet is low in fat** (the Fourth Law cometh). If you are a typical digital man, you don't do either of these things very well. The reason for that is the primordial genetic blueprint does not have a clue what to do with a sedentary, digital lifestyle. For example, adrenaline triggers lipolysis on the adipocyte's membrane through receptor sites called β -1-adrenoceptors (6.2, Limbird, 1988; 6.2, Viru, 1995; 6.2, Wahrenberg, 1987). In the Tremblay, Romijn, and Pohlman studies

(6.10, multiple studies), the resting RQ was lower and the resting adrenaline and noradrenaline levels were about the same when comparing the trained to the untrained state. When you divide the resting rate of lipolysis by the fat body mass, you realize that trained adipocytes have much greater sensitivity to adrenaline, probably because of some change to the β -1-adrenoceptors or to the activation process of the enzymes that mobilize fat, called lipases. On the other side of the coin, untrained adipocytes are adrenaline resistant relative to the trained state. The trained subjects are mobilizing far more plasma lipids at rest, the rate of triglyceride and fatty acid cycling is almost four times higher, and rate of fat oxidation is 22 to 38% greater, depending upon which study you look at. Digital man, in spite of large amounts of adrenaline secretion when the “hunt” commences, mobilizes very little fat relative to being in the trained state. In fact, in the Romijn study, they found that the resting lipid kinetics of the trained athletes were greater than the rates found in subjects after 84 hours of *starvation* (6.10, Romijn, 1993). In other words, you are mobilizing fat faster than if you were starving on the miraculous, infomercialized, high protein, emperor’s new diet except you are eating well and feeling like you can walk on water. Neat trick, wouldn’t you say?

Benefits to the Business Professional

Now I am going to clarify what all of this means to the business professional as interpreted by the primordial genetic blueprint. All of the research findings I have summarized on fat mobilization and oxidation make perfect sense when you take the trained subjects I have told you about and substitute them with woolly mammoth man. For woolly mammoth man to survive, he did a mixed intensity, longer duration version of the woolly mammoth workout every single day of his life. Every day he was depleting liver and muscle glycogen stores and IMTG stores in the process of finding food. The reason for the lower resting RQ is obvious; with a lower RQ that means more ingested carbohydrate is available for conversion to glycogen and also less carbohydrate is being used at rest and on the hunt and climbing a tree, and

hiding from a saber-tooth, and so on. *That is what a lower RQ means*— at a given level of energy expenditure, you are using more fats relative to carbohydrate for fuel. The reason for the amplified lipolysis and glycerol and fatty acid re-esterification—the massive recycling process that is like stepping on the gas and brake at the same time—seems like a futile, wasteful process, out of character for the primordial genetic blueprint’s inherent intelligence, but it isn’t. It does this to get a jumpstart on burning fat instead of glycogen when the hunt commences and it is the process to mobilize fat from adipose cells into muscle cells to reload IMTG between hunts. In trained as well as untrained subjects, it takes a good hour or longer for adrenergic response to drop RQ to a level where fat metabolism (fats coming from the plasma) is at full throttle (6.3, multiple references). By having a low RQ on standby 24 hours a day, this long ramp up time is negated. The cost? The cost is burning more fat instead of carbohydrate, *something the body wants to do anyways*. The body can have its cake and eat it too; use more fats at rest to accelerate glycogen re-synthesis *and* mobilize fat from adipose cells to reload IMTG. Neat trick, again. The body is trying to protect the limited capacity glycogen stores; what better way than burning fats instead of carbohydrate? If it didn’t work that way, your survival is threatened because without adequate glycogen stores your brain doesn’t function well and you can’t produce enough power to escape from a saber-tooth or hunt a woolly mammoth. When glycogen stores are being restored, the body replenishes muscle stores before the liver. This must have to do with survival; the physical ability to capture food and prevent becoming food takes precedence over central navigation. I don’t know why. We will have to ask the primordial genetic blueprint.

The unbelievable sensitivity to adrenaline, especially when expressed as a function of fat body mass, means a small amount of fight-or-flight response yields a lot of get up and go. Woolly mammoth man, when in hot pursuit during a long hunt, used much less adrenaline, liver glycogen, and muscle glycogen than digital man doing the same hunt. And finally, woolly mammoth man, when triggered by flight-or-fight, used much less adrenaline than digital man. This has grave circumstances. Because digital man, living a digital life, perceives so many saber-toothed tiger attacks every single day and his sympathetic nervous system is not calibrated like woolly mammoth man’s, digital man is causing

enormous stress and damage to himself because of the large secretion of catecholamines for no real reason. *What I am saying is that if digital man does the woolly mammoth workout religiously, that one of the adaptations is much less self-inflicted damage caused by the numerous phantom saber-toothed tiger attacks that plague him nearly every waking second.* To reduce negative stress, you can eliminate the cause of the stress and/or minimize the physiological impact of the triggered phantom threat. Thus, much of the answer to the First Law of Biological Preservation is being an obedient scholar of the Second Law.

Wait. There are more benefits to discovering and using your body as nature intended. Our primordial genetic blueprint is hardwired to make it virtually impossible to store excess carbohydrate as body fat—*de novo* fat synthesis is inhibited. It would have been suicidal if this *did not* happen. Why does it happen? The enzyme that stores glucose as glycogen, glycogen synthase, is strongly upregulated and the muscle and adipose membrane's sensitivity to insulin is greatly increased. Woolly mammoth man tapped into this adaptive response on a daily basis—digital man does not. This is one reason—and there are more—why woolly mammoth man couldn't get fat and digital man can't stop getting fat. It worked for woolly mammoth man thousands of years ago and Tour de France cyclists and it will work for you. Over the course of weeks and months, you are slowly building a V-12 that burns more and more fat—24 hours a day, seven days a week, and on holidays.

A good example of how this works conceptually is the principle of compound interest. When you first start saving money and collecting interest, you work hard to build up your principal but very little money is contributed by interest relative to your personal efforts. As time goes on, and assuming you are still making equal contributions, a point is reached where the contributions from interest exceed the contributions from your effort. From that point on, you can semi-retire and live off the interest. In direct comparison, your body fat represents debt; your endurance capacity, RQ and daily activity are your job income sources; and your food intake represents your expenses and taxes. If you eat more than you earn in fat-calorie credits from your training, sleep, and daily activity, you get fatter. Now, if you get promoted and start to earn more fat-calorie credits than you eat, you start to lose some of the debt. At this point you are probably still in serious debt so you need quite a

few more promotions. As you get promoted to upper management, you will reach a point where you are receiving more **additional** fat-calorie credits from your sleep and daily activity per day than you would have received from 60 minutes of training on your first day of training. Please note that you couldn't even do 60 minutes of training on day one — and now you are hardly doing any training at all! This is the goal; you want to reach the maintenance phase, where you are promoted to CEO of Your Body, Inc. You no longer are a lowly laborer putting in all those hours in the engine-building phase just described. You will be able to lose body fat doing just maintenance-level exercise — all because you are living off the interest generated by your V-12. I hope you are not one of the millions of Americans shoehorning in 20 to 30 minute aerobic sessions three to four times a week because you believe that is going to make you fit and thin. That process over a lifetime is like existing paycheck to paycheck and still going into debt while the woolly mammoth workout is like living dividend to dividend and generating compound interest.

You might be thinking, wait a minute. Are you telling me if I stick to this training program that I will be burning more fat clicking my mouse and watching TV per day than doing 60 minutes of training the day I started? Yes. As you convert non-endurance trained fast twitch fibers into endurance-trained fast twitch fibers and non-trained slow twitch fibers into highly trained slow twitch fibers, one of the natural wonders of the world is unveiled — a turbocharged fat burning V-12 and all the health benefits that no Dutch boy will ever produce. Woolly mammoth man inside is willing to give you this — and a lot more — for free. It has a multi-million-year track record. It is safer than any other form of training except walking. It works incredibly well for 65-year old digital men and women. The woolly mammoth workout will burn more fat than any other form of training over the course of a digital lifespan simply because that is how we were designed to work. The most amazing thing about the woolly mammoth workout and its relationship to the other laws is that they have been asleep under your nose ever since woolly mammoth man gazed at the stars. It's up to you to recognize and seize it; it's part of your lost inheritance.