# *2) SKELETAL MUSCLES*

## *a) Basic Skeletal Muscle Physiology*

This is intended to be a bare-bones review of physiology of muscle function. There are numerous sources on the internet for those who are interested in a more in-depth exploration of skeletal muscle physiology. The concepts here have direct application to understanding how specific training improves (or decreases) endurance performance capacity.

### Basic Architecture

A single muscle fiber is a cyclindrical, elongated cell. Muscle cells can be extremely short, or long. The sartorious muscle contains single fibers that are at least 30 cm long. Each fiber is surrounded by a thin layer of connective tissue called endomysium. Organizationally, thousands of muscle fibers are wrapped by a thin layer of connective tissue called the perimysium to form a muscle bundle. Groups of muscle bundles that join into a tendon at each end are called muscle groups, or simply muscles. The biceps muscle is an example. The entire muscle is surrounded by a protective sheath called the epimysium. Between and within the muscle cells is a complex latticework of connective tissue, resembling struts and crossbeams that help to maintain the integrity of the muscle during contraction and strain. It is an amazing cellular system even before it contracts!

### Interior Components

Every muscle cell contains a series of common components that are directly associated with contraction in some way, and influenced by training. I will briefly describe these. For now we will not worry about the rest (like the nucleus, ribosomes etc.).

**1. The Cell Membrane:** Controls what enters and leaves the cell. Contains regulatory proteins that are influenced by hormones like epinephrine (adrenalin) and insulin. The blood concentration of these hormones greatly influences fuel utilization by the muscle cell.

**2. Contractile Proteins:**- The contractile machinery of a muscle fiber is organized into structural units called sarcomeres. Muscle length is determined by how many sarcomeres are lined up in series, one next to the other. Muscle thickness ultimately depends on how many sarcomeres line up

in parallel (one on top of the other). The sarcomere structures consist of two important proteins, actin and myosin (about 85% by volume). Several other important proteins called troponin and tropomyosin, and proteins with cool names like titin, nebulin, and desmin help to hold these units together. The sarcomeres are organized as many thin myofibrils. A single muscle fiber will contain 5 to 10,000 myofibrils. Each myofibril in turn contains about 4500 sarcomeres. Multiply the number of muscles in the body by the number of muscle fibers per muscle by the number of myofibrils per fiber by the number of sarcomeres per myofibril and well, the numbers become pretty staggering. It is the individual myofibrils, long chains of sarcomeres, which actually produce force in the muscle cell. All of the rest of the machinery plays a supporting or repair function.

**3. The Cytosol:** This is the aqueous fluid of the cell. It provides a medium for diffusion and movement of oxygen, new proteins, and ATP within the cell's interior. The cytoplasm also contains glycogen, lipid droplets, phosphocreatine, various chemical ions like magnesium, potassium and chloride, and numerous enzymes.

**4. Mitochondria**: - The organelles in each muscle cell that contain oxidative enzymes consume oxygen during exercise. Recent research suggests that mitochondria may look more like an interconnected network than little isolated oval "powerhouses" shown in most old textbooks. Mitochondria convert the chemical energy contained in fat and carbohydrate to ATP, the only energy source that can be used directly by the cell to support contraction. Ultimately, glucose and fat molecules (and certain amino acids) break down and combine with oxygen to form ATP, carbon dioxide, water, and heat energy. This occurs via enzymatic processes occurring first in the cytosol and then the mitochondria. The carbon dioxide and excess water leave the body through our breath. The ATP generated provides a usable energy source for muscle contraction and other cell functions. Heat removal occurs by sweating and as radiant heat transfer from the skin to the surrounding air. Clearly, each by-product of energy metabolism has significance to the exercising athlete.

**5. Capillaries**: - These microscopic size blood vessels are not actually part of the muscle cell. Instead, capillaries physically link the muscle with the cardiovascular system. Each muscle cell may have from 3 to as many as 8 capillaries directly in contact with it, depending on fiber-type and training. One square inch of muscle cross-section contains 125,000 to 250,000 capillaries! The volume of blood forced through the heart's aorta (about the diameter of a heavy duty garden hose) is spread so thin among the billions of capillaries

that red blood cells must squeeze through in single file like soldiers marching along a path. Distributing the blood flow through such an immense network of vessels is critical so every individual cell maintains a supply line and waste removal system. This and other “infrastructural challenges” are the price multi-celled organisms (we humans) pay for our complex organization. Endurance exercise increases the demands on nutrient supply and waste removal, but also stimulates the growth of more capillaries. Endurance training improves the delivery and removal function of this fantastic network of vessels. The total number of capillaries per muscle in endurance-trained athletes is about 40% higher than in untrained persons. Interestingly, this is about the same as the difference in VO2 max between well-trained and untrained people. In contrast, strength training tends to decrease the capillary to muscle fiber diameter ratio. This occurs because muscle fibers grow in diameter, but the number of capillaries essentially remains unaltered.

## The Motor Unit

A motor unit is the name given to a single alpha motor neuron and all the muscle fibers it activates (neurophysiologists use the term innervates). With 250 million skeletal muscle fibers in the body (give or take a few million), and about 420,000 motor neurons, the average motor neuron branches out to stimulate about 600 muscle fibers. Interestingly, large muscles may have as many as 2000 fibers per motor unit, while the tiny eye muscles may have only 10 or so fibers per motor unit. The size of a motor unit varies considerably according to the muscle’s function. Muscles with high force demands but low fine control demands (like a quadriceps muscle) are organized into larger motor units. Muscles controlling high precision movements like those required in the fingers or the eyes are organized into smaller motor units. The motor neuron branches into many terminals, and each terminal innervates a specific muscle fiber. The motor unit is the brain’s smallest functional unit of force development control; if a motor unit comprising 600 muscle fibers in the left biceps is stimulated, then all 600 of those fibers will contract simultaneously and contribute to the total force produced by the biceps. The brain cannot stimulate individual fibers one at a time. Even for our sophisticated nervous system, that would require far too much wiring.

## Regulation of Muscular Force

The brain combines two control mechanisms to regulate the force a single muscle produces. The first is **RECRUITMENT**. The motor units that make up

a muscle are not recruited in a random fashion. Motor units are recruited according to the *Size Principle*. Smaller motor units (fewer muscle fibers) have a small motor neuron and a low threshold for activation. These units are recruited first. As more force is demanded by an activity, progressively larger motor units are recruited. This has great functional significance. When requirements for force are low, but control demands are high (writing, playing the piano) the ability to recruit only a few muscle fibers gives the possibility of fine control. As more force is needed the impact of each new motor unit on total force production becomes greater. It is also important to know that the smaller motor units are generally slow units, while the larger motor units are composed of fast twitch fibers.

The second method of force regulation is called **RATE CODING**. Within a given motor unit there is a range of firing frequencies. Slow units operate at a lower frequency range than faster units. Within that range, the force generated by a motor unit increases with increasing firing frequency. If an action potential reaches a muscle fiber before it has completely relaxed from a previous impulse, then force summation will occur. By this method, firing frequency affects muscular force generated by each motor unit.

**Firing Pattern**

If we try and relate firing pattern to exercise intensity, we see this pattern. At low exercise intensities, like walking or slow running, slow twitch fibers are selectively utilized because they have the lowest threshold for recruitment. If we suddenly increase the pace to a sprint, the larger fast units will be recruited. In general, as the intensity of exercise increases in any muscle, the contribution of the fast fibers will increase.

For the muscle, intensity translates to force per contraction and contraction frequency/minute. Motor unit recruitment is regulated by required force. In the unfatigued muscle, a sufficient number of motor units will be recruited to supply the desired force. Initially desired force may be accomplished with little or no involvement of fast motor units. However, as slow units become fatigued and fail to produce force, fast units will be recruited as the brain attempts to maintain desired force production by recruiting more motor units. Consequently, the same force production in fatigued muscle will require a greater number of motor units. This additional recruitment brings in fast, fatiguable motor units. Consequently, fatigue will be accelerated toward the end of long or severe bouts due to the increased lactate produced by the late recruitment of fast units.

Specific athletic groups may differ in the control of the motor units. Top athletes in the explosive sports like Olympic weightlifting or the high jump appear to have the ability to recruit nearly all of their motor units in a simultaneous or *synchronous* fashion. In contrast, the firing pattern of endurance athletes becomes more *asynchronous*. During continuous contractions, some units are firing while others recover, providing a built in recovery period. Inital gains in strength associated with a weight training program are due to improved recruitment, not muscle hypertrophy.

## *b) Training Adaptations in Skeletal Muscle*

### Introduction

**Adaptability** is a fundamental characteristic of skeletal muscle (and the body in general). The nature of this adaptation can be summarized using the following principle: cells will adapt in a manner that tends to minimize any movement away from **homeostasis**, or resting conditions. In exercise physiology we refer to the acute changes that occur in a sytem, organ, or cell during exercise as **responses**. An example is the increase in heart rate that occurs when we jump up from our chair and start jogging. The long-term changes that occur as a result of repeated bouts of exercise are called **adaptations**. Cellular adaptations generally involve an increase or decrease in the rate of synthesis of a specific cellular protein. All muscle cells are in a constant state of synthesis and degradation. If synthesis rate exceeds degradation rate, an increase in the cellular component occurs. A change in protein synthesis requires a cellular signal. Biologists and physiologists continue to explore the communiction process by which different forms of muscular work induce cellular changes. At the cellular level, there are some theories, but no complete understanding. However, we do know quite a bit about **what** adaptations do occur, even if all the details regarding **how** remain unclear just yet.

### Contrast Between Maximal Strength and Maximal Endurance

If we could build a skeletal muscle for the purpose of endurance, what would the recipe be? Since the heart is the supreme endurance muscle, let's cheat by taking a look at it first.

**Characteristics of Fatigue Resistant Muscle Cells**

• Heart cells are **smaller** in diameter than skeletal muscle cells. This results in very short diffusion distance between oxygen molecules coming from capillaries and the mitochondria where they are used.

• The surrounding network of capillaries is extremely well developed. This characteristic also facilitates even and rapid oxygen distribution to all myocardial cells.

• The mitochondrial density of heart cells is extremely high, 20-25% of cell volume in adults. Mitochondria use oxygen to metabolise carbohydrate and fat and produce ATP.

• The cytoplasmic enzymes responsible for breaking down fatty acid molecules into 2 carbon fragments that can enter the mitochondria are present in high concentrations.

• Contractile protein makes up about 60% of cell volume. The ATPase subtype found in heart is slower than that seen in skeletal muscle. Consequently, the rate of force development is slower, although absolute tension/cell diameter is the same.

• Heart lactate dehydrogenase, the enzyme that converts pyruvate to lactic acid competes poorly with pyruvate dehydrogenase. This contributes to the very low lactate production in heart cells despite high metabolic flux. So, heart cells display almost zero fatiguability due to the tremendous capacity they have to receive and consume oxygen. Fatigue resistance is traded for anaerobic capacity. This is why the heart has little tolerance for oxygen deprivation, the dreaded heart attack. If we want to build a skeletal muscle that is highly fatigue resistant, it must resemble heart muscle in its basic features.

Now let's build a muscle that is optimized for brief efforts and maximum force production. Here are the characteristics needed.

**Characteristics of Maximal Strength Muscle Cells**

o Each muscle cell should contain a high volume of contractile protein. Since oxygen diffusion is not a concern, making the cell diameter larger will help it hold more contractile protein (actin and myosin).

o To make more room for actin and myosin, mitochondrial density should be minimized to that necessary to maintain resting cell function.

o Since fat can only be metabolized aerobically, high levels of fat- cleaving enzymes in the cytosol are also unnecessary.

o The capacity for anaerobic glycolysis should be high to allow brief but high capacity energy production without oxygen. The capacity for lactic acid production should be high.

What you should notice is that these two lists are exactly opposite. The optimal muscle for endurance CAN NEVER be maximally strong or powerful. And the muscle fiber that produces the most force CANNOT be optimally developed for endurance as well. The two conditions are mutually exclusive. This is one of the most important concepts to understand when designing a training program.

**Three Points to Remember:**

o There are identifiable proteins in the muscle that contribute to its ability to produce high force at high rates (strength and power respectively).

o There are also identifiable proteins and structural characteristics that confer high fatigue resistance (endurance).

o There is no identifiable specific protein or structure that confers the quality "Strength-Endurance". When we train for strength-endurance, what we are really doing is training in a way that fails to stimulate either strength or endurance adaptations optimally. An example of this "best of neither worlds" approach is circuit training.

As a coach / athlete, your sucess begins with your ability to accurately understand the muscular demands of your sport. Then, a training program can be designed that will result in muscular development suited to the

combination of strength and endurance that your sport requires. Here are two real world examples.

**Strength and Endurance in the Proper Dose**

**Example Number One:**

In 1994, I joined three other rowers in Austin, Texas to train for the Masters National Championships in the 4x. One of my teammates was a fellow named Jason. Then 29, he is a computer engineer, former varsity lightweight sweep rower at Cornell University, turned sculler. Now several years out of college, Jason was no longer a lightweight. He carried 190 pounds of muscle on his frame, and little fat. Jason had been training on his own for several years. His routine was demanding. He did a lot of weight training in the gym. On the water 6 days a week in his 1x, he almost always performed intervals. These usually consisted of 1 to 4 minutes race pace intervals with lengthy recovery between. Both the volume (total number of intervals), and intensity of rowing training were quite high. This training program had generated an athlete with excellent anaerobic capacity, and excellent speed through 500 meters of a rowing race. Unfortunately, Masters rowing races are 1000 meters and the open events are 2000! At the Masters that year, Jason showed two weaknesses that were a direct result of his training. He began to bog down over the last 200 meters, and his overall performance deteriorated over the course of 4 days of heats and finals. He was fast enough to reach three finals in 1x, 2x, and 4x competition, but left with only a silver in the 4x to show for it.

He (and we) wanted more. Each of us had specific work to do for 95. As the exercise physiologist on the team, I made a few suggestions. Jason had to put away the barbells and go back to the basics of endurance training. He had emphasized strength training too much. Despite training with great intensity, He had lost much of the aerobic capacity he had as a lightweight in college. He had no aerobic base!

The weight training volume was reduced. Almost all interval training was eliminated from his training for 6 months and replaced by long, steady state rows of 60 to 90 minutes, and road cycling. After 6 months, interval training was re-introduced, but the interval length was extended to 4, 8, or 20 minutes depending on the specific workout. Steady state training remained a major component of the total training volume. Intensity was monitored with heart rate. Only in the final month before the competition were short (500) meter, supra-race pace intervals included in the training program.

The anaecdotal results observed in the final months of training were a slightly less muscular athlete who retained his 500 meter speed, but now was difficult to hold off in a 20 minute interval as well. The hard (metallic) evidence was even more obvious: Three gold medals at the 1995 Masters Nationals (4x, Mx 8+, and 8+). Two finals were held within one hour of each other, with the second being the all important 4x final we had lost in 94. A 0.7 sec loss became a 2.5 sec victory in the 3 minute race. Recovery was never a problem, and after 500 meters the race was never in doubt. Of course, Jason wasn't the only guy in the boat. But, we all followed the same training program.

**Take Home Message:** Even for races that only last 3 minutes, endurance capacity is critical! Too much emphasis on strength training results in a sub-optimal muscular system for the competitive task.

**Example Number Two**

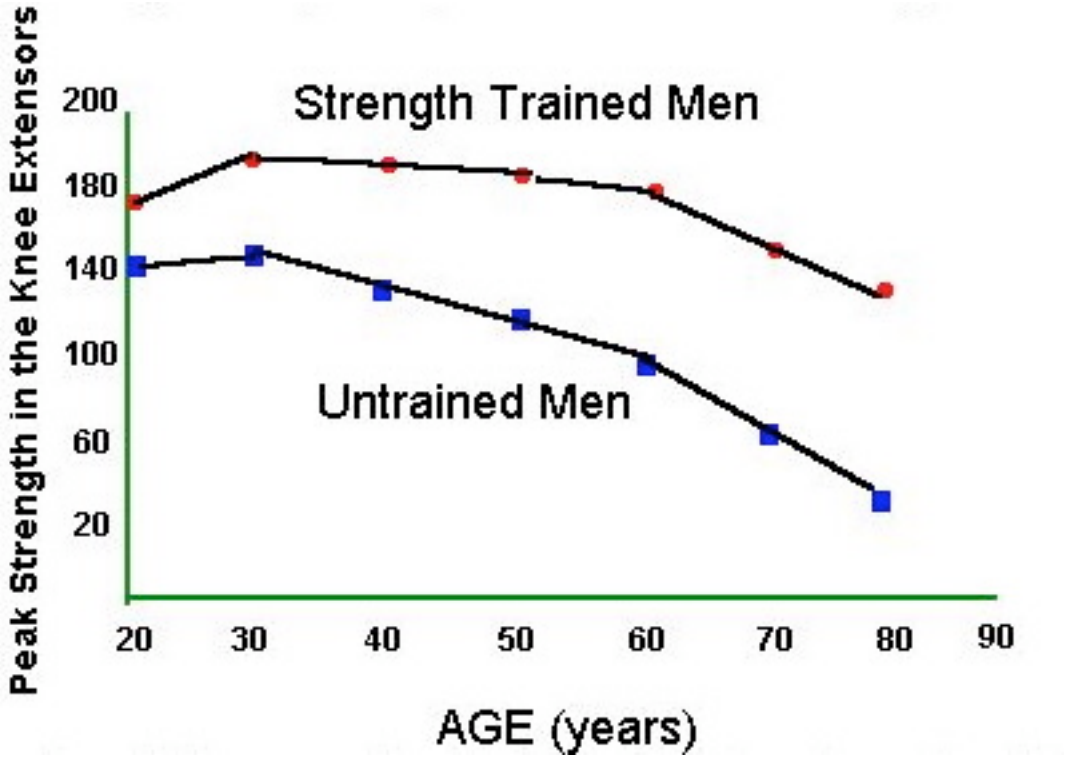
In my second example, I will use someone even closer to home, my wife. Hilde is a natural endurance athlete. She has competed in X-country skiing, cycling, and triathlon races. In general, the longer the distance, the stronger she becomes. However, when I met her, she complained of painful hips that made running more than 40 minutes difficult. She had undergone physical therapy and employed all manner of flexibility training to relieve the apparent immobility. After watching her run, I could see she was very weak in her hip extensors. Sadly, all her physical therapy had never included appropriate strength training!

I started her with lunges, then eventually progressed to 1 legged squats from atop a 3 foot bench. Initially, she did not have sufficient strength to even approach a true parallel, 1 legged squat. But she grimaced and persisted. Now, 5 months later, her extensor strength is much improved. The payoff has been obvious in her hill climbing. She runs from her hips, instead of from her knees. She has also reported improved strength on the skies. In Hilde's case, a specific strength training program was added to an endurance regime, and the results were very positive. Her total strength training investment (including upper body and back extension exercises) is two, 30 minute sessions per week.

## *c) Aging Effects on Skeletal Muscle*

### Changes in Muscular Strength

It is well documented that a person's maximal strength decreases with increasing age. Is this due to an unavoidable effect of aging or the typical decrease in physical activity that often accompanies getting older? The answer appears to be BOTH.




From the figure above, it is apparent that strength training remains highly effective in maintaining muscular strength throughout life. However, after about age 60, strength levels fall more rapidly, independent of training. This is probably influenced by marked changes in the hormonal mileau. Both testosterone and growth hormone appear to decline more dramatically after about age 60. Reduction in the circulating concentration of these hormones will result in a shift in the balance between muscle protein synthesis (anabolism) and protein breakdown (catabolism). The decreased strength is due to atrophy of muscle fibers. It is important to notice that with strength training, the maximal strength of a 60 year old can exceed that of his untrained sons! And, several studies have demonstrated that strength gains are possible even at 90 years old. So it is never too late to begin a strength training program!

## Fiber Type and Aging

There have been conflicting reports and myths developed regarding fiber type changes with aging. Cross-sectional studies of post-mortem bodies between age 15 and 83 have suggested that fiber type composition is unchanged throughout life. This is also supported by comparing muscle biopsy results of younger and older endurance athletes. In contrast, one longitudinal study of a group of runners examined in 1974 and again 1992, suggested that training could play a role in fiber distribution. Those athletes who continued training showed unchanged fiber composition. Those who stopped training appeared to have greater slow-twitch fiber percentage. This was primarily due to selective atrophy of the fast fibers. This is not difficult to explain since they are seldom recruited. There is also some evidence that the actual number of fast motor units decreases slightly with aging after age 50, about 10% per decade. The reasons or mechanisms for such a change are unclear. So, the net effect of aging for the endurance athlete is unchanged fiber composition or a slight *relative* increase in Slow fiber type due to selective Fast fiber loss. The Fast motor units **do not** become Slow motor Units.

## Muscle Endurance Capacity and Aging

The good news for the endurance athlete is that there appears to be little change in skeletal muscle oxidative capacity with age, as long as training is maintained. The number of capillaries per unit area of muscle is the same in young and old endurance athletes. Oxidative enzyme levels are similar or slightly lower in older athletes. This small decrease is probably attributable to decreased training volume in the older athletes. Furthermore, it appears that the older individual who starts endurance training retains the potential to improve muscle enduranace capacity.

## Summary

It appears that the Masters athlete who continues endurance training at high intensities and mantains a maintenance strength training program experiences few changes in skeletal muscle through age 50. After age 50, declines in the quantity, but not the quality of muscle occur. These declines are also deminished by continued training. In general the changes that occur diminish maximal strength and power more than endurance capacity. This helps to explain the tendency for older athletes to move toward longer events within their sports discipline.

## *d) Skeletal Muscle Fiber Type*

Have you ever sat down for Thanksgiving dinner and found yourself wondering why turkeys have some dark meat and some white meat? Well, you were not the first. A scientist named Ranvier reported differences in muscle color within and among animal species back in 1873. The explanation for the color differences is pretty simple and has a basis in physiology. The dark meat of the turkey, or chicken, is "red" or slow-twitch muscle. The white meat is "white" or fast-twitch muscle. Most animals have some combination of these two fiber types, though the destinctions may be less obvious. Why are they differently colored? The slow muscles have more mitochondria (full of red pigmented cyctochrome complexes), and more myoglobin packed within the muscle cells. This gives them a darker, reddish color. Humans also have dark and white meat. Some of our muscles, like the soleus in the lower leg are almost all slow twitch fibers. Others such as those controlling eye movements are made up of only fast twitch fibers. Function dictates form in these highly specialized muscles. The majority of human muscles contain a mixture of both slow and fast fiber types. From an evolutionary standpoint this makes sense. Our not so very distant ancestors' daily survival sometimes dictated a long walk or jog in search of food. Other times, a fast sprint or jump may have kept one out of harm's way. The exact composition of each muscle is genetically determined. On average, we have about 50% slow and 50% fast fibers in most locomotory muscles, with substantial intra-individual (and muscle to muscle) variations. This variation helps make sports interesting!

### Olympic Champions are Oddballs

If you want to win an Olympic medal in the 100 meter dash, you had better be born with about 80% fast twitch fibers! Want to win the Olympic marathon? Put in an order for 80% slow twitch fibers in your quads. The fast twitch fibers benefit the absolute sprinter because they reach peak tension much faster than their slow twitch counterparts. Gram for gram, the two types are **not different in the amount of force they produce**, **only their rate of force production.** So, having a lot of fast twitch fibers only makes a positive difference when the time available for force production is very limited (milliseconds), like the 100ms or so the foot is in contact with the ground during a sprint or long jump. It makes no difference to the powerlifter who may use 3-4 seconds to execute a slow, smooth lift. In cycling, the only event that they are decidedly advantageous for is the match sprint, analogous to the track 100 meter dash, but with more anticipatory tactics and theatrics.

For the pure endurance athlete, more slow twitch fibers are advantageous. These fibers give up lightning contraction and relaxation velocity for fatigue resistance. Lots of mitochondria and more capillaries surrounding each fiber make them more adept at using oxygen to generate ATP without lactate accumulation and fuel repeated contractions, like the 240 or so in a 2000 meter rowing race, or the 15,000 plus in a marathon.

**Does Fiber Type Change with Training?**

This has been one of the 10,000 dollar questions in exercise physiology. It has been documented that elite endurance athletes possess a higher percentage of slow twitch fibers in the muscles they use in their sport, compared to untrained individuals. Is this due to genetic endowment or years of rigorous training? The answer is difficult to get at directly because we don't have comparative muscle biopsies ***(see subcategory-(i) below)*** of great athletes before and after they started training and excelling in their sport. However, good basic investigation using experimental models has helped generate some answers. The critical knowledge to remember is that fiber type is controlled by the motor nerve that innervates a fiber. Unless you change the nerve, you won't change fiber types from fast to slow or vice versa. Just this type of experiment has been performed in animals (generally rats). **So, remember, there is no compelling evidence to show that human skeletal muscle switches fiber types from "fast" to "slow" due to training.**

**Then Why Am I Training So Hard?**

Two reasons; first, skeletal muscles respond to chronic overload (training), by trying to minimize the cellular disturbance caused by the training. With intense endurance training, fast fiber types can develop more mitochondria and surrounding capillaries. So can the slow fibers. So training improves your existing fiber distribution's ability to cope with the exercise stress you create for it.

Second, even among a group of elite endurance athletes, fiber type alone is a poor predictor of performance. This is especially true in the intermediate duration events. There are many other factors that go into determining success! In fact, there is also evidence to suggest that a mixed fiber composition is ideal for success in an event like the mile run, or if good performances are to be possible in a range of events. More about Fiber type and training. **(*see subcategory-(ii) below*)**

## *i) The Muscle Biopsy*

In humans, samples of muscle are extracted using a biopsy needle or bioptome. This is 3-piece instrument about the diameter and length of a pencil. The outside is a hollow, stainless steel cylinder with a point on the end and a small oval "window" cut out about 1 inch above the tip. Inside the cylinder is a circular blade attached to a long plunger or handle. Finally, inside that is a plunger to push the extracted piece of muscle out of the biopsy needle. They look like this:


An athlete is given a local anaesthetic like xylocaine, by injection, above the site to be biopsied. A small ~1cm long incision is then made through the numbed skin, fat and fascia with a regular surgical scalpel. The biopsy needle is then introduced into the muscle, about 3 inches deep. The athlete has been riding on a cycle ergometer and at some point is asked to lay back as quickly as possible for a biopsy in order to capture the cellular conditions in the muscle corresponding to the exercise situation as closely as possible. Since the preliminary incisions have already been made, if all goes well, the muscle sample will be extracted and frozen within 15 seconds of exercise cessation.

Since there are no sensory nerves within the muscle, this will only be detected as pressure. Well, that is true most of the time. I had 8 biopsies during my student days. On a couple of those I got a jolt when one or another nerve got a good tickle, but it was not really pain I felt, at least not the kind

you would feel if something cut through your skin. When the needle is in the leg, a small portion of muscle will push inside the window of the barrel. Then the blade is passed through the barrel, cutting off the small piece (about the size of a green pea, or smaller), which remains inside the barrel as it is extracted from the muscle. This small piece is quickly frozen in liquid nitrogen or pentane for later analysis.

In sports science, the principle value of the muscle biopsy has been to generate histological cross-sections for fiber type staining, and for the determination of muscle glycogen concentration. Because of the heterogeneity of fiber distribution within most muscles, fiber type determination from a single biopsy should be considered only a reasonable estimate.

I sometimes receive emails from athletes enquiring where they might have biopsies performed to determine their fiber type percentage. My answer is that this is not a normal type of athlete testing offered by performance testing facilities, or even elite training centers. There really is no value to the athlete in knowing his/her fiber type distribution, unless it is to use as an excuse for performances that do not meet expectations. You aren't going to learn anything that will guide you to train differently for your chosen sport. Muscle biopsies, when performed on healthy athletes, are almost always done in the interest of some physiological research project.

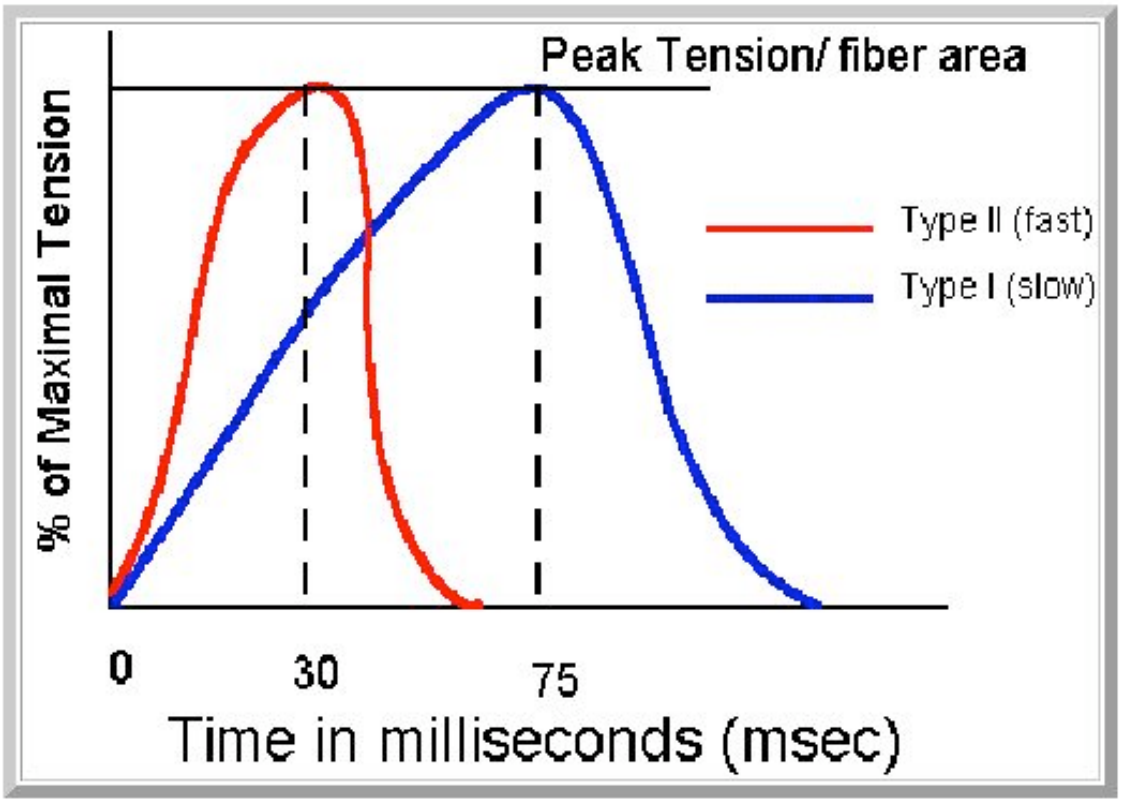
## *ii) Skeletal Muscle Fiber Type - Part Two*

Like most things, there is the simple story, and the real story. Physiological investigations in the late 60s and early 70s have done a great deal to shape our knowledge of skeletal muscle function and fiber type. The biopsy technique, enzyme histochemistry, and physiological studies all advanced this issue. From this work, we now know the fiber types differ: 1) in contractile speed, 2) in myosin ATPase enzyme characteristics, and 3) in metabolic enzyme profile. From these three differences, three different fiber type classification schemes have emerged.

Dr. Phil Gollnick and colleagues studied differences in contractile speed in different muscles. They found that the fiber types were distinguishable based on the time it took them to reach peak tension when stimulated. That difference is graphically demonstrated here.

**Skeletal Muscle Fiber Type and Contractile Velocity**

When a muscle contraction occurs, it is initiated by an electrical impulse originating in the brain. All of the fibers in one motor unit are activated by a single motor nerve. The two fiber types differ in the time it takes for the fiber to fully contract, or reach peak contractile tension. This difference is depicted graphically below. Notice that there is a big difference in the rate of both force development, and relaxation. The peak force attained is not different when measured per unit cross sectional area of the fiber. This is one of the critical concepts to be grasped in order to understand the impact of fiber type on different sports activities.




They proposed the distinction *slow*, and *fast*. This turned out to be an oversimplification. Meanwhile, even before this study, Brooke and Engle distinguished the fiber types based on differences in Myosin ATPase enzyme activity. They arbitrarily divided the muscles into two groups and called them *Type I* and *Type II.*

Around the same time Gollnick and colleagues were classifying muscles based on contractile speed, Dr. J.B. Peter and his group investigated the properties of the two categories of fibers established by Brooke and Engle. They proposed another set of terminology created by combining tension generating and metabolic properties. Type I cells were termed *Slow*

*Oxidative*. That was simple. The slow fibers had a lot of mitochondria (containing oxidative enzymes) and capillaries. However the *Type II* or *Fast fibers* had to be further divided into two sub-categories. Type II cells were either *Fast Glycolytic (FG)* or *Fast Oxidative Glycolytic (FOG).* The FG fibers stored lots of glycogen and had high levels of enzymes necessary for producing energy without oxygen, but contained few mitochondria. The FOG fibers had the best of both worlds, high speed and glycolytic capacity, plus high levels of oxidative enzymes. These INTERMEDIATE fibers were termed type *II*A fibers by a fourth research group (Brooke & Kaiser, 1970). The pure fast fibers (FG) were termed Type *IIb*. This last lingo system seems to have stuck within the physiological research community.

For the athletic community, the important information is this. It does appear that pure fast (Type IIb) fibers can transition to "hybrid" (Type IIa) fibers with chronic endurance training. Biopsies of elite endurance athletes reveal that after years of training, they have almost no IIb fibers, but often have a significant percentage of the intermediate, IIa fibers. BUT, the majority of the available research suggests that Type IIa fibers do not transition to Type I. This is the more accurate way of saying what I said at the end of Part I of the Fiber type discussion.

| | **TYPE of FIBER** | | |
|---|---|---|---|
| **Characteristic** | **Slow Oxidative (I)** | **Fast Oxidative ( IIa)** | **FastGlycolytic (IIb)** |
| Myosin ATPase activity | LOW | HIGH | HIGH |
| Speed of Contraction | SLOW | FAST | FAST |
| Fatigue Resistance | HIGH | Intermediate | LOW |
| Oxidative Capacity | HIGH | HIGH | LOW |
| Anaerobic Enzyme Content | LOW | Intermediate | HIGH |
| Mitochondria | MANY | MANY | FEW |
| Capillaries | MANY | MANY | FEW |
| Myoglobin Content | HIGH | HIGH | LOW |
| Color of Fiber | RED | RED | WHITE |
| Glycogen Content | LOW | Intermediate | HIGH |
| Myoglobin Content | HIGH | HIGH | LOW |
| Fiber Diameter | SMALL | Intermediate | LARGE |