

Part II: Training Fast Twitch Muscle Fibers: Why and How

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Abstract

With the finding that short, intense sprints can improve aerobic capacity (45), 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 to Part II.

In Part I of this paper the thesis was presented that an athlete's endurance capacity could be enhanced through the use of high intensity training in the form of sprints and middle distance repeats swum very rapidly. This is because high intensity training causes greater numbers of fast twitch muscle fibers to be recruited into the effort, which, in turn, increases their aerobic capacity. To substantiate this thesis, the characteristics of fast and slow twitch were discussed as well as the manner in which they are recruited during work. In Part II, the results of research where the effects of high intensity training on aerobic and anaerobic endurance were studied will be presented. Some repeat sets that could enhance aerobic capacity will be suggested from this research. The connection between this information and its assumed contradiction with the Anaerobic threshold concept of training will also be discussed. Finally, some avenues for future research will be suggested.

Training fast twitch muscle fibers.

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. As indicated previously, training which is usually considered anaerobic has, surprisingly, been shown to improve aerobic endurance. This writer has suggested that those improvements occurred because high intensity training recruited and increased the aerobic capacity of fast twitch muscle fibers, thus increasing the subjects' total aerobic capacity. Studies concerning the best methods for improving the endurance of fast twitch muscle fibers are incomplete as concerns the most effective combinations of repeat distances, numbers of repetitions, and rest periods for this purpose. Nevertheless, I will present what is available. In the first section the effect on endurance and performance of repeats that are 30 seconds and longer will be

discussed. The effect of repeats that are shorter than 30 seconds in length, so-called ultra-short repeats, will be described in the second section.

Repeats of 30 secs. and longer. 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 (4). 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 (21) 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 (27). 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.

Burgenmeister and associates (8) 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% (28). Edge, *et al.*, (12) showed that high intensity training was at least as effective as traditional training for improving VO_{2max} and lactate threshold velocity and more effective for improving buffering capacity. These researchers 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 (44) 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 $\text{VO}_{2\text{max}}$. 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 (29) 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 $\text{VO}_{2\text{max}}$ 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 shorter than 30 seconds in length. 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. In two studies, Linossier and associates (32, 33) determined 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 pieces of research 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 by 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 (45), the sprints were 20 seconds in duration with rest periods of only 10 seconds between each sprint. Nevertheless, I suspect that both aerobic and anaerobic endurance may be improved to a greater extent with longer repeat efforts.

In this respect, I would like to mention the results of one final study where 6 second and 30-second sprints were compared (38). 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 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 (12.5 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. This being said, the possibility that ultra-short sprints performed with equally short rest periods of 5 to 20 seconds may improve both aerobic and anaerobic endurance should not be discounted.

Training to recruit FTa and FTx muscle fibers.

Research suggests that significant numbers of FTa muscle fibers will be recruited when training speeds reach and exceed those where the production and elimination of lactate are in balance. Consequently, repeats for improving the aerobic and anaerobic endurance of these fibers should be swum at speeds that are faster than lactate threshold velocity. 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 be structured so athletes can 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 sufficient to recruit them until very late in the set, if at all. Quite possibly, athletes might have to slow their training pace during the set because of fatigue in the FTa fibers before a significant number of FTx fibers have been recruited. Therefore, 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, (25 to 75 m) and rest periods should be long enough to permit the completion of several repeats before athletes show signs of failing. Research cited earlier suggests that 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 increased. Based on research cited earlier, 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 aerobically. It also seems apparent that training faster than lactate threshold speeds is necessary for improving the buffering and lactate removal mechanisms of FTa fibers while speeds in excess of those corresponding to VO_{2max} may be necessary for the same purposes where FTx fibers are concerned. I believe the importance of these two processes, buffering and lactate removal, for extending an athlete's ability to maintain near-maximum speeds has been largely overlooked in the literature on endurance training and should be emphasized more in training programs.

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, myoglobin and aerobic enzymes without also producing large amounts of lactic acid. Additionally, high levels of intramuscular lactate will necessarily accompany efforts to both increase the buffering capacity and lactate removal rates of fast twitch muscle fibers.

Despite what was just said about the inevitability of producing lactic acid when fast twitch fibers are recruited, 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. Providing adequate recovery time for the fast twitch fibers is the solution to this dilemma. For this reason, athletes should be constantly monitored for signs of failing adaptation when they are training intensely.

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 slow twitch and some fast twitch

fibers will be recruited, a decrease in the quantity of blood lactate at race speed may be a better indicator of improving adaptation while an increase should 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 muscle fibers while also improving the aerobic capacity of slow twitch fibers. Perhaps equally important, however, is the possibility that the aerobic capacity of slow twitch muscle fibers can be improved to a greater extent by swimming slower than threshold speeds, than by training at faster speeds even though slow twitch fibers are also recruited at those speeds.

Human research on this point is scarce. However, there are some indications in the literature that training at sub-maximum speeds may improve the aerobic capacity of slow twitch muscle fibers to a greater extent than high-intensity training. 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). Another advantage of studying rats as opposed to humans is that the former are easier to control during training and the effects of training on their muscle fibers can be measured with greater accuracy.

In the first study, (26), 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 found in the mitochondria, that speeds up aerobic metabolism.), 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 measure 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 that ran at different intensities and for different amounts of time in training (19). Cytochrome c increased most

(92%) for the group that trained 120 minutes/day at speeds between 50% and 60% of VO_{2max} . This same group also had the longest runs to exhaustion (average, 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 (11) and Harms and Hickson (24) trained groups of rats at a variety of speeds ranging from moderate (65% of VO_{2max}) 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. The results were remarkably similar in both studies. They 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 27 and 15 mins. daily.

Harms and Hickson 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. In Table 5, the ranges of 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.

This indicates that the FOG fibers of rats 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 and 27 mins/daily improved a similar amount. If a similar result occurred with human subjects, it would suggest that the aerobic capacity of FTa fibers could be improved a comparable amount by either 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. The activity of cytochrome c in the FOG fibers of rats was only increased by 18% to 30% at speeds corresponding to between 65% and 70% of VO_{2max} (10, 11, and 20, and 22m/min).

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 27 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 result 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 of 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 humans. The unanticipated result was that the percentage of cytochrome c in ST fibers fell precipitously, (-15% to -82%), in groups training at faster speeds (50 and 60 m/min). As indicated earlier, this result suggests that the aerobic capacity of slow twitch muscle fibers can be improved more with low intensity than with high intensity training.

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 also 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. It's function is to transport oxygen from the outer membrane of muscle fibers to their mitochondria. It was surprising that the greatest increase of this substance in ST muscle fibers took place at the slowest training speed in this study, 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 training speeds of 44 m/min and slower. This result also supports the notion that speeds in excess of those corresponding to VO_{2max} are required to recruit and train the FG fibers of rats and perhaps also the FTx fibers of humans.

Harms and Hickson also included a test of running time to exhaustion in their study. The group of rats that 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 and anaerobic capacities 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 to those groups that trained at slower speeds. 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 trained at both slower (10, 20 and 30 m/min.) and faster (60m/min.) speeds would have reached exhaustion in considerably less time than the groups training at 40 and 50 m/min. 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 as it was improved by training at faster speeds so that the rats overall aerobic capacity was lower. If a quicker time to exhaustion had occurred in the group trained at 60 m/min it would probably have been because improvements in the aerobic capacity of their slow twitch fibers were so minimal that they could not be compensated for by the increased aerobic and anaerobic endurance of their fast twitch muscle fibers. In which case their overall aerobic capacity would also have been lower than the groups that had trained at 40 and 50 m/min.

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 would 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. In other words, at speeds where a significant number of slow twitch muscle fibers are recruited to do the work with very little involvement of the lactic acid producing fast twitch fibers. 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 should be included in a training program because it 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.

Balancing high-intensity and lower intensity training.

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, there 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, (25) while in others, mitochondrial enzyme activities were only increased with low-intensity training (22).

It is not surprising that increases of mitochondrial density can be produced in samples of mixed muscle tissue with training that is either low or high intensity in nature. 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) that 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. I suspect overdistance training

increases the mitochondrial density of slow twitch fibers through an increase of calcium, and, that both an increase of calcium and a reduction of ATP augment the mitochondrial density of fast twitch fibers.

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 (15) 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 (14) 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 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 (17) used a longitudinal approach to study the impact of differing amounts of high intensity and low intensity training on performance. They studied the training programs of 21 different international medal-winning Norwegian rowers who each competed for some period of time between the years of 1970 and 2001. During that time the best 6 minute rowing ergometer performance by a medal-winning Norwegian rower increased by 10% and the best VO_{2max} score increased by 12%. During this same time period the low intensity training mileage for medal-winning 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.

A study by Davies, Packer and Brooks (10) also supports 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, another group of rats also increased their VO_{2max} by 15% with sprint training, but they did not increase the mitochondria in their muscles by a significant amount 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 improved running performance in the endurance-trained rats, 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 also in some of their 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 some of their FOG muscle fibers.

Results with human subjects have generally shown that training at high or low intensities can be equally effective for improving sprint and middle-distance performances. Such research is far from conclusive, however. Faude and associates (16) 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 a 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 a 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, this is one of the first questions that researchers need to answer. Another 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 some of 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, suggests 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 aerobic capacity in the lion’s share 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 could be recommended for sprinters with a large percentage of fast twitch fibers because this kind of training may be essential for improving the aerobic and anaerobic endurance of the bulk 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 while doing so. 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. Sprinters 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 my training sacrificed power for endurance? It is well documented that fast twitch fibers become smaller and slower with endurance training (39). 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 (36). 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.

What happens to muscle fibers during taper?

As I mentioned earlier, a study by Andersen and associates (2) showed evidence that subjects FTx fibers took on some of 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 value of 7.7% 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. Those 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 and reconverting FTa fibers to FTx fibers so that an athlete's potential for power and speed are improved.

Pertaining to this same topic, Andersen and Aagard (1) 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

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 the 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 of its effect on slow twitch muscle fibers. While slow twitch fibers are also recruited during high intensity training, it may be that lower intensity training will improve their aerobic capacity more effectively than training at faster speeds.

References

1. Andersen, J.L. and P. Aagaard. (2000). Myosin heavy chain IIX overshoot in human skeletal muscle. *Muscle and Nerve*. 23: 1095-1104.
2. Andersen, L.L., J.L. Andersen, S.P. Magnussen, C. Suetta, J.L. Madsen, L.R. Christensen, and P. Asgaard. (2005). Changes in human muscle force-velocity relationship in response to resistance training and subsequent detraining. *Journal of Applied Physiology*, 99: 87-94.
3. Andersen, J.L., H. Klitgaard, and B. Saltin. (1994). Myosin heavy chain isoforms in single fibres from m.vastus lateralis of sprinters: influence of training. *Acta Physiologica Scandinavia*, 151: 135-142.
4. Barnett, C., M. Carey, J. Proietto, E. Cerin, M.A. Febbraio, and D. Jenkins. (2004). Muscle metabolism during sprint exercise in man: influence of sprint training. *Journal of Science and Medicine in Sport*, 7: 314-322.
5. Bogdanis, G.C. (2009). Fatigue and training status. In C. Williams and S. Ratel (Eds.) *Human Muscle Fatigue*, (pp.164-204). New York, N.Y.: Routledge.
6. Bonen, A. (2006). Skeletal muscle lactate transport and transporters. In M. Hargreaves and L. Spriet (Eds.), *Exercise Metabolism* (pp. 71-87). Champaign, IL: Human Kinetics.
7. Brooks, G.A., T.D. Fahey, and K.M. Baldwin. (2005). *Exercise Physiology: Human Bioenergetics and its Applications*. New York, N.Y.: McGraw-Hill Companies.
8. Burgonmeister, K.A., S.C. Hughes, G.J. Heigenhauser, S.N. Bradwell, and M.J. Gibala. (2005). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of Applied Physiology*, 98: 1985-1990.
9. D'Antona, G., F. Lanfranconi, M.A. Pellegrino, L. Brocca, R. Adami, R. Rossi, G. Mora, D. Miotti, M. Canepari, and R. Bottinelli. (2006). Skeletal muscle hypertrophy and structure and function of skeletal muscle fibres in male body builders. *Journal of Physiology*, 570: 611-627.
10. Davies, K.J.A., L. Packer and G.A. Brooks. (1981). Biochemical adaptation of mitochondria, muscle, and whole-animal respiration to endurance training. *Archives of Biochemistry and Biophysics*. 209: 539-554.
11. Dudley, G.A., W.M. Abraham, and R.I. Terjung. (1982). Influence of exercise intensity and duration on biochemical adaptations in skeletal muscle. *Journal*

- of Applied Physiology, Respiratory, Environmental, and Exercise Physiology*, 53(4): 844-850.
12. Edge, J., D. Bishop, and C. Goodman. (2006). The effects of training intensity on muscle buffer capacity in females. *European Journal of Applied Physiology*, 96: 97-105.
 13. Edington, D.W. and V. R. Edgerton. (1976). *Biology of Physical Activity*. Boston, MA: Houghton-Mifflin.
 14. Esteve-Lanao, J., C. Foster, S. Seiler, A. Lucia. (2007). Impact of training intensity distribution on performance in endurance athletes. *Journal of Strength and Conditioning Research*, 21: 943-949.
 15. Esteve-Lanao, J. AF San Juan, CP Earnest, C. Foster, and A. Lucia. (2005). How do endurance runners actually train? Relationship with competition performance. *Medicine and Science in Sports and Exercise*, 37: 496-504.
 16. Faude, O., T. Meyer, J. Sharhag, F. Weins, A. Urhausen, W. Kindermann. (2008). Volume vs. intensity in the training of competitive swimmers. *International Journal of Sports Medicine*, 29: 906-912.
 17. Fiskerstrand, A., and KS Seiler. (2004). Training and performance characteristics among Norwegian international rowers, 1970-2001. *Scandinavian Journal of Medicine and Science in Sports*, 14:303-310.
 18. Fitts, R.H. and J.J. Widrick. (1996). Muscle mechanics: Adaptations with exercise-training. In J.O. Holloszy (Ed.) *Exercise and Sports Sciences Reviews*, (pp. 427-443) Baltimore MD: Williams and Wilkins.
 19. 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.
 20. Gaitanos, G.C., C. Williams, L.H. Boobis, and S. Brooks. (1993). Human muscle metabolism during intermittent maximal exercise. *Journal of Applied Physiology*, 75: 712-719.
 21. Gibala, M.J., J.P. Little, M. van Essen, G.P. Wilkin, K.A. Burgonmaster, A. Safdar, S. Raha, and M.A. Tarnopolsky. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *Journal of Applied Physiology*, 575(3): 901-911.
 22. Gorostioga, EM, CB Walter, C Foster, and RC Hickson. (1991). Uniqueness of interval and continuous training at the same maintained exercise intensity. *European Journal of Applied Physiology and Occupational Physiology*, 63: 101-107.
 23. Harber, M. and S. Trappe. (2008). Single muscle fiber contractile properties of young competitive distance runners. *Journal of Applied Physiology*, 105: 629-636.
 24. Harms, S.J., and R. C. Hickson. (1983). Skeletal muscle mitochondria and myoglobin, endurance, and intensity of training. *Journal of Applied Physiology*, 54(3): 798-802.
 25. Henriksson, J. and J.S. Reitman. 2008. Quantitative measures of enzyme activities in Type I and Type II muscle fibers of man after training. *Acta Physiologica Scandinavica*, 97(3): 392-397.
 26. Holloszy, J. (1967). Effects of exercise on mitochondrial oxygen uptake and Respiratory enzyme activity in skeletal muscle. *The Journal of Biological Chemistry*. 242(9): 2278-2282.
 27. Iaia, F.M., M. Thomassen, H. Kolding, T. Gunnarson, J. Wendell, T. Rostgaard, N.

- Nordsborg, P. Krstrup, L. Nybo, Y. Hellsten, and J. Bangsbo. (2008). Reduced volume but increased training intensity elevates muscle Na-K pump α 1 subunit and NHE1 expression as well as short-term work capacity in humans. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 294: R966-R974.
28. Jensen, L., J. Bangsbo, and Y. Hellsten. (2004). Effect of high intensity training on capillarization and presence of angiogenic factors in human skeletal muscle. *Journal of Physiology*, 557: 571-582.
29. Jeul, C., C. Klarskov, J.J. Nielsen, P. Krstrup, M. Mohr, and J. Bangsbo. (2004). Effect of high-intensity intermittent training on lactate and H⁺ release from human skeletal muscle. *American Journal of Physiology, Endocrinology and Metabolism*. 286: E245-E251.
30. Korhonen, M.T., A. Cristea, M. Alen, K. Makkinen, S. Suppila, A. Mero, J. T. Viitalo, L. Larsson, and K. Suominen. (2006). Aging, muscle fiber type, and contractile function in sprint-trained athletes. *Journal of Applied Physiology*, 101(3): 906-917.
31. Lindsay, F.H., J.A. Hawley, K.H. Murburgh, H.H. Schomer, T.D. Noakes, and S.C. Dennis. (1996). Improved athletic performance in highly trained cyclists after interval training. *Medicine and Science in Sports & Exercise*, 28: 1427-1434.
32. Linossier, M.T., C. Denis, D. Dormois, G. Geysant, and J.R. Lacour. (1993). Ergometric and metabolic adaptation to a 5-s sprint training programme. *European Journal of Applied Physiology and Occupational Physiology*, 67: 408-414.
33. Linossier, M.T., D. Dormois, C. Perier, C. Frey, A. Geysant, and C. Denis. (1997). Enzyme adaptations of human skeletal muscle during bicycle short-sprint training and detraining. *Acta Physiologica Scandinavica*, 161: 439-445.
34. MacDougall, J.D. (1986). Morphological changes in human skeletal muscle following strength training and immobilization. In N.L. Jones, N. McCartney, and A.J. McComas (Eds.), *Human Muscle Power* (pp. 269-288) Champaign, IL: Human Kinetics,
35. MacDougall, J.D., A.L. Hicks, J.R. MacDonald, R.S. McKelvie, H.J. Green, and K.M. Smith. (1998). Muscle performance and enzymatic adaptations to sprint interval training. *Journal of Applied Physiology*, 84: 2138-2142.
36. Malisoux, L, M. Francaux, H. Nielens, P. Renard, J. Lebacqz, and D. Theisen. (2007). calcium sensitivity of human single muscle fibers following plyometric training. *Medicine and Science in Sports and Exercise*, 38: 1901-1908.
37. McArdle, W.D., F.I. Katch, and V.L. Katch. (1996). *Exercise Physiology: Energy, nutrition, and human performance*. Baltimore, MD: Williams and Wilkins.
38. Mohr, M., P. Krustup, J.J. Neilsen, L. Nybo, M.K. Rasmussen, C. Jeul, and J. Bangsbo. (2007). Effect of two different intense training regimens on skeletal muscle ion transport proteins and fatigue development. *American Journal of Physiology, Regulatory, Integrative and Comparative Physiology*, 292: R1594-R1602.
39. Noakes, T. (2001). *Lore of Running*. Champaign, IL: Human Kinetics.
40. Pette, D. (1985). Metabolic heterogeneity of muscle fibers. *Journal of Experimental Biology*, 115: 179-189.
41. Philips, S.M. (2006). Endurance training-induced adaptations in substrate turnover and oxidation. In M. Hargreaves and L. Spriet. (Eds.), *Exercise Metabolism* . (pp. 187-213). Champaign, IL: Human Kinetics.
42. Pilegaard, H., C. Jeul, and F. Wibrand. (1993). Lactate transport studies in sarcolemmal giant vesicles from rats: Effects of training. *American Journal*

- of Physiology, Endocrinology and Metabolism*, 264: E156-E160.
43. Roman, W.J., J. Fleckenstein, S.E. Stray-Gundersen, R. Always, R.P. Always, and W.J. Gonyea. (1993). Adaptations in the elbow flexors of elderly males after heavy-resistance training. *Journal of Applied Physiology*, 74(2): 750-754.
 44. Stepto, N.K., J.A. Hawley, S.C. Dennis, and W.G. Hopkins. (1999). Effects of different interval-training programs on cycling time-trial performance. *Medicine and Science in Sports and Exercise*, 31:735-741.
 45. Tabata, I, K. Nishimura, M. Kouzaki, Y. Hirai, F. Ogita, M. Miyachi, and K. Yamamoto. (1996). Effect of moderate-intensity endurance and high-intensity intermittent training on anaerobic capacity and VO_{2max} . *Medicine & Science in Sports & Exercise*, 28(10): 1327-1330.
 46. Widrick, J.J, J.E. Stelzer, T.C. Shoeoe, and D.P. Garner. (2002). Functional properties of human muscle fibres after short-term resistance exercise training. *American journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 283: R408-R416.
 47. Widrick, J.J. S.W. Trappe, C.A. Blaser, D.L. Costill. 1996. Isometric force and maximal shortening velocity of single muscle fibers from elite master runners. *American Journal of Physiology*, 271: C666-675.
 48. Widrick, J.J., S.W. Trappe, D.L. Costill, and R.H. Fitts. (1996). Force-velocity and force-power properties of single muscle fibers from elite master runners and sedentary men. *American Journal of Physiology*, 271: C676-683.
 49. Wilmore, J.H. and D.L. Costill. (1999). *Physiology of Sport and Exercise*. Champaign, IL: Human Kinetics.
 50. Wilmore, J.H., D.L. Costill, and W.L. Kenney. (2008). *Physiology of Sport and Exercise*. Champaign, IL.: Human Kinetics.