

Training Fast Twitch Muscle Fibers: Why and How

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Abstract. With the finding that short, intense sprints can improve aerobic capacity (Tabata, *et al.*, 1996), there has been a huge increase in the number of experts who advocate this kind of training over traditional endurance training. Several successful swim coaches are among those recommending more high-intensity training while an equal or even greater number are warning of the pitfalls of training in this manner. A theory is presented in this paper that high-intensity training is essential for improving aerobic endurance. An argument is also presented for a balanced approach to training that includes adequate quantities of moderate-, and low-intensity swimming.

Introduction:

In 1996 Dr. Izumi Tabata and associates published the results of a study that challenged traditional assumptions about endurance training. These researchers reported that training with a series of short sprints at very fast speeds was just as effective for improving VO_{2max} as traditional endurance training at moderate speeds. High-intensity training, as it was termed, also produced an additional benefit. The group that trained with sprints improved their anaerobic capacity by 28% while the traditional endurance-training group did not improve on this measure. The training protocols and results of the study are summarized in Table 1.

Table 1 High intensity versus traditional endurance training.

Training groups	Training program	VO_{2max}	Anaerobic capacity
Traditional training group	Cycling at 70% VO_{2max} for 60 mins./daily, 5 days/wk. for 6 weeks	Inc. 10% (53 to 58 ml/kg/min.)	No change
High-intensity training group	8 x 20 sec sprints with 10 secs. rest between at 170% VO_{2max} , 5 days/wk. for 6 weeks.	Inc. 14% (48 to 55 ml/kg/min.)	Increased 28%

Data from "Effect of moderate-intensity endurance and high-intensity intermittent training on anaerobic capacity and VO_{2max} .", by I. Tabata, K. Nishimura, M. Kouzaki, Y. Hirai, F. Ogita, M. Miyachi, and K. Yamamoto, (1996), *Medicine & Science in Sports & Exercise*, 28(10): 1327-1330.

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Dr. Tabata's results were no "fluke". They have been replicated in several additional studies. In one of these, previously well-trained elite cyclists improved their 40 km time trial performances by 3% after only four weeks by replacing 15% of their aerobic-base work with six high-intensity training sessions (Lindsay, *et al.* 1996). On the high-intensity days the cyclists performed several five-minute efforts at 86% of VO_{2max} , with one-minute rest periods between each repeat. In another study, participants increased VO_{2max} by nearly 9% following seven weeks of sprint training. The subjects trained three times per week, doing four to ten, 30-second sprints each session, with 4 minutes rest between each sprint (MacDougall *et al.* 1998).

How is it that training which has traditionally been thought of as anaerobic can produce improvements of aerobic capacity that equal and in some cases surpass those of traditional methods of endurance training for both untrained and previously well-trained subjects? I believe the answer to this question has to do with the way that fast twitch muscle fibers are recruited during exercise.

Purpose

The major purpose of this paper will be to describe a theory that explains why high-intensity training can improve aerobic capacity. Additional purposes will be to outline some types of repeat sets that are effective for training fast twitch muscle fibers and to describe some questions that need to be answered about their training. The paper will be presented in two parts. In Part I the characteristics of the various muscle fiber types and how they are recruited during training will be discussed. Part II will focus on the how information in Part I can be used to training swimmers. Let's begin with a description of the muscle fibers types and their characteristics.

Characteristics of muscle fiber types in humans

Over the last several decades it has become common knowledge that the muscles of humans (and animals) contain two distinct categories of fibers, slow twitch and fast twitch. Slow twitch muscle fibers are also commonly labeled Type I, or red fibers, while fast twitch fibers have also been identified as Type II, or white fibers. Slow twitch fibers are genetically very well suited for aerobic metabolism and, therefore, endurance work. At the same time, they have a limited capacity for anaerobic metabolism. Fast twitch muscle fibers are very well suited for anaerobic metabolism but much less so for aerobic metabolism. They contract rapidly and powerfully but also fatigue more quickly than their slow twitch counterparts. Consequently, they are genetically best suited for sprint and power work.

Slow twitch muscle fibers got their name because their time to peak contraction is approximately 110 milliseconds. While contraction velocities in this range cannot really be considered slow, they are relatively slower than those of fast twitch muscle fibers which are in the range of 40 to 60 milliseconds (Brooks, Fahey, and Baldwin, 2005).

Slow twitch fibers have a generous supply of myoglobin, a substance that enhances the oxygen supply of those fibers. Myoglobin has a reddish pigment that gives slow twitch fibers their dark red appearance. A function of myoglobin is to transport oxygen to the mitochondria of muscle cells where it can be oxidized, releasing energy for contraction in the process. Figure 1a is a drawing of a single muscle fiber showing the location of the various components that will be discussed in the following paragraphs. An electron micrograph of a muscle fiber enlarged 250,000 times is displayed in figure 1b. It shows the orderly arrangement of mitochondria, (multiple jagged black lines) within that fiber.

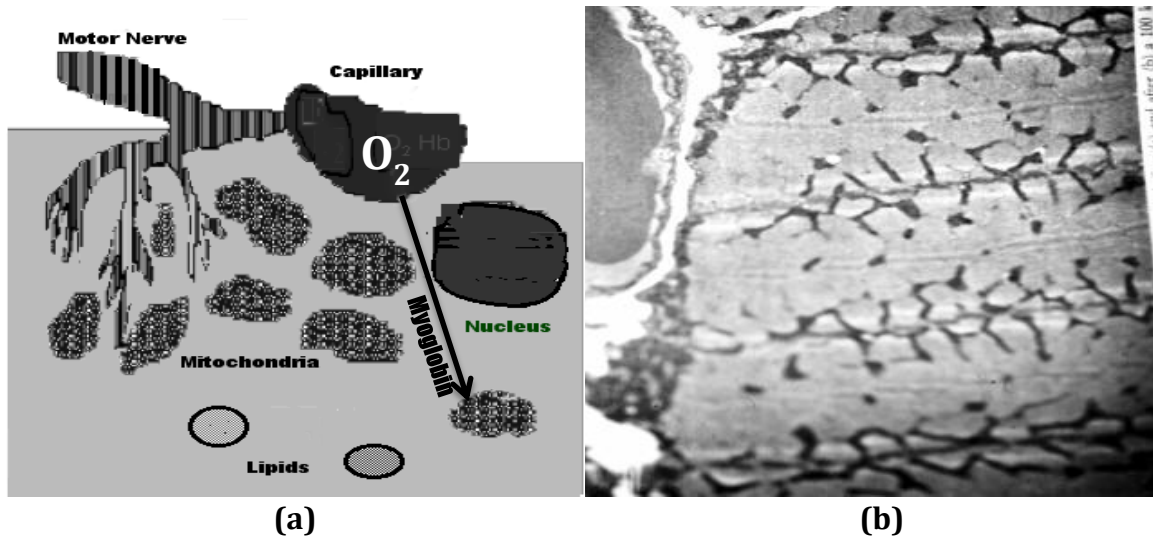


Figure 1 Figure 1a is a drawing of a single muscle fiber. It shows the motor end plate where the contractile impulse arrives and a capillary where oxygen diffuses into the fiber. Also shown are mitochondria. They are the structures where aerobic metabolism takes place. The role of myoglobin in transporting oxygen to the mitochondria is also represented. Figure 1b is an electron micrograph of a muscle fiber showing the orderly arrangement of mitochondria (the black, jagged objects).

As indicated, mitochondria, the so-called “power plants” of muscle cells, are the structures wherein aerobic metabolism takes place. Mitochondria contain large supplies of aerobic enzymes, such as citrate synthase (CS) and succinate dehydrogenase (SDH), that catalyze the aerobic breakdown of pyruvate and hydrogen ions to carbon dioxide and water. Slow twitch muscle fibers naturally contain more mitochondria than fast twitch fibers.

Another factor that enhances the endurance of slow twitch muscle fibers is the large number of capillaries surrounding them. This allows more oxygen to be delivered to them via the circulatory system.

Fast twitch muscle fibers tend to be naturally larger than slow twitch, and this, together with their faster speed of shortening, makes them capable of generating more power with their contractions. They also have greater supplies of myosin ATPase, an enzyme that catalyzes the rapid release of energy from ATP and they contain more creatine phosphate, the substance that replaces ATP more rapidly

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than any other chemical in the body (Bogdanis, 2009). In addition, they contain larger quantities of anaerobic enzymes, such as phosphofructokinase (PFK) and the muscle form of lactate dehydrogenase (M-LDH) that enable them to deliver energy faster via anaerobic glycolysis, (the breakdown of muscle glycogen to lactic acid) (Pette, 1985). On the negative side, these fibers have less myoglobin and fewer mitochondria, which reduces their capacity for aerobic metabolism. Untrained fast twitch muscle fibers usually have a smaller number of capillaries surrounding them than do slow twitch fibers which compromises their ability to absorb oxygen. Consequently, they fatigue more quickly.

In humans, fast twitch muscle fibers have been further classified into two sub-categories, FTa, and FTx fibers. The latter were formerly designated FTb fibers. However, that changed with the advent of a new system of fiber typing that relies on identifying the number of myosin heavy chain filaments they contain. Heavy chain myosin can combine with actin more strongly and at a faster cycling rate during contractions because it contains more of the enzyme myosin ATPase. (Brooks, Fahey, and Baldwin, 2005). As a result, fibers with heavy chain myosin will contract faster. Fast twitch muscle fibers contain heavy chain myosin while the myosin in slow twitch fibers is of a lighter chain variety.

Fast twitch A fibers, (also known as FT red, and Type II red fibers) have greater quantities of myoglobin and mitochondria than FTx fibers, but, as mentioned earlier, less of these substances than naturally occurs in ST fibers. This gives the FTa fibers a somewhat more pinkish appearance than FTx fibers and makes them capable of greater aerobic activity and, therefore, greater endurance than FTx fibers.

FTa fibers have greater anaerobic capacity than slow twitch fibers. To avoid confusion in terms, let me explain that I refer to anaerobic capacity as the ability to metabolize muscle glycogen to lactic acid without the use of oxygen. It is common for some experts to also include the processes of lactate removal and muscle buffering under this heading. The first of these training effects allows more of the lactic acid and other metabolites that are produced during exercise to be removed from muscles while they are working (Brooks, Fahey, and Baldwin, 2005). The second effect occurs when muscle fibers improve their ability to weaken (buffer) lactic acid. This slows the decline of muscle pH from its normally alkaline level of 7.04, allowing them to release more energy anaerobically before acidosis (low muscle pH) occurs. Both of these training effects are considered anaerobic because they do not require oxygen. However, they are additional mechanisms for delaying muscular fatigue and are not considered in my definition of anaerobic capacity.

When the effect of these two processes, buffering and lactate removal, work together to delay muscular fatigue, it is often referred to as anaerobic endurance or speed-endurance. I prefer the former term. It should be noted that FTa fibers have faster rates of lactate removal and greater buffering capacity and, therefore, greater anaerobic endurance than either FTx or ST fibers (Noakes, 2001).

There is still much to learn about FTx muscle fibers.. We do know that they are the largest and the fastest contracting muscle fibers of all. Consequently, they are capable of producing the greatest amount of power. The power that fibers can generate is determined by a combination of their contractile force and contractile velocity. As mentioned previously, contractile force is largely a consequence of fiber size. Fast twitch fibers are larger and faster than slow twitch with FTx being the largest and fastest of all. Accordingly, FTx fibers, are capable of generating greater power. FTa fibers can generate up to 5 times the power of ST fibers while FTx fibers are nearly twice as powerful as FTa fibers (Widrick, *et al*, 1996b). The graph in figure 2 illustrates differences in contractile power among the three fiber types at various percentages of maximum load.

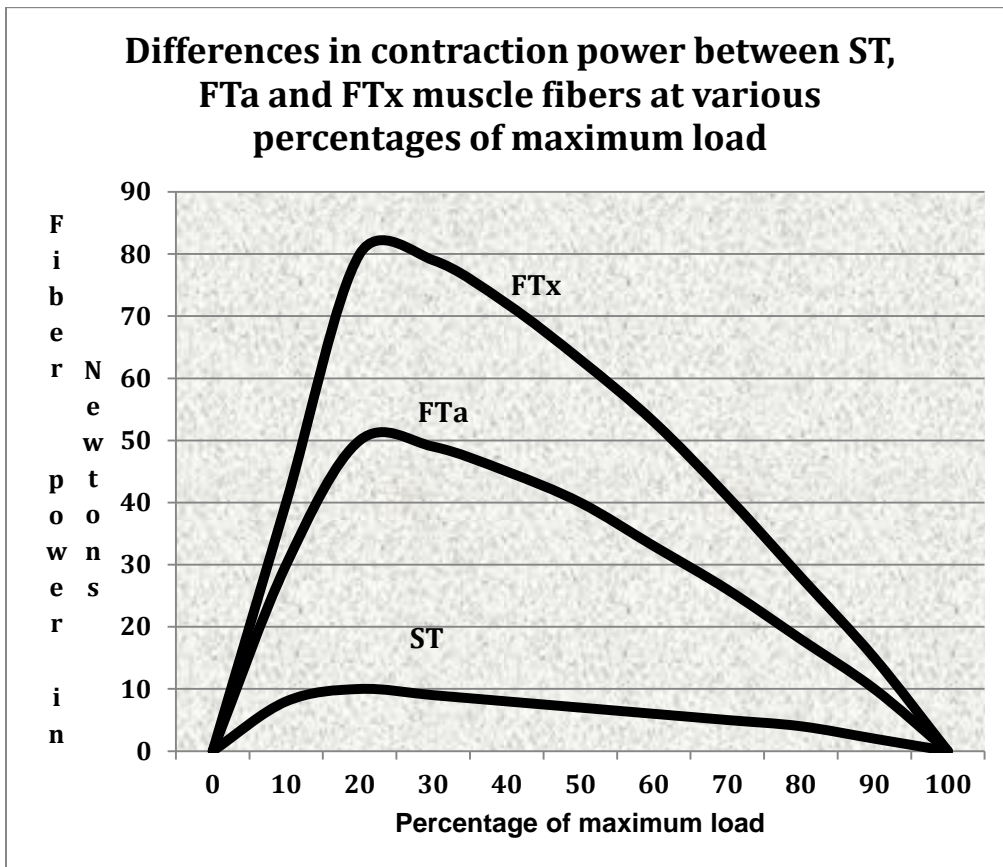


Figure 2 Notice that all fibers achieve their greatest power production when the load is approximately 20% of the maximum capable by the fiber. This is because, the fiber is still able to contract rapidly at this load, whereas, contractile velocity is slowed considerably at higher loads. Modified with permission from, "Muscle mechanics: Adaptations with exercise training," by R.H. Fitts and J.J. Widrick. (1996), in J.O. Holloszy (Ed.), *Exercise and Sports Sciences Reviews* (pp. 427-443). Baltimore MD: Williams and Wilkins,

FTx fibers have the lowest aerobic capacity of the three muscle fiber types. They have less myoglobin and mitochondria and, therefore, a decidedly less pink appearance than FTa fibers. On the other hand, they, like FTa fibers, are rich in anaerobic enzymes and capable of rapid energy release via anaerobic glycolysis.

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FTx muscle fibers have less buffering and lactate removal capabilities than their FTa counterparts and this contributes to their rapid fatigability. The different characteristics of ST, FTa and FTx muscle fibers are summarized in Table 2.

Table 2 Characteristics of ST, FTa and FTx muscle fibers.

Characteristics	Muscle Fiber Types		
	Slow Twitch	Fast Twitch a	Fast Twitch x
Aerobic (oxidative) Capacity	High	Moderate	Low
Anaerobic (glycolytic) capacity	Low	High	Highest
Myosin ATPase	Low	High	Highest
Creatine Phosphate	Low	High	Highest
Buffering capacity	Low	Highest	High
Lactate removal rate	Low	Highest	High
Capillaries per fiber	High	Moderate	Low
Contractile velocity	Slow	Fast	Fastest
Fibers per motor unit	<300	>300	>300
Contractile force	Low	High	Highest
Power	Low	High	Highest

Most humans are born with approximately equal proportions of fast twitch and slow twitch muscle fibers. The FTa fibers tend to predominate within the fast twitch category, and the percentage of FTx fibers is generally lower.

While the middle 68% of the population have nearly equal percentages of fast twitch and slow twitch fibers, there are persons at either end of the bell-shaped curve who are born with either a much higher percentage of slow twitch fibers or a much higher percentage of fast twitch fibers than the general population. These people tend to excel in endurance or sprint/power events respectively.

How are slow and fast twitch muscle fibers recruited during work?

A common misconception is that slow work is performed by slow twitch muscle fibers and fast efforts are executed by fast twitch fibers. Actually, neither of these statements is entirely true. Sub-maximal work is performed by the more aerobically

efficient slow twitch muscle fibers while progressively more and more fast twitch fibers are recruited to assist them as the effort increases toward maximum.

The orderly arrangement of muscle fibers into contractile units is depicted in figure 3. Large muscles like the deltoid, are made up of groups of muscle fibers served by a single motor nerve. These groups of fibers are termed motor units. Each motor unit contains fibers of a similar type. Thus, even though a large muscle may contain all three fiber types, the fibers within a particular motor unit will be of the same type, either ST, FTa or FTx.

Motor units obey the “all or none” law. That is, if the nervous stimulation is sufficient to cause the fibers within a motor unit to contract, all of the fibers in the unit will contract with maximum force. Thus, the muscular force that can be applied by an athlete is largely due to the maximum number of motor units contracting at any one time and the types of motor units that are contracting. The motor units of slow twitch fibers usually contain fewer than 300 fibers, whereas, the motor units of fast twitch fibers have anywhere from several hundred to thousands of fibers. Since all of the fibers in a motor unit contract at once, and fast twitch motor units contain, not only larger fibers, but also more fibers, it is understandable that fast twitch motor units will generate considerably more force when they contract.

What has become known as the “size principle” of muscle fiber recruitment is also illustrated in figure 3. The order of recruitment is from ST to FTa to FTx motor units as the intensity of work increases. This is because of the size of the motor nerve innervating the different categories of motor units. Smaller motor nerves require the least amount of nervous stimulation to excite their motor units to contract. Slow twitch muscle fibers have the smallest motor nerves so they will be recruited to perform work that is easy to moderate in nature. Motor units with FTa fibers have larger motor nerves and require a greater neural drive before they will be excited to contract, therefore, they will not be recruited until the work intensity is beyond moderate. The motor nerves of FTx fibers are the largest of all so they will not be recruited until the need for force and power approaches maximum. The illustration in figure 3 also portrays differences in contractile velocity, contractile force, and fatigability between the three fiber types.

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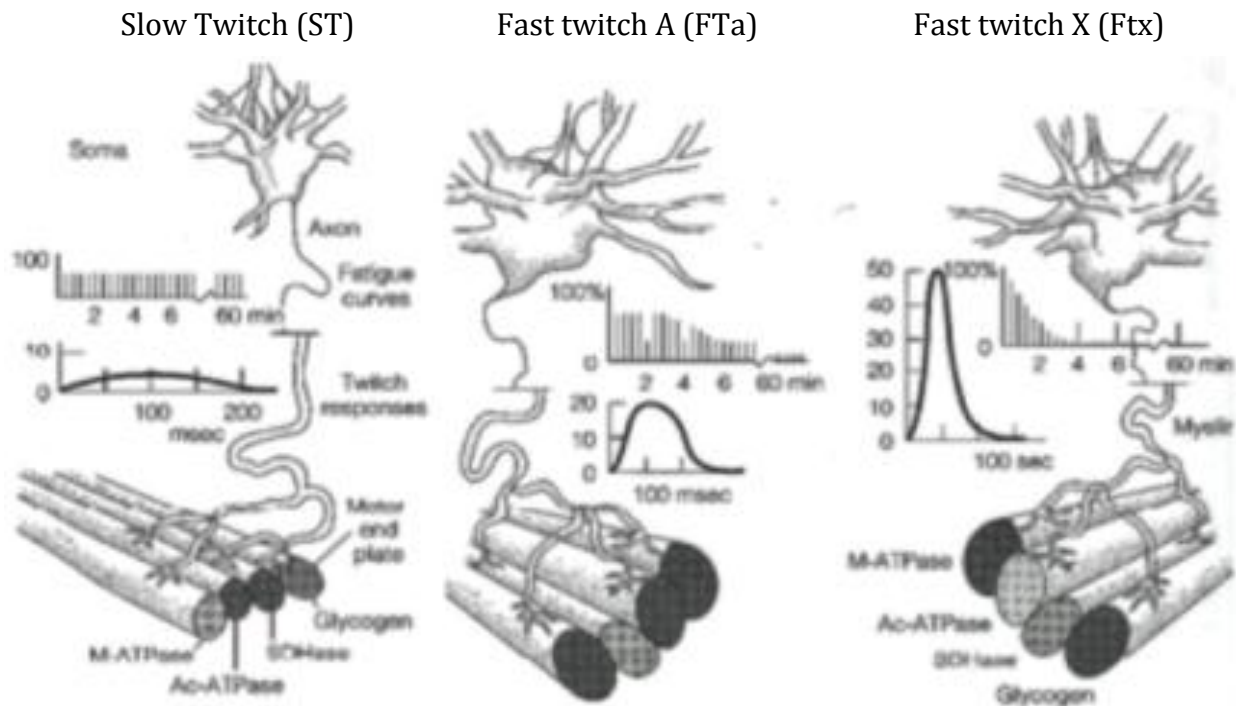


Figure 3 This figure illustrates several characteristics of ST, FTa and FTx motor units. Notice that the motor nerve that serves the ST fibers is smaller than the other two. It has a lower threshold for excitation so that slow twitch fibers can be recruited at low levels of effort. The motor nerve serving FTa fibers is larger and requires a greater amount of excitation before the electrical messages it sends to its fibers causes them to contract. Thus, FTa fibers will not be recruited until the level of effort is moderate to high. The motor nerve that innervates FTx motor units is the largest of all so it will require the most stimulation for its fibers to contract. The set of graphs just below each motor nerve indicate the fatigability of the fibers in that motor unit. You will notice that the FT motor units fatigue more rapidly than the ST, and that, within the fast twitch group, FTx motor units fatigue more quickly than FTa motor units. The next set of graphs displays contractile speed and force. ST motor units contract slowest and with the least force. FTa contract considerably faster and with greater force. FTx motor units are the largest and fastest contracting, so they generate the greatest amount of force.

Modified from *Biology of Physical Activity* (p. 53), by D.W. Edington, and V.R. Edgerton. (1976), Boston, MA: Houghton-Mifflin, with permission.

To be precise, the order of muscle fiber recruitment results from the force or power required to perform a movement, and not the speed needed to perform it. For example, slow twitch muscle fibers will be recruited to perform a fast movement that requires little force, like spinning on a bicycle ergometer, while lifting a heavy weight very slowly would require the recruitment ST, FTa and FTx fibers nearly simultaneously. It happens, however, that in most athletic activities, an increase in effort is also accompanied by an increase in speed, so, in a sport like swimming,, increases in effort are usually accompanied by increases of speed. Hence, the terms force, power, intensity and speed are often used interchangeably when discussing the order of muscle fiber recruitment.

The proposed pattern of recruitment for fast and slow twitch muscle fibers can be represented by a graph like the one in figure 4. This graph illustrates the so-called “ramp effect of muscular contraction” (Wilmore and Costill, 1999).

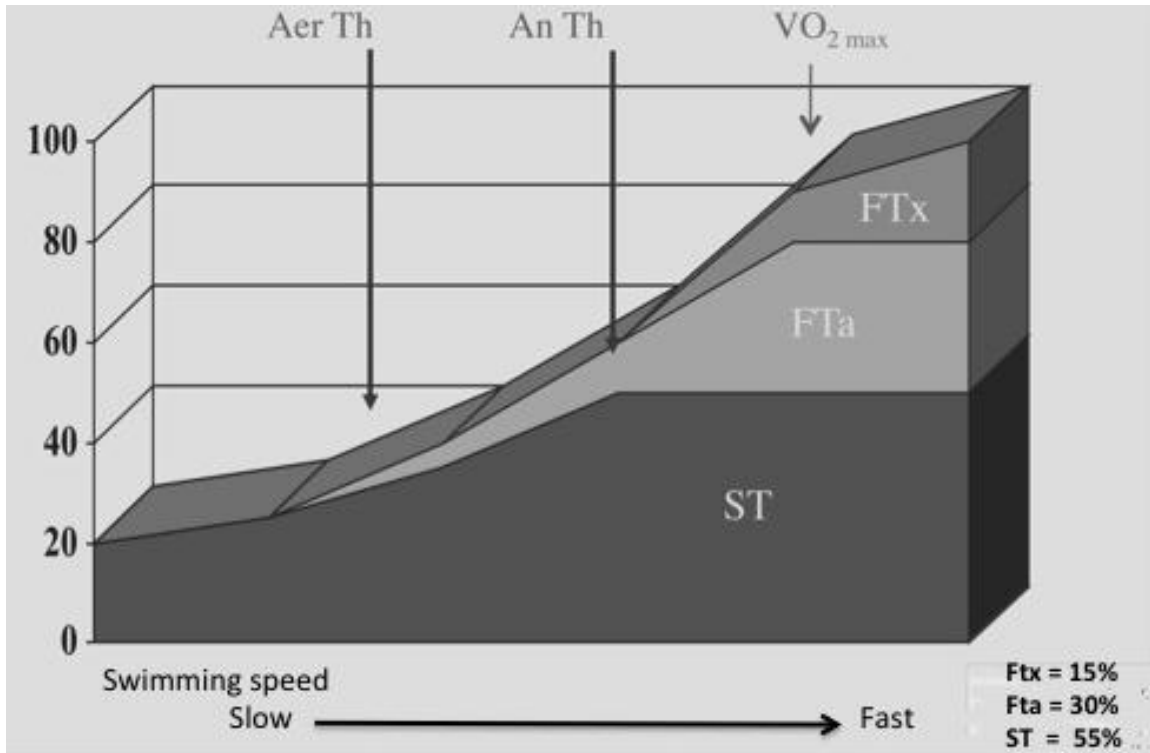


Figure 4. The ramp effect of muscle contraction. The graph shows that progressively greater and greater numbers of muscle fibers are recruited as athletes swim faster. Slow twitch are the first fibers to be recruited. FTa fibers are added to the effort as speed increases. FTx are the last fibers to be recruited and this occurs when speeds are near maximum. Chart modified with permission from *Physiology of Sport and Exercise*, (p. 50) by J.H. Wilmore, and D.L. Costill, (1999), Champaign, IL: Human Kinetics.

To summarize what was said before, at low levels of effort it is primarily the slow twitch muscle fibers that do the work. When the effort increases, fast twitch muscle fibers will be recruited to assist (not replace) their ST counterparts. FTa fibers are the first of the fast twitch group to be recruited as the effort increases, with FTx fibers recruited to assist both the ST and FTa fibers as the effort approaches maximum.

The threshold for significant FTa fiber recruitment is believed to approximate a workload that corresponds to the lactate threshold (Brooks, Fahey, and Baldwin, 2005). Since fast twitch fibers produce more lactate than slow twitch fibers during work, the exponential rise in blood lactate at workloads exceeding the lactate threshold probably occurs because significant numbers of FTa fibers are now contracting. Although no threshold for recruitment of FTx fibers has been posited, it is reasonable to assume, based on research with rats that will be presented later,

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that they will not contribute substantially to a particular effort until the intensity approaches or exceeds 100% of VO_{2max} .

Before leaving this section, I should mention that muscle fatigue causes a similar effect on muscle fiber recruitment to that of work intensity. As indicated earlier, ST fibers, and, perhaps, some low threshold FTa muscle fibers will be recruited first during long training sessions when most of the swimming is done slower than lactate threshold speed. However, after 1 to 2 hours many of these fibers will lose a large portion of their fuel supply and become fatigued. When this happens, the nervous system will recruit additional FTa fibers to maintain the desired swimming speed. Even later, when most of the ST and FTa fibers are nearly exhausted, FTx fibers will be recruited to preserve that speed (Wilmore, *et al.*, 2008). Unfortunately, the FTx fibers will fatigue quickly after recruitment and the swimmer will have to reduce his or her pace considerably soon after they join the effort.

The fact that FTa and FTx fibers are recruited when ST fibers lose glycogen could explain why long-slow distance training has been effective for improving the endurance of fast twitch muscle fibers. But, is this the most efficient and effective way for doing so. I doubt it. For one thing, muscle glycogen depletion will be severe after each session and trying to train from day to day with an inadequate supply of fuel may cause failing adaptation. For another, there is no need to train fast twitch fibers in this manner when there are potentially less harmful ways to do so. In part two of this paper I will discuss the effects of training on muscle fibers and how to train to achieve those effects.

The Effects of Training on Muscle Fibers:

It is well documented that training improves the aerobic capacity of FTa fibers so that they fatigue less rapidly. The mitochondria become larger and more numerous and the number of capillaries surrounding them increases (Holloszy, 1967). The aerobic capacity of trained FTa fibers often approaches that of untrained ST fibers (McArdle, Katch and Katch, 1996). Training will also elevate their buffering capacity and lactate removal rates, which will increase the time they can assist ST fibers in maintaining a particular pace.

There are strong indications in the literature that the aerobic capacity, buffering capacity and lactate removal rates of FTx muscle fibers can also be increased with training until they function much like FTa fibers. One such indication is that fibers previously typed as FTx become less numerous while those typed as FTa fibers become more numerous after training. The data in Table 3 shows the results of a study where a group of college students were subjected to eight weeks of resistance training (Andersen, *et al.*, 2005). Muscle biopsies and subsequent fiber typing showed a decrease in the number of FTx fibers and a concomitant increase in FTa fibers after training. The training program in this study consisted of 4 to 5 sets of leg presses, hack squats, knee extensions and leg curls, performed three days weekly, for three months. Subjects began training with 10-12 repetitions of each

exercise, increasing weight and decreasing repetitions through the course of training until they were performing 6 to 8 repetitions of each.

Table 3 The effects of training on muscle fibers.

Fiber type percentages			
Fiber types	Before training	After training	Difference
Slow twitch	60	60	0
Fast twitch a	34	39	+5
Fast twitch x	6	1	-5

Data from “Changes in the human muscle force-velocity relationship in response to resistance training and subsequent detraining,” L.L Andersen, *et al.* (2005), *Journal of Applied Physiology*, 99: 87-94.

These results seem counterintuitive in that one would expect training with a high power output would encourage a switch from FTa toward FTx fibers types instead of the other way around. This is not the case, however. A consistent finding is that FTx fibers respond by gaining aerobic and anaerobic endurance whether the training program is made up of endurance or sprint/power activities.

An unwelcome consequence is that the contraction speed and, therefore, the power generating capacity of FTx and FTa fibers decreases as they become trained even when that training consists of sprints and weight training (Andersen, Klitgaard, and Saltin, 1994; Widrick *et al.*, 2002).

Slow twitch muscle fibers are also amenable to training despite their already generous supplies of capillaries, mitochondria, and aerobic enzymes. The quantities and activities of each will be enhanced considerably by training, in addition to improving the ability of ST fibers to metabolize fat for energy (Philips, 2006).

Information in the literature is scarce concerning the effects of training on the mechanisms of anaerobic metabolism, buffer capacity and lactate removal in ST fibers. I would assume each of these would also be improved with high intensity training. However, considering the affinity of these fibers for aerobic metabolism, I suspect the degree of improvement would be slight. One significant finding has been that any type of exercise, whether it is endurance or sprint in nature, will increase the contractile velocity of slow twitch muscle fibers and thus, their ability to generate power (Widrick, *et al.*, 1996a). Resistance training will also increase their size, further increasing their ability to generate force and, thus, power (MacDougall, 1986; Roman, *et al.*, 1993).

One recently discovered response to training the slow twitch muscle fibers is an increase in the lactate transporter MCT1 (Bonon, 2006). MCT1 is one of several types of monocarboxylate transporter proteins. Its purpose is to transport lactate from the blood and adjacent fast twitch muscle fibers into slow twitch fibers where it can be carried to their mitochondria and metabolized. MCT1 is found in greatest

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quantity in ST muscle fibers and in smaller quantities in FTa fibers. Very little, if any of this particular lactate transporter is found in FTx fibers. An increase should delay fatigue by increasing the ability of slow twitch muscle fibers to absorb lactate. The chain reaction effect would be that acidosis, and thus, fatigue, would be delayed in FT fibers if, during training and competition, more of their lactate was transported into slow twitch fibers where it could be stored and oxidized. (Lactate is transported out of fast twitch muscle fibers by another lactate transporter, designated MCT4. This transporter is also increased with training)

The results of some studies have shown that fast twitch fibers can gain enough myoglobin and mitochondria to be typed as slow twitch muscle fibers after training and vice versa. In one of these, the percentage of ST fibers decreased from 52% to 41% after three months of strength and interval training (Andersen, Klitgaard, and Saltin, 1994). At the same time, the percentage of FTa fibers increased from 35% to 52% and the percentage of FTx fibers decreased from 13% to 5%. This leaves open the possibility that training could cause a conversion of slow to fast twitch muscle fibers or the other way around. In fact, some experts have postulated the existence of an FTc fiber that may, through training, be converted to either a FTa or ST fiber (Wilmore, Costill, and Kenney, 2008). Brooks and his associates (2005) have also indicated the presence of hybrid muscle fibers that contains elements of both ST and FTa fibers. It is conceivable, but not proven, that hybrids of this type could be converted in either direction with proper training.

Despite these findings, the prevailing opinion is that FT fibers cannot be converted to ST and vice versa. At the present time, most experts believe that conversions of the two fiber types can only be produced by surgically changing the motor nerve innervating them. In other words, the motor nerve that serves a slow twitch motor unit would have to be surgically connected to a fast twitch motor unit before a conversion of FT to ST fibers could take place. The reason for reports of training-induced changes of slow to fast twitch fibers and vice versa in the literature may be due to difficulties in fiber typing. It is a complex and painstaking procedure fraught with possibilities for error. Nevertheless, the controversy concerning whether slow and fast twitch muscle fibers can be converted from one to the other with training is far from resolved.

Implications for training rowers.

The previous information was presented for the purpose of drawing the reader's attention to an important, but frequently overlooked, feature of training. It is, that an athlete must improve the aerobic capacity of both their slow and fast twitch muscle fibers in order to maximize VO_{2max} . It is not sufficient to work only at lactate threshold and sub-threshold speeds to accomplish this. Those training intensities will improve the aerobic capacity of slow twitch muscle fibers and perhaps some low-threshold FTa fibers. At the same time, however, most of the FTa and very few FTx fibers will be recruited and trained.

As indicated earlier, the major effect of training on fast twitch muscle fibers appears to be very similar regardless of their type. They undergo changes that make them more enduring. When they are used to perform work they improve their capacity for aerobic metabolism by increasing capillarization and mitochondria regardless of whether they are FTa or FTx fibers, and regardless if the work is intense, rapid, and of short duration. Since training that approaches and surpasses VO_{2max} speeds is probably necessary to recruit high threshold FTa and all FTx fibers into the effort, it is no wonder that improvements of VO_{2max} have been reported where athletes trained with very short, intense efforts.

This, I believe, explains why high-intensity training has produced increases in maximal oxygen consumption in so many pieces of research. A greater number of fast twitch muscle fibers are recruited with high-intensity training. As a result, these fibers increase their ability to absorb oxygen from the bloodstream, and transport it to a larger number of mitochondria where it can be made available for aerobic metabolism. Thus, when the pool of muscle fibers capable of taking up oxygen has been increased, the result should be an improvement of VO_{2max}

As I mentioned earlier, it appears that high intensity training also produces endurance benefits that are not achieved during low- and moderate-intensity training. In most studies, subjects who performed high-intensity training also improved buffering capacity and lactate removal rates, while those engaging in moderate intensity training did not (Edge, Bishop and Goodman, 2006; Pilegaard, Jeul, and Wibrand 1993). These two processes contribute to endurance in FTa and FTx fibers by allowing them to continue contracting with greater power for a longer period of time. Training may also increase the buffering capacity of ST muscle fibers, although to a lesser extent.

On the negative side, there is a distinct possibility that high-intensity training reduces contractile velocity in fast twitch muscle fibers. This could be potentially damaging in events that require power. That does not mean that high-intensity training cannot improve speed and power in other ways, however. It may, for example, improve the efficiency and rapidity with which fast twitch (and slow twitch) fibers can be recruited to apply force. Consequently, the nervous system will recruit only those fibers that are needed to perform the work and they will be recruited more rapidly, in a sequence that enhances the proper application of that force. The result should be that athletes will generate more power that can be used to swim faster despite a reduction in the contractile velocity of some of their fast twitch muscle fibers. In the next section I want to discuss some methods of high-intensity training that have been proven effective for recruiting FTa and FTx fibers.

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At this point in time, the purpose of research in this area has been to determine the effect of high-intensity training on aerobic endurance. Studies concerning the best methods for improving that endurance are incomplete as concerns the most

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effective combinations of repeat distances, numbers of repetitions, and rest periods for this purpose. Nevertheless, I will present what is available.

High-intensity repeats of 30 seconds and longer have repeatedly been shown to improve both aerobic and anaerobic endurance. In one study, subjects trained with 30 second sprints, three times per week for seven weeks (Barnett, *et al*, 2004). The number of sprints increased from 4 to 10 throughout the training period. The rest period between sprints was four minutes. Subjects increased their VO_{2max} by 8%. This was accompanied by an increase in power output of 7.1% over 30 secs .

Subjects in a study by Gibala and associates (2006) also did 4 to 6 x 30 second sprints on a bicycle ergometer with 4 minutes rest between efforts. A control group did 90 to 120 minutes of continuous cycling at 65% of VO_{2max} . The experimental group improved an average of 10% on an endurance time trial while the average improvement of the control group was 7.5%. The difference between improvements for the two groups was not significant. Nevertheless, the amount of improvement in the group training with 30-second sprints was, at the least, equal to that of the group that trained continuously.

In still another study using 30 sec. efforts, well-trained distance runners did 8 to 12 repeats at 90% to 95% of maximum speed with 3 minutes rest between each repeat (Iaia, *et al*, 2008). They did this 4 times per week for 4 weeks. A control group continued their usual endurance training. The experimental group improved their running time to exhaustion at 130% of VO_{2max} by an average of 27%. Surprisingly, they did not improve VO_{2max} nor did they improve their 10 km. running times. The control group did not improve on any of these measures.

Burgonmeister and associates (2005) reported an improvement of 100% in time to exhaustion at 80% of VO_{2max} after only 6 high-intensity training session performed over a two week period. Training consisted of 4 to 7 x 30 second sprints with 4 minutes rest between each sprint.

Similar results have been reported for longer repeat distances. In one report, sets of 15 x 1 minute leg extensions at 150% VO_{2max} increased the number of capillaries around both ST and FT muscle fibers by 20% (Jensen, *et al.*, 2004). Edge, *et al.*, (2004) trained a group of athletic females with 6 to 10 x 2-minute efforts on a bicycle ergometer. Work was done at 120% to 140% of lactate threshold, 3 times per week, for 5 weeks. A control group trained with 20 to 30 minutes of continuous cycling at 80% to 95% of lactate threshold speed. Maximal oxygen consumption, (VO_{2max}) and speed at the lactate threshold improved by 10% to 14% in both groups. The difference between groups was not significant. Subjects in the high-intensity group improved buffering capacity by an average of 25% while the control group showed no improvement on this measure.

Stephano and associates (1999) reported results that were similar with interval repeats ranging in time from 30 seconds to 8 minutes at 80% to 175% of peak

power. The athletes in this study increased both their peak power and VO_2 peak. VO_2 peak is synonymous with, but not exactly the same as VO_{2max} . It is a measure of the maximum oxygen consumption reached on a particular test. The subjects also improved their time trial speeds after high-intensity training.

Jeul and associates (2004) conducted a unique study in which subjects trained only one leg, with the untrained leg serving as a control. The training program consisted of leg extensions performed at 150% of VO_{2max} for 1 minute, 15 times daily, for seven weeks. Time to exhaustion improved 29% in the trained leg. No significant improvement was noted for the untrained leg

Repeats that are shorter than 30 seconds do not usually produce improvements in aerobic and anaerobic endurance, at least not with the procedures that have been used in most research studies. Linossier and associates (1993, 1997) studied the effects of high-intensity efforts of 5 to 6 seconds. Repeats ranged from 4 to 30 with rest periods between repeats of 1 to 4 minutes. In both studies, subjects improved their rates of anaerobic energy release (anaerobic capacity) and their peak power. They did not improve either their anaerobic or aerobic endurance, however.

These results are understandable from a metabolic point of view. The energy for short sprints is derived from the release of energy by ATP and its rapid replacement via the breakdown of creatine phosphate and anaerobic glycolysis. Rest periods of 60 to 90 seconds will replace most of the creatine phosphate that was used so that it is available for each successive sprint. This will reduce the demand for anaerobic glycolysis and, therefore, the accumulation of lactic acid in muscles. Thus, even though fast twitch fibers will be recruited in large numbers during ultra-short sprints, the stimulus for improvements in the mechanisms of both aerobic and anaerobic endurance will be slight.

This fact notwithstanding, it is probable that both aerobic and anaerobic endurance could be improved if the rest periods between ultra-short sprints were less than 1 minute. This is because replacement of creatine phosphate would be incomplete and subjects would be required to rely more on anaerobic glycolysis and aerobic metabolism. In that event, the stimulus for increased oxygen consumption, buffering and lactate removal might be sufficient to enhance these mechanisms. Remember, that in the study by Tabata and associates (1996), the sprints were 20 seconds in duration with rest periods of only 10 seconds between each sprint. Despite this result, I suspect the results might not be as substantial as with longer repeat distances where both aerobic and anaerobic endurance are concerned.

In this respect, I would like to mention the results of one final study where 6 second and 30-second sprints were compared (Mohr, *et al*, 2007). One group trained with 15 x 6 second sprints with 1 minute rest between each while the other group did 8 x 30 second sprints with 1:30 sec rest between each repeat. Both groups trained 5 times weekly for 8 weeks. The test consisted of five, 30 sec. maximum effort sprints. The drop-off, or increase in time from the first to fifth sprint, was used as a measure

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of aerobic and anaerobic endurance with a smaller increase in time on the post-training test indicating an improvement of one or both. The increase in time from the first to fifth sprint was reduced by 54% in the 30-second group while the group that trained with 6-second sprints did not reduce their drop-off times.

These results seem to indicate that, if your purpose is to improve muscle power through a faster rate of energy release, the efforts should be 5 to 10 seconds in length (121/2 to 25 m repeats) and the rest periods between repeats should be 1 to 3 minutes. On the other hand, if your purpose is to use high-intensity training for improving aerobic and anaerobic endurance, efforts should be 30 seconds to several minutes in length with rest periods that allow the athletes to swim at near-maximum speeds.

Training to recruit FTa and FTx muscle fibers.

Research suggests that FTa muscle fibers are recruited at speeds exceeding those of the lactate threshold. Consequently, repeats for improving the aerobic and anaerobic endurance of these fibers should be swum at speeds corresponding to the lactate threshold and faster. These repeats can be any distance that permits the proper speed, up to and including 1500 meters. The number of repeats and the rest periods between them should allow athletes to swim at the required intensity.

A different approach may be necessary for training the aerobic and anaerobic endurance of FTx muscle fibers. Although you might expect that FTx fibers would be recruited during long repeats swum at near-maximum effort, it is possible that, the speed of those repeats might not be fast enough to recruit them until very late in the set, if at all. It is also conceivable that athletes might have to slow their training pace because of fatigue in the ST and FTa fibers before a significant number of FTx fibers have been recruited. A more reliable method for recruiting FTx fibers might be to include shorter, high-intensity repeats in the training program. These repeats should probably be short enough to allow near-maximum speeds, (50 to 75 m) and rest periods should be long enough to permit the completion of several repeats before athletes show signs of failing. Apparently rest periods as long as three to four minutes can be used effectively for this purpose although shorter rest periods might be even more effective.

The anaerobic threshold theory of training.

How does the information in this paper relate to the concept that endurance training is most effective when it is completed at the anaerobic (lactate) threshold? We have been told that speeds corresponding to this measure are optimum for improving aerobic endurance. But, are they? Not if the aerobic capacity of a large portion of an athlete's fast twitch muscle fibers is not improving. Based on what has been said, it would seem that athletes must spend some of their time swimming faster than threshold speeds to achieve this result where a large portion of their FTa fibers are concerned. By the same token, it may be necessary to spend some time

sprinting at near-maximum speeds to train the FTx fibers. It also seems apparent that training at faster than lactate threshold speeds is necessary for improving the buffering and lactate removal mechanisms of all fiber types. I believe the importance of these two processes for extending an athlete's ability to maintain near-maximum speeds has been largely overlooked in the literature on endurance training.

What of the notion that attaining high levels of blood lactate may be disadvantageous during training? Nothing could be further from the truth. The threshold for recruiting fast twitch fibers is high and cannot be reached without an elevated rate of anaerobic glycolysis. Consequently, these fibers cannot be stimulated to increase their capillaries, mitochondria, and aerobic enzymes without also producing large amounts of lactic acid. Additionally, high levels of intramuscular lactate are required to stimulate the muscles to increase their buffering capacity and lactate removal rate.

Despite what was just said, it is entirely possible that too much high-intensity training could lead to failing adaptation. I must admit that I have found this to be true during my coaching career. While it is necessary to train at fast speeds on a regular basis, athletes must be conscious of the signs of deteriorating performance when they do so. Otherwise they may experience failing adaptation and overtraining. Swimmer and coach must do enough high-intensity training to improve the aerobic and anaerobic endurance of fast twitch muscle fibers but not so much that failing adaptation occurs. Providing adequate recovery time for these fibers is the solution to this dilemma. For this reason, athletes should be constantly monitored for signs of failing adaptation.

Is the lactate-velocity curve a good measure to use for this purpose? In a word, yes. Blood lactate is relatively easy to measure and although it may not accurately reflect the actual rates of lactate production and removal in muscles, an increase in speed at the lactate threshold has still been shown to have a high relationship with endurance performance. I suggest, however, that changes in blood lactate at race speed may be a better measure of training status than measurements taken at the lactate threshold. While the lactate threshold provides an excellent guide for determining minimum training speeds where fast twitch fibers are recruited in significant numbers, a decrease in the quantity of blood lactate at race speed may be a better indicator of improving adaptation while an increase might signal the opposite effect.

Is it a waste time to train slower than lactate threshold speed?

I have indicated several times in this paper that high-intensity training provides both aerobic and anaerobic benefits to muscles that exceed those of training at lower intensities. Despite this, I do not want to leave you with the impression that swimming longer mileage at low to moderate speeds has no value. It is well known that swimming longer and slower provides recovery and repair time for fast twitch

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muscle fibers. Perhaps equally as important, however, is the possibility that the aerobic capacity of slow twitch muscle fibers may be improved to a greater extent by swimming slower than threshold speeds, than by training at faster speeds.

Human research on this point is scarce. However, there are some indications in the literature that this may, in fact, be the case. Let me cite the results of four studies that can provide some insight into this matter. The subjects were rats in all four. This should not invalidate the findings, however. Many of the most significant breakthroughs in human research began with studies where rats were used as subjects. Rats have a similar muscle structure to humans. They have slow twitch and two categories of fast twitch fibers. In this case, the fast twitch fibers are designated as fast oxidative glycolytic (FOG) and fast glycolytic (FG). The FOG fiber corresponds to the FTa fiber and the FG fiber is similar to FTx fiber in humans. (The existence of an FTb fiber has also been suggested in rats but its significance remains obscure). Rats are also easier to control than humans during training and the effects of training on their muscle fibers can be measured with greater accuracy.

In the first study, (Holloszy, 1967), a group of rats ran on a treadmill for 120 mins per day for 12 weeks at an intensity equal to between 50% and 75% of VO_{2max} . Changes in their mitochondrial density, and cytochrome c activity (an enzyme of aerobic metabolism found in the mitochondria), were compared to those of a control group of rats who simply rested in their cages for the same length of time. The trained rats increased mitochondrial density by 50% to 60% and doubled their concentration of cytochrome c while no change in either was reported for the control group of rats.

In a later study from the same laboratory, increases of cytochrome c and running time to exhaustion were reported for groups of rats who ran at different intensities and for different amount of time each day (Fitts, et al., 1975). Cytochrome c increased most (92%) for the group that trained 120 minutes/day at speeds between 50% to 60% VO_{2max} . This same group also had the longest runs to exhaustion (111 minutes). The results of this study are summarized in Table 4.

Table 4. The effect of training at 50% to 60% VO_{2max} for different amounts of time on cytochrome c, and running time to exhaustion.

Training	10 mins/day	30 mins/day	60 mins/day	120 mins/day
Cytochrome c	+16%	+31%	+38%	+92%
Time to exhau.	22 mins.	41 mins.	50 mins.	111 mins.

Fitts, R.H., F.W. Booth, W.W. Winder, and J.O. Holloszy. (1975). Skeletal muscle respiratory capacity, endurance and glycogen utilization. *American Journal of Physiology*, 228: 1029-1033.

In the latter two studies, Dudley, Abraham, and Terjung (1982) and Harms and Hickson (1983) trained groups of rats at a variety of speeds ranging from moderate (slower than lactate threshold speed) to very fast (116% of VO_{2max}). They measured the effect of training at these speeds on enzymatic markers of aerobic capacity in the ST, FOG, and FG muscle fibers of the rodents. Their results were remarkably similar and are summarized in Table 5.

Table 5. The effect of training at various intensities on increases in the activity of markers for aerobic capacity in rats.

Training speeds						
In m/min.	10/11	20/22	30	40/44	50	60
Slow twitch fibers	17-30%	23-35%	39%	30-31%	15%	7%
FOG fibers	21-33%	36-49%	47%	47-72%	42%	47%
FG fibers	0-13%	0-29%	47%	48-80%	90%	180%
Running time to exhaustion	127 mins	314 mins		569 mins		

Data from "Influence of exercise intensity and duration on biochemical adaptations in skeletal muscle," by G.A. Dudley *et al.*, (1982), *J. of Appl. Physiol., Resp., Environ., and Ex. Physiol.*, 53(4): 844-850; and "Skeletal muscle mitochondria and myoglobin, endurance, and intensity of training," by S.J. Harms and R.C. Hickson (1983), *J. of Appl. Physiol.*, 54(3): 798-802.

There were 6 training groups in the study by Dudley and colleagues. Each of the groups trained at only one designated speed. Those speeds ranged from 10 to 60 m/min in 10 m/min increments. The rats were trained, in most cases, for 60 to 90 mins/day, 5 days/wk for 8 weeks. However, the rats that trained at speeds of 50 and 60 m/min. ran for only 15 and 27 mins. daily.

Harms and Hickson (1983) had three training groups. Each group trained at 11, 22 or 44 m/min, for 40 min/day, and 6 days/wk, for 14 weeks. Ranges for improvement in the markers for aerobic metabolism are reported for both studies where the groups trained at similar speeds, (10/11, 20/22, and 40/44 m/min). Percentage improvement is indicated by only one value at each of the speeds exclusive to the study by Dudley and colleagues, (30, 50 and 60 m/min.).

The greatest improvements in the markers for aerobic capacity in FOG fibers (FTa in humans) occurred in the groups that trained at speeds of 30, 40 and 44 m/min for 40 to 90 mins/ daily. Improvements in cytochrome c activity ranged from 47% to 72%. These training speeds are considered to be in the range of 85% to 94% of VO_{2max} , for rodents, indicating that FOG fibers improved most with training speeds that were between lactate threshold and VO_{2maz} efforts. It should be mentioned, however, that the rats that were trained at speeds of 50 and 60 m/min for 15 to 30 mins/daily improved a similar amount. This suggests that the aerobic capacity of

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FTa fibers can be improved similarly by running for shorter periods at near maximum speeds or by running at slower speeds for longer periods. It should also be noted that these data suggest that running at speeds below 80% VO_{2max} for long periods of time produced vastly inferior results. Improvements of cytochrome c in the FOG fibers of rats were only increased by 18% to 30% at speeds corresponding to between 65% and 70% of VO_{2max} .

In the study by Dudley and colleagues, the group that trained at 60 m/min (116% of VO_{2max}) for 15 minutes daily nearly doubled the cytochrome c content of their FG fibers. As mentioned earlier, the FG fibers of rats correspond to the FTx fibers of humans. Groups that trained at the intermediate speeds (30, 40, 44 and 50 m/min) for 30 to 90 mins/daily had very inferior results while those that trained at the slowest speeds (10, 20 and 22 m/min) for 40 to 90 mins./daily did not increase the cytochrome c in their FG fibers at all in the study by Dudley and colleagues and only minimally (13% to 29%) in the Harms and Hickson study. These results support the belief that efforts must be in excess of VO_{2max} speeds to recruit and train the FTx muscle fibers of humans.

Where the ST muscle fibers of the rats in these studies were concerned, the markers for aerobic capacity were improved most in those groups that trained at speeds between 20, 22, 30 and 40 m/min. for 40 to 90 mins./daily. Those speeds correspond to efforts in the vicinity of 70% to 90% of VO_{2max} which is often where the anaerobic threshold is found in moderately-trained to well-trained persons. The unanticipated result was that the percentage of cytochrome c in ST fibers fell precipitously, (-15% to -82%), in groups training at faster speeds.

Harms and Hickson also reported that the markers for aerobic capacity declined approximately 14% in the slow twitch muscle fibers for the group of rats that trained at 44m/min. This training speed corresponds to 94% of VO_{2max} in rats.

Harms and Hickson charted increases of muscle myoglobin that took place at each running speed as an additional indicator of improved aerobic capacity. Myoglobin is a substance found in the cytoplasm of muscle fibers. Its function is to transport oxygen that enters the fiber to its mitochondria. It was surprising that the greatest increase of this substance in ST muscle fibers took place at the slowest training speed, 11m/min. This corresponds to an effort in the vicinity of 65% of VO_{2max} . On the other hand, the greatest increase of myoglobin in FOG fibers occurred at 44 m/min indicating that speeds approximating those of the anaerobic threshold and VO_{2max} were necessary for their recruitment and training. No increase of myoglobin was observed for the FG fibers at 44 m/min or slower training speeds. This result also supports the notion that speeds in excess of those corresponding to VO_{2max} are required to train the FG fibers of rats and perhaps the FTx fibers of humans.

Harms and Hickson also included a test of running time to exhaustion in their study. The group of rats trained at a speed of 44 m/min for 40 minutes daily improved considerably more than the two slower trained groups. They ran continuously for

569 mins. This may be because they improved the aerobic capacity of their FOG and FG fibers to a greater extent than the slower trained groups, while, at the same time, improving the aerobic capacity of their slow twitch fibers a similar amount. The two groups that trained at slower speeds, 11 and 22 m/min, were only able to run for 127 mins. and 314 mins. respectively before becoming exhausted. It is unfortunate that Dudley and colleagues did not include a running test to exhaustion in their study. I suspect the group of rats who trained at 40 or 50 m/min would have had the best results for the same reasons cited for the rats in the Harms and Hickson research. I would also speculate that groups that trained at both slower (10, 20 and 30 m/min.) and faster (60m/min.) speeds would have covered less distance at exhaustion. In the former groups, this might have been because they did not improve the aerobic capacity of their fast twitch fibers to the same extent, and, in the latter group, because they did not improve the aerobic capacity of their slow twitch fibers a significant amount.

If the findings in these four studies can be extended to humans, and I believe there is a good possibility they can, one important inference may be that slow twitch muscle fibers will improve their aerobic capacity most when they are trained at speeds where aerobic metabolism supplies most of the energy with very little contribution from anaerobic metabolism. Consequently, it is possible that a large amount of sub-threshold (slower than lactate threshold) swimming is necessary to maximize improvements in the aerobic capacity of slow twitch muscle fibers, and that too much super-threshold (faster than lactate threshold) swimming may reduce this training effect. Don't forget, however, that some swimming at very fast speeds is essential for training the aerobic capacity (and anaerobic capacity) of fast twitch muscle fibers. This may explain why, as many experts have concluded, a combination of low and high intensity training seems to produce the greatest improvements in performance.

The proportions of high and low intensity training that are most effective for improving performance is a matter of considerable debate within both the scientific and coaching communities. On the one hand, are those who favor large amounts of overdistance training at speeds that are slower than lactate threshold while others believe that better results can be obtained with greater amounts of race speed training and smaller quantities of overdistance training.

Unfortunately, very few studies have been conducted where the effects of high and low intensity training have been determined by fiber type. In most studies, the contrasting effects of low and high intensity training have been reported on mixed samples of muscle tissue containing both slow and fast twitch fibers. Even then, the results have been equivocal with some persons reporting similar increases in the activities of mitochondrial enzymes, (a marker for improved aerobic capacity), for both types of training, (Henriksson and Reitman, 1976) while in others, mitochondrial enzyme activities were only increased with low-intensity training (Gorostioga, et al. 1991).

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It is not surprising that increases of mitochondrial density can be produced with training that is exclusively low or high intensity. Two of the triggers for increasing mitochondrial mass are (1) an increase in muscle calcium, and (2) lower ATP concentrations in muscle. High concentrations of calcium activate a genetic mitochondrial messenger called calcium-calmodulin kinase that encourages the growth of mitochondrial tissue. This effect is best produced by prolonged endurance exercise at slow speeds. By the same token, a reduction of ATP in muscle causes an increase of AMP (adenosine monophosphate) which activates a different mitochondrial messenger called AMP-activated protein kinase (AMPK) that also triggers mitochondrial growth. Reductions in ATP are more prevalent during high-intensity exercise.

The importance of low-intensity training.

My experience over 30 years of coaching has been that high-intensity training, while necessary, should be administered judiciously with adequate recovery time provided. Otherwise, the athletes may become overtrained.

Consequently, the importance of low intensity training should not be overlooked by coaches and athletes in this time of renewed emphasis on high intensity training. In support of this notion, Esteve-Lanao and co-workers (2005) reported strong relationships between the volume of low intensity training and running performance over 4 km ($r=0.79$) and 10 km ($r=0.97$).

In a later study, Esteve-Lanao and colleagues (2007) investigated the effects of mixed training on performance. Two groups, each consisting of 6 sub-elite runners, performed equal amounts of high-intensity training (8.4% of total volume). One group, called the low-intensity group, did more low intensity training, (81% of total volume) while the training of another other group, termed the moderate-intensity group, consisted of more moderate intensity running, (67% of total volume). After 5 months of training, the 10.4 km running performance of the low intensity group had improved an average of 157 seconds as compared to an improvement of 122 seconds for the moderate intensity group.

In another study, Fiskerstrand and Seiler (2004) used a longitudinal approach to study the relative impact of high intensity and low intensity training on performance. They studied the training programs of 21 different international medal-winning Norwegian rowers who competed between the years of 1970 and 2001. During that time the best 6 minute rowing ergometer performance increased by 10% and VO_{2max} increased by 12%. During this same time the low intensity training mileage for medal-winning Norwegian rowers increased from 30 hours/week to 50 hours/week, while the amount of high intensity training declined from 23 hours/week to 7 hours/week.

Two studies by Davies, Packer and Brooks (1981 and 1982) also support the need for adequate volumes of low intensity training. Using rats as subjects, they reported

that endurance training increased the size and number of mitochondria in the rats by 100%, which was credited with producing a 400% increase in their running time to exhaustion. VO_{2max} was also improved by 15% for this group. At the same time, a control group also increased their VO_{2max} by 15% with sprint training, but they did not increase mitochondria significantly nor did they improve their running endurance. These results suggest that it is was the increase in muscle mitochondria and not an increase of VO_{2max} that was responsible for improving running endurance and further, that while sprint training may improve VO_{2max} similarly to low-intensity training, it is not nearly as effective for improving endurance performance. Although fiber specific effects were not reported in this study, I suspect that the endurance trained rats increased mitochondrial density considerably in their slow twitch and FOG fibers while the sprint trained rats did not improve their running endurance because they failed to increase mitochondria in their slow twitch, and, perhaps also many of their FOG muscle fibers.

Results with human subjects have generally concluded that training at high or low intensities can be equally effective for improving sprint and middle-distance performances, at least where sprint and middle distance performances are concerned. Faude and associates (2008) found no difference in performances over 100 and 400 meters for a group of swimmers that trained exclusively with high or low intensity repeats. Using a randomized cross-over experimental design the swimmers were separated into two groups. Group A used low intensity, high volume training for 4 weeks followed by 4 weeks of high-intensity, lower volume training. Group B used the opposite approach, performing high intensity training during the first 4 weeks and low intensity training during the subsequent 4 week period. Each 4 week training period was followed by an identical 1 week taper. The 100 and 400 meter time trials were swum at the end of each taper week.

It is unfortunate that this study did not include at least three additional training groups. One that trained with a large amount of low-intensity mileage mixed with some lesser amount of high-intensity mileage. A second group that trained with equal amounts of both and a third group that trained with a large amount of high-intensity swimming and some lesser percentage of low-intensity mileage.

It may be that studies of the type just suggested and studies where the effects of exclusive high or low intensity training are determined according to fiber type will steer us to the proportions of these two categories of training that are most effective for improving sprint, middle distance and distance performances in humans. As I indicated earlier, it is quite possible that lower intensity training is most effective for improving the aerobic capacity of slow twitch muscle fibers while high intensity training is needed to achieve the same result with fast twitch muscle fibers. Consequently, training of only one type, long and moderate, or short and fast may not maximize a swimmer's aerobic capacity as effectively as a mixture of the two. Determining the proper mixture for different race distances and for different types of athletes (sprint, middle distance, and distance) is certainly a topic worthy of study.

Some questions that require further research.

The science behind training individual muscle fiber types is in its infancy and there will be much more information available in the future. I have indicated that speeds in excess of VO_{2max} intensity may be required to recruit and train FTx fibers.. I have also hypothesized that speeds slower than lactate threshold intensity may be optimum for training the aerobic capacity of slow twitch muscle fibers. Neither of these suppositions has been proven conclusively, however. Consequently, one of the first questions that researchers need to answer is, **“What are the optimum combinations of repeat distances, repetitions, repeat speeds, and rest periods for improving the aerobic and anaerobic endurance of fast twitch and slow twitch muscle fibers in humans?”** Another way to approach this issue might be to ask, **“What effect do many of the common repeat sets used in competitive swimming have on the aerobic and anaerobic endurance of fast twitch and slow twitch muscle fibers?”**

Additionally, I alluded to the possibility that fast twitch muscle fibers may suffer failing adaptation with too much high-intensity training. This leads me to wonder, **“What is the optimum dosage for high-intensity training in terms of mileage per year, per season, and per week?”**

Another issue of concern is, **“Are the training intensities required to recruit FTa and FTx fibers reduced when those fibers become trained?”** In other words, “Do FTa fibers become easier to recruit, i.e. recruited at slower speeds, when they take on more of the characteristics of slow twitch fibers?” Likewise, can FTx fibers be recruited at lower training intensities when they take on the characteristics of FTa fibers?

Most coaches are aware that distance swimmers and sprinters should train with different types and combinations of endurance and sprint repeats. Could this be because of their muscle structure? An important question in this respect is, **“In what ways should the training of athletes with a preponderance of slow twitch or fast twitch muscle fibers differ from the general population of athletes?”** Some of the information presented in this paper, could be interpreted to mean that athletes with a large percentage of slow twitch fibers should do a substantial volume of training slower than their lactate threshold speeds because doing so will improve the aerobic capacity of the majority of their muscle fibers. While they should also do some high-intensity training to improve the aerobic and anaerobic endurance of their fast twitch fibers, they probably do not require as much high-intensity mileage as athletes with a greater percentage of fast twitch fibers for this purpose.

Conversely, more intense training with longer periods of recovery could be recommended for sprinters with a large percentage of fast twitch fibers because this kind of training may be essential for improving aerobic and anaerobic endurance for the majority of their muscle fibers. Nevertheless, it may also be advisable to balance

their high-intensity training with some slow and moderate endurance mileage to provide recovery and repair time for their fast twitch fibers and to improve the aerobic capacity of their slow twitch muscle fibers. Perhaps most important, is the possibility that an adequate amount of moderate intensity swimming might improve the ability of their ST fibers to take up and oxidize lactate during races which will reduce the accumulation of lactic acid in their FT fibers and delay fatigue. These athletes should probably, also include a significant amount of power training in their programs so they can improve, or at least maintain, the contractile velocity and power output of their fast twitch fibers during hard training.

In this same vein, **the possibility that sprint athletes may lose speed and power by training the aerobic and anaerobic endurance of fast twitch fibers should be addressed.** During my career I noticed a trend for sprint athletes I coached to improve more in their 100 and 200 events but relatively less in their 50 event. Is this because we sacrificed power for endurance? It is well documented that fast twitch fibers become smaller and slower with endurance training (Noakes, 2001). Despite what I said earlier about the effect of muscle recruitment on speed, it is conceivable that too much aerobic and anaerobic endurance training, (or too little power training) will cause fast twitch fibers to decrease in size and contractile velocity so much that athletes lose power and sprint speed. In this respect, I should mention there have been indications that plyometric training can increase contractile velocity in single muscle fibers (Malisoux *et al.*, 2007). Therefore, it is probably wise to include this type of training in the programs of sprinters and middle distance swimmers. There have also been some interesting findings on the role of taper in this regard.

As I mentioned earlier, a study by Andersen and associates (2005) showed evidence that subjects FTx fibers took on the properties of FTa fiber during training. I did not mention that these researchers also studied the effects of detraining on those fibers. It was determined that the proportion of FTx fibers increased to 7.7% after three months of no training following a training period of similar length. Surprisingly, this was significantly greater than the subjects' average pre-training FTx percentage of 5.6%. The increase of FTx fibers was accompanied by improvements of angular velocity (14%) and power (44%) during unloaded knee extensions. These improvements of unloaded angular velocity and power after detraining also represented a significant increase over the subjects pre-training scores on the same tests. The rate of force development (a measure of time to reach peak power output) was also increased by 23% after detraining. It is possible, therefore, that one of the effects of taper is to increase the percentage of FTx fibers beyond pre-training values by, perhaps, converting (or reconverting) FTa fibers to FTx fibers so that the potential for power and speed are improved.

Pertaining to this same topic, Andersen and Aagard (2000) reported an increase of fibers in transition from FTa to FTx during detraining. The percentage of FTax and FTxa fibers increased from 2% before training to 17% after a period of training followed by an identical period of detraining. These results raise the possibility

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that a short period of tapering might produce the best of all worlds. Fast twitch fibers might retain most or all of the adaptations that improved their aerobic and anaerobic endurance, while, at the same time, regaining most or all of the contractile speed and power they lost during training.

Summary.

With this paper I have presented a theory that a certain amount of high-intensity training is necessary to maximize aerobic and anaerobic endurance because it improves these attributes in fast twitch muscle fibers. At the same time, I have cautioned that a significant amount of lower intensity training is also needed because it may improve the aerobic capacity of slow twitch muscle fibers more effectively.

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